# ADHD

## Dividing and Drugging

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## De-constructing the ADHD narrative in the UK

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## Introduction

i) Purpose of this essay

The purpose of this essay is to critically examine the discourse around “ADHD” in the UK. We will do this by looking at two influential “ADHD” studies. One of the studies is from the US but is influential in shaping ADHD policy in the UK. The other study was widely promoted to the press world-wide as having found evidence that “ADHD is a genetic disease”, a claim we deconstruct in this essay. We also examine the official NICE Guideline on ADHD. NICE (National Centre for Clinical Excellence) is the body in the UK tasked with recommending treatments and best practice to the NHS. The NICE Guideline was published in 2009.

The first study we consider is a genome-wide association study. Such studies attempt to find statistical correlations between a disease or label and genetic factors. This study looked at statistically significant differences in possession of two kinds of chromosomal abnormality between an ADHD group and a control group. The study is called “Rare chromosomal deletions and duplications in attention-deficit hyperactivity disorder: a genome-wide analysis” and was published in the Lancet in September 2010. (Nigel M Williams *et al. 2010*) [1]  The second study we review is the Multi Modal study into ADHD treatment models. This was a major study sponsored by the US National Institute of Mental Health (NIMH). The study compared four “treatment” models for ADHD; “medication”, behaviour training, “medication” plus behaviour training and “community care”. The “treatment” options were compared by putting young people onto the different “treatment” programmes and measuring the outcomes against a set of 6 criteria for each one. The measures were in the main behaviour check-list reports by parents and teachers. The study was published as “A 14-Month Randomized Clinical Trial of Treatment Strategies for Attention-Deficit/Hyperactivity Disorder” in the Archives of General Psychiatry in December 1999. (The MTA Co-operative group. December 1999) [2]

We also consider the drugs used to “treat” “ADHD”, what they are and what they do, and note that, perhaps surprisingly, there is no coherent scientific explanation for how they are supposed to work.

The debate about ADHD and stimulant drugging tends to be framed by some parties in terms of science v. non-science. Apologists for ADHD-stimulant drugging tend to refer to the “science” alleged to be driving their position. One of the aims of this essay is to look at the role that “science” plays in the ADHD narrative.  The narrative does sometimes use actual findings from peer reviewed studies. But the ADHD narrative is constructed partly by pre-loading the construction of the studies and partly by filtering the results through a certain language game. Sometimes (often in fact) results are selectively mined by the study authors themselves to tell the story they want to tell. Sometimes when the study (despite the best of intentions) fails to produce the required result the data from the study is subject to further statistical analysis in a follow-up paper to produce the

required result.

The ADHD narrative is an example of what Michel Foucault called a “dividing practice”. Through manipulative processes people are divided into groups. Lepers, the poor, the “mad” have all, since the Middle Ages, been incarcerated in special institutions, segregated and controlled. Foucault has shown how this objectification of certain marginalised groups has its counter-part in the creation of certain forms of subjectivity associated with power. The “ADHD” label acts is an example of a dividing practice. It creates a situation of internal exile. The young person with the label is separated off from and divided from his peers. Designated as an “ADHD child” the young person is objectified by power. There is no biological test for “ADHD” and the observed behaviour (if indeed it exists other than in the minds of the observers) may be the result of any one of a range of factors. However; *all* young people labelled “ADHD” are treated as if they *have* an objective condition, the *same* condition in fact. The prestige and status of the psychiatric profession is linked to its ability to make such designations.

It is important to realise that just because people in positions of social authority can divide people into groups based on their behaviour no “condition” has been discovered scientifically. Equally; because it is possible to show that members of such a group *statistically* are *more likely* to possess certain physical characteristics it does not follow that a condition has been established empirically, that is as something which people “have”. ADHD has no biological account of its aetiology; as measles does for example. When there is no actual “biological marker” for the category, as is the case with “ADHD”, the value of the category will depend entirely on how helpful it is to people. We contend that the category of ADHD is almost totally harmful to those so categorised. The main purposes of the category appear to be to legalise the prescription of drugs (most of which are seen as dangerous in other contexts), to extend the authority of the psychiatric profession which makes the “diagnosis”, and to resolve management problems in schools.

ii) What exactly is ADHD? How do I get it?

To get ADHD in the UK involves a visit to a psychiatrist or paediatrician, probably following a referral from a school-teacher or parent. The psychiatrist, or paediatrician, will make an assessment based on his or her observation of the behaviour of the young person together with background information provided by the school or parents. One of the two main systems for “diagnosing” ADHD is DSM-IV. This is a tick-box check-list produced by the American psychiatric profession. DSM-IV is attached here as Appendix i). To “have” ADHD by the DSM-IV system a young person has to be assessed for meeting criteria such as "Often fails to give close attention to details or makes careless mistakes in school-work, work, or other activities", “Often gets up from seat when remaining in seat is expected” or “Often does not follow instructions and fails to finish school-work, chores, or duties in the workplace (not due to oppositional behaviour or failure to

understand instructions).” A quick review of DSM-IV will show how much it is focussed on non-compliant / non-aligned behaviour in school. 4 out of 9 of the DSM-IV inattention criteria specifically reference school situations and almost all relate to school type tasks. 3 of the 6 hyperactivity-impulsivity criteria are specifically related to school situations, but most of the others would be likely to occur in a school. In DSM-IV the behaviour has to be present to the extent that it is “disruptive and inappropriate for developmental level”. The word “disruptive” shows what the problem is. ADHD is not an illness which young people experience and which they need to be cured of. The concern is with their “disruptive” behaviours. That is with the impact of their behaviour on other people. A “reduction in symptoms” will mean that they are less “disruptive”.

In the UK an alternative system is also in use known as ICD-10. This is attached as Appendix ii). This is a similar check-list style system. At this point we should note that the “diagnosis” a) involves no biological test whatsoever, b) relies on statements by parents and teachers, c) does not involve any input from the young person whatsoever and d) is made in individual cases by a psychiatrist or paediatrician. The “diagnosis” is designed to look for deficits, deficits which will require the services of professionals (clinical psychologists), or drugs, to correct. There is nothing in the “diagnosis” which looks for positive compensatory behaviours. There is no attempt to understand the young person. The young person is not asked if he or she is suffering. The “diagnosis” is made within the context of a nexus of social authority figures and relates to non-negotiable expectations parents and the school-system have about the young person's behaviour. The young person is objectified relative to these arbitrary standards.

There is no “biological marker” involved in the “diagnosis of ADHD”. It is not a “diagnosis” in the usual sense of the word. When a doctor diagnoses measles he may do so on the basis of certain symptoms. In every case his diagnosis could be confirmed by a laboratory test; the presence of the Morbillivirus type virus. If the virus was not found it would have been a mistaken diagnosis. Similarly if a doctor diagnoses Cystic Fibrosis, in every case there is a known problem with a specific gene. In these cases the diagnoses refer to something which exists, a physical reality. But the “diagnosis” of “ADHD” relates to the behaviour of a young person. Young people may be fidgety and impulsive for any number of reasons. To categorise and label behaviour as a “disorder” is an act of power.

There is no biological test “for” “ADHD”. It is a “diagnostic category” of psychiatry. [3] This is the term the authors of the NICE Guideline on ADHD use themselves. They are clear. ADHD is not a “diagnosis” of a medical or “neurological” disorder. There is no “biological” condition “ADHD”. It just doesn't exist:

> The diagnosis of ADHD does not imply a medical or neurological cause. [4]
>
> Nevertheless, the disorder remains one that is defined at a behavioural level, and its

> presence does not imply a neurological disease. [5]

At the same time the ADHD discourse (including that in the NICE Guideline) implies that “ADHD” has a physical, ontological, existence. This happens every time a reference is made to a young person “with ADHD” or who has “symptoms of ADHD”. This kind of language implies that the young person *has* something *(objective, physical, actual).* The ADHD narrative has a two-tier structure. In general the 'inner', official, narrative is more cautious in its claims-making. A second, more folksy, narrative is in circulation at a 'lower' level. The more folksy rendition of the ADHD narrative always insists on a biological “cause” directly. Here, for example is Gateshead Council in the North-East of England, in a leaflet providing “Information and Guidance for schools” on ADHD:

> While the exact cause of ADHD is as yet unknown, it is generally accepted that it is likely to be biological in nature. A great deal of scientific and medical research has identified a number of factors which appear to influence its development. [6]

Since the “diagnosis” of ADHD is based purely on observed behaviours “ADHD” *cannot* be said to *“*have a biological cause”. Another characteristic confusion here is between the small correlations which can be shown between possession of an ADHD label and biological factors and a clinical condition. The “it” in the above text implies a single biological condition. But even the scanty material showing correlations to physical factors on a statistical basis shows that such factors are varied and disparate. There is no biological clinical condition “ADHD”. Yet this text is typical of vast swathes of the folksy narrative about “ADHD”.

Psychiatry is essentially facing both ways; on the one hand there is a narrative line which holds to the idea of a biological disorder. This is the narrative strand present in the Gateshead Council text. On the other hand the “official” position is that this is not necessary for the “diagnosis”. This two-fold account; on the one hand a full admission that there is no medical or neurological cause associated with an ADHD label and on the other the tendency to promote just such an explanation, is an unresolved contradiction within the narrative. The promotion of the biological narrative though is not limited to external actors. The NICE Guideline itself with its talk of “symptoms of ADHD” and young people “with ADHD” seems to imply a biological basis “for ADHD” even as they accept at the same time that there is no biological basis for the “disorder”. Such language implies a biological model. A considerable amount of resources are spent trying to establish various kinds of biological factors which can be statistically linked to the label. The statistical factors which can be shown to correlate (usually with quite small levels of significance) to the label are as varied as genetic variations, diet, food additives, brain injuries and certain kinds of brain activity. There is no single “condition” ADHD even on the basis of statistics.
The debate around ADHD in public discourse and in the press often seems to centre on whether or

not “ADHD” is a “scientific” or a “real” condition. The Royal College of Psychiatrists will perhaps be very happy to see the “controversy” framed in this way. This discussion is a red herring. It shifts attention away from a more critical questioning. The Royal College of Psychiatrists were one of the principal contributors to the NICE Guideline on ADHD. And, as we have seen above, this document does not hold (officially and when asked to be precise) that ADHD is a real, objective, condition. There is in fact no argument about whether “ADHD is a real condition”. The *actual* controversy is about the purpose and consequences of ADHD labelling and drugging. Some questions that could be asked are: how do psychiatric terms (such as ADHD) come to have the status that they do? How is it for example that an “ADHD” “diagnosis” has more status than a designation of “EBD” (Educational and Behavioural Difficulties) though scientifically speaking both are equivalent? What are the consequences of an ADHD label for one so labelled? What function does the ADHD “diagnosis” play in the educational system? Who in fact benefits from the system of ADHD labelling and drugging? Questioning ADHD properly also means questioning the role of psychiatry as an institution in society.

ADHD promoters use a linguistic model borrowed from medicine about “diagnosis”, “symptoms” and “treatment”. This is a misappropriation of medical terminology; its application to a process which owes little to either science or medicine. The language of “symptoms”, “diagnosis” and “treatment” masks what is happening.

A person “gets” “ADHD” because their parents or teachers decided that there was something wrong with them. Teachers receive briefings “informing” them “about ADHD” and are guided towards “spotting” potential cases. After ADHD is “suspected” the parents will then take the young person (typically a boy) to a psychiatrist or paediatrician who favours the ADHD “diagnosis”. This almost always effects young people though there is a small but emerging market for “adult ADHD”.

The construction of a narrative that “ADHD is a disease” is probably done to facilitate drugging. The public might baulk at drugging young people purely for behavioural reasons but they will accept it if ADHD is a disease. We have discussed the harm which ADHD drugs do in another paper [7] but we review this matter briefly in this paper in Section 3).

iii) A circular argument

The ADHD narrative is sometimes supported by a circular argument. The impulsive behaviours of those with an ADHD label are taken as evidence which confirms the label.

In 2007 NICE commissioned the National Collaborating Centre for Mental Health to produce a Guideline on ADHD to inform treatment in the NHS. The recommendation was published in 2009 in a document called “The NICE Guideline on Diagnosis and Management of ADHD in children,

young people and adults". [3] We review this document in Section 4).

This document contains a particularly egregious example of the extent to which ADHD comes into being via a circular argument:

> Children and young people with ADHD have been shown to have greater impaired attention, less impulse control, and greater off-task, restless and vocal behaviour (Fischer et al., 1990). [8]

It is perhaps worth pausing for a minute to consider the full folly of this statement. In fact inattention and problems with impulse control as manifested in behaviour are what it takes to get "diagnosed" "with" "ADHD". There is no other test for "ADHD". The "diagnosis" occurs simply if the terms of the DSM-IV or ICD-10 behaviour check-list are met. Of course then a young person "with ADHD" can be shown to "have greater impaired attention, less impulse control". That is how, and only how, they got the label in the first place. Nothing has been "discovered" here. It is purely circular. This is an especially egregious example of the kind of circularity prevalent in ADHD discourses. The circularity however, as it goes round and round, serves to promote the "ADHD" story and keep it alive.

The above is not an isolated instance in the NICE ADHD Guideline. This is a "conclusion" the authors have drawn about "ADHD" after "reviewing evidence":

> There is evidence for psychological, social and educational impairments in both children and adults with ADHD. [9]

But "impairment" is a key element in making the "diagnosis" of ADHD. This is one of the criteria in DSM-IV:

> There must be clear evidence of significant impairment in social, school, or work functioning. (See Appendix i)).

People with certain behavioural characteristics are given the label. People with the label are then "found" to have the characteristics. This is a kind of solipsism of psychiatry. The designation has no existence other than in terms of itself. It can "prove" itself only by repeating its own definition. Endless "research" projects on ADHD also promote and reinforce the label in a circular way. The basis on which many ADHD research projects are carried out is an assumption that there is a biological marker. The job of the study is to find it. If a study does not find the supposed biological

marker it does not disprove the theory. Researchers can simply construct a new study. While the game is going on all parties act as if it is only a matter of time until the biological marker is found. One variant on this game is to accept that there is no single biological marker for ADHD (a tacit admission that it is not like measles or Cystic Fibrosis) but that there are a multiplicity of biological markers. This makes it easier to assemble the "evidence" for the "condition" at the cost of admitting that the "condition" is not given by a single aetiology. The researchers on these kinds of studies invariably appear to be convinced that they are "researching" something real called "ADHD". But they are in fact simply finding a varied range of statistical correlations to the label. The endless research projects "into ADHD" serve to reinforce the idea that it is a "real condition" - something that *really exists*. Many researchers appear not to be able to tell the difference between a word which points to something which really exists (e.g. "measles") and a word which is simply an invented designation to help manage populations of children ("class 2B", "ADHD").

iv) ADHD is a dividing practice

ADHD is an example of what the French writer Michel Foucault called a "dividing practice". A marginal group is defined as separate from the majority and is subjected to some kind of external or internal exile. In the case of ADHD both the label and the physical effects of the drugging that often goes with it create for the young person a situation of internal exile (separation from his peers while remaining in the same physical space as them). He is segregated by the label, as well as by the physical effects of the drugs. (His behaviour is controlled by drugs).

ADHD studies tend to all share the same basic structure. This is the case whether they are Magnetic Resonance Imaging (MRI) scan studies, Positron Emission Tomography (PET) scan studies, or genome wide association studies. The process starts with the separation of young people into two groups: the ADHD group and the normals. The study then finds that statistical differences can be observed *between the groups*. For example a typical MRI study will find that the average reactions times to a computer test were slightly slower in the ADHD group than in the group of normals and will correlate this to brain activity (as measured by the MRI scanner). This "evidence" is then used to offer some kind of explanation about "ADHD" or, at the least, to confirm that the "label is not just a social construct". Look, the researchers seem to be saying, they really are different. But the "ADHD" so demonstrated is in the realm of statistics. The differences are based on comparing averages between the groups. This is not medical science. Medical science researches biological pathways and aetiologies with a view to ending human suffering.

In most studies those "with ADHD" have been pre-sorted out of the normal group. The normal group specifically does not include those with unusually high inattention. The "ADHD children" are not being compared even with the population average but with a group with better than average attentiveness. The dice are thus loaded from the start. Given this initial division into two contrasting

groups with different behaviours it is inevitable that statistically significant differences will be found. This leads to a misleading reinforcing of the idea that “ADHD” is “something real”. (The genome study we review in Section 1) is a rare exception in that the control group was a genuine control group representing a total range of the population. This came about because the researchers were using an old data set of genetic data which could not be filtered “for ADHD”).

The forces which drive academic publishing support the finding of difference not similarity. See for example the study “Publication bias in clinical research” published in The Lancet in April 1991 (Easterbrook P.J *et al.*. April 1991):

> Studies with statistically significant results were more likely to be published than those finding no difference between the study groups (adjusted odds ratio [OR] 2.32; 95% confidence interval [CI] 1.25-4.28). [10]

All these “research” projects thus act to support the ADHD narrative. This is not an investigation into empirical reality. It is a process of assembling evidence to support the narrative. A narrative about difference. The ”evidence” is based on comparisons of averages between the groups. Rather than a process of scientific discovery this is a persecutory process which uses scientific methodology. This is the case even without the engineering of a result by removing those with the to be tested factor from the group of “normals”.

The kinds of research project which are carried out and published are ones which one way or another will lead to support for drugging. This is perhaps inevitable given the nature of the funding regime. We discuss some aspects of research funding in Sections 4) vii) and 5) ii) and the tendency to look for manipulative and profitable “solutions” to social problems in Section 5) i).

v) Outline of the essay

In Section 1) of this essay we review a recent genome-wide association study. Such studies look for statistically significant differences in genetic make-up between a group of young people “with ADHD” and group taken from the general population. This particular study found a correlation between a particular type of genetic abnormality and inattentiveness/impulsiveness. 14% of the “ADHD group” possessed the particular genetic abnormality compared to 7% of the general population. This study was part-funded by the Wellcome Trust. The Wellcome Trust is a non-governmental body which funds research into “biomedical science”. The Wellcome Trust announced in the headline of their press release about this study that the study had found "first direct evidence that ADHD is a genetic disorder". This was not in fact even remotely demonstrated by the study. Furthermore, as we discuss, there are a number of findings in the study which do not

support the current ADHD narrative and which should call it into question. These were not amplified to the press.

In Section 2) we review the US Multi Modal study. (This study is sometimes referred to as the MTA study and we follow suit). This study compared “treatments for ADHD”. There were in total four “treatment” groups. An intensive “medication” “treatment”, a behavioural intervention, routine outpatient care and a programme which combined both intensive “medication” and the behavioural intervention. The study took place over 14 months. Behaviour (“symptoms”) was recorded mainly by parents and teachers. The MTA study is riddled with methodological flaws. However; this hopelessly flawed study is used world-wide to justify ADHD-drugging. It is probably the single most important pro-drugging study in the ADHD narrative. It used more subjects and was conducted over a longer period than most studies. The study was undermined when a follow-up study conducted at 36 months using the same subjects and measurement methods found that the benefits claimed in the first study for stimulant drugging over behavioural treatments had faded away. The reactions to this finding by some of the researchers as well as by the authors of the UK's NICE Guideline on ADHD are illuminating. Having uncritically accepted the flawed methods in the original study which managed to produce the right result this second result is now subject to extensive re-evaluation and detailed criticism in an attempt to reverse its findings or at least limit the damage. These gymnastics illustrate how in the ADHD narrative “science” will be used to produce the right result (drugging) whatever the research actually shows.

In Section 3) we review the drugs used to “treat” ADHD. We consider the harms that they do and the ever increasing revenues that they generate for their manufacturers. We note that, surprisingly, they are prescribed despite it being acknowledged (in the case of the two main drugs used in the UK at any rate) that there is no clear scientific-medical explanation for how they are supposed to “work”. In as much as they do “work” the claims relate entirely to claims of “symptom reduction”. But in ADHD a “symptom” is not a medical problem which someone suffers from. It is a behaviour which is “disruptive and inappropriate for developmental level” (DSM-IV). The well documented harms that ADHD drugs routinely cause young people are acknowledged, though minimised, by enthusiasts for drugging.

In Section 4) we consider the ADHD Guideline issued by the UK's National Institute of Clinical Excellence in 2009. The document and its recommendations were produced for NICE by the National Collaborating Centre for Mental Health. The National Collaborating Centre for Mental Health is a partnership between The Royal College of Psychiatrists and The British Psychological Society. The document offers recommendations which provide for both drugging and behavioural treatments to be used “for ADHD”. Strikingly the document offers very little “evidence” to support its recommendations. The behavioural treatments recommended by the Guideline are the provenance of members of The British Psychological Society. The drugs which are called for are prescribed by members of The Royal College of Psychiatrists. These two professional membership

groups appear to have essentially divided up the work between themselves.

In Section 5) we attempt to situate the ADHD narrative in its social context. Who are the interested parties in the narrative and what are their relationships? What happens when the media asks awkward questions?

## 1) The genome study; a study in the misuse of science

“Rare chromosomal deletions and duplications in attention-deficit hyperactivity disorder: a genome-wide analysis” (Nigel M Williams *et al.* 2010) was published in the Lancet in September 2010. [1] The work was carried out at Cardiff University. This study is significant because it is used to lend weight to the “ADHD is a genetic disease” narrative.

i) The construction and findings of the study

The study we are reviewing here is a genome-wide association study. Studies of this kind attempt to find correlations between certain traits and genetic factors. The study took a group of 410 “ADHD children” and used genetic analysis methods to count the number of large chromosomal abnormalities, of a certain kind, they had. The study authors then compared these findings with a dataset of controls based on the population at large.

The chromosomal abnormalities under consideration are large chromosomal deletions and duplications. Deletions are when there is a bit of genetic material missing on the chromosome. Duplications are when a section of genetic material is duplicated on the chromosome. The researchers call these two types of abnormalities collectively “CNVs”. Once they had excluded some subjects where the genetic analysis had produced unreliable results the researchers were left with 366 “patients”. The rate of CNVs in this group was compared with a data set from a group of controls supplied by the Wellcome Trust. The control data set was from a cohort of people born in 1958. Overall the researchers found that there was a higher rate of CNVs in the “ADHD” group than in the control group. Rate here is a statistical term. It is total number of CNVs / number of people. Thus it is the average number of CNVs per person. This is a type of analysis in which persons are eroded from sight beneath mathematical sums. The figure thus obtained does not relate to any one individual. Does the methodology give us a clue as to the orientation of the study? Does the “rate” calculation have any medical application? Or is it concerned with assembling numeric proof of difference between the two groups? The researchers also supplied “clinical data”. The “clinical data” recorded the number of individual human beings with one of more large CNVs. In the ADHD group there were 50 out of the total of 366 with one of more large CNVs. In the controls 75 out of 1047. That is 13.6% of the “ADHD children” had one or more large CNVs and 7.2% of the controls. (Using the alternative more statistical approach; in the “ADHD” group the figure was an average of 0.156 CNVs per person compared to 0.075 in the controls). Whichever counting approach is used this is a statistically significant difference using reasonably large sample groups. On the basis of this finding the authors concluded that:

> Our findings provide genetic evidence of an increased rate of large CNVs in individuals with ADHD and suggest that ADHD is not purely a social construct. [1]

The study also found that:

> CNVs identified in our ADHD cohort were significantly enriched for loci previously reported in both autism (p=0· 0095) and schizophrenia (p=0· 010). [1]

The study included further data from Iceland. The researchers stated that the findings were replicated in this data set.

The interpretation of the genome study researchers that “ADHD is not purely a social construct” is somewhat surprising. This is not a report of an empirical result or a scientific claim. It is a political claim. In fact what they have done is show that it is possible to statistically correlate possession of an ADHD label with the genetic factor they studied. Given what is known about the links between behaviour, environment and genes this is not all that surprising. We would expect that if people are divided into groups based on behaviours there would be some genetic correlates. The specific genetic factor chosen was probably chosen because it was a strong candidate for statistical correlations. Arguably the surprise is that the significant genetic variant was only found in a small percentage of the ADHD group.

What is the purpose of showing that the ADHD label or “diagnostic category” of psychiatry can be shown to have a genetic correlation *statistically across large groups?* Precisely because the finding is a statistical one across groups and not related to individual clinical pathology the finding has little use in terms of treating any individuals for any actual disease. It can however be used to develop a wider political narrative about some people “having a genetic disease”. The way they articulate their findings politically rather than empirically shows that the study authors saw providing support for the ADHD narrative as one of their mission objectives.

The researchers broke down their findings into two groups: one of “ADHD” people with an IQ of less than 70 and one for those having an IQ of 70 or above. Having an IQ < 70 is an accepted (though arbitrary) definition of “intellectual disability”. The researchers found a significantly higher rate of CNVs in the ADHD group with an IQ < 70 than with an IQ >= 70. In the ADHD group with an IQ of less than 70 36.36% of people had one or more large CNVs. In the ADHD group with an IQ of 70 or greater 11.41% had one or more large CNVs. The average IQ for this group was 89. In the control group 7.2% had one or more large CNVs. The average IQ of this group is, by definition, 100. These findings raise the question whether the kind of behaviours which may get an “ADHD” “diagnosis” may not be a secondary “effect”. The CNVs “cause” low IQ and in turn this leads, in certain social contexts, to impulsive/inattentive behaviour. We discuss this matter in sub-section vii) below.

In the following we discuss a number of the points of interest of this study. We do not question the

essential finding of the study, that a statistical correlation can be established between possession of an ADHD label and a certain genetic abnormality. However we should be cautious about what has actually been “found”. Statistical correlations do not make a disease as one of the study authors appeared to claim.

ii) Professor Thapar's stories

That the purpose of the study was to provide material to support a certain narrative is borne out by how it was promoted to the press. One of the researchers on the project gave a series of interviews to the press in which she made a series of fictitious claims about her study and what it shows. This was Professor Anita Thapar. For example, Professor Thapar told the Independent:

> Now we can say with confidence that ADHD is a genetic disease and that the brains of children with this condition develop differently to the brains of other children. [2]

And, the Wellcome Trust press office:

> Now we can show people that these children have a neurodevelopmental disorder with an observable genetic contribution. [3]

The claim made here by Professor Thapar about a “genetic disease” which effects “these children” is simply wrong and was in no way established by her study. Taking her statement at face value any member of the public would be forgiven for thinking that the study has established that every “child” “with” “ADHD” has a genetic condition. The study has not shown anything of the sort. She is making all this up. She makes these claims to the press outside a context where they would be subject to peer review but she claims the weight of her peer-reviewed study to justify them.

The Independent headlined their report:

> Bad behaviour down to genes, not poor parenting, says study [2]

This is fiction. It would appear though that this was the kind of headline which Professor Thapar was aiming at. Recall; the study simply showed that somewhat more of the “ADHD group” possessed the deleterious genetic variant than the control group. The figures were 14% in the ADHD group and 7% in the control group. Even in the 14% of the ADHD group who possessed one

or more large CNVs the study did not show a causal pathway. The study did not even show that everyone who possessed this particular genetic variant would be unusually inattentive/impulsive.

Many press outlets simply repeated the stories put out by Professor Thapar. The Daily Mail's headline for this story could have been written by Professor Thapar:

> ADHD is 'in a child's genes' as scientists provide hope to ending bad behaviour stigma. [4]

The Guardian published a piece by their Health Editor headlined "Hyperactive children may suffer from genetic disorder, says study" and the Health Editor informed her readers that:

> But today the furore around ADHD moves into a different space. Researchers, funded not by drug companies but by the Wellcome Trust and other bodies, are publishing the results of a study which for the first time identifies genetic changes in children diagnosed with ADHD. [5]

The detail that the correlation was only found in a percentage of the ADHD group is mentioned half-way down the page in this article. The above statement from the Guardian's Health Editor that the fact that the study was (part) funded by the Wellcome Trust ("not a drug company") somehow ensures its reliability is naive to the point of absurdity. Firstly; a study can be assessed for its scientific merit on the contents of the paper; it doesn't matter who funded it. (The contentious point about funding by pharmaceutical companies is how it can distort the literature as a whole because more studies which are linked to commercial products are carried out than ones which are not linked to commercially exploitable products). Secondly; the Wellcome Trust is a not-for-profit with roots deep in the pharmaceutical industry. It appears to fund research which lends itself to supporting the ADHD-drugging narrative. (Section 5) ii)).

The BBC provided a rare critical commentary in this sea of uncritical journalism. This is the BBC's Medical Correspondent:

> There is a danger of reading too much into new research in the Lancet on attention-deficit hyperactivity disorder (ADHD). The headline of the Lancet press release says: "Study is the first to find direct evidence that ADHD is a genetic disorder". One of the authors, Professor Anita Thapar is quoted as saying: "Now we can say with confidence that ADHD is a genetic disease and that the brains of children with this condition develop differently to those of other children". That's that then. Or perhaps not. Because those bold claims do not seem to be borne out by the actual research paper.

[6]

In her narrative to the press Professor Thapar does not appear to have made it clear that in fact only 14% of the young people in the study had the deleterious genetic variant. Time and time again, in the press she gave, she gives the impression that the study has shown that *all* young people labelled “ADHD” “have a neurodevelopmental disorder” or “a genetic disease”. Furthermore; she does not make it clear that even in the case of the 14% who did have the deleterious genetic variant no direct causal link has been established. The talk about “a genetic disease” and “Now we can show people that these children have a neurodevelopmental disorder with an observable genetic contribution.” [3] is pure fiction. It is quite hard to believe that Professor Thapar was simply making a number of mistakes. It seems more likely that she was simply telling stories. Her claim for a “genetic disease” is no more accurate than saying, based on a small statistical correlation between fish oil and less chance of '”getting ADHD” (which has also been established by studies which look for statistical correlations [7]), that “ADHD is a dietary disease caused by lack of fish oil”.  Or, indeed that hyperactivity is a disorder related to food additives based on studies which show that food additives are linked to hyperactive behaviour in young people. [8]

It seems to be the case,  based on a review of several press reports (The Independent, The Daily Mail, and the Guardian) [9][10][11], that Professor Thapar has kept largely quiet about the fact that there was also a strong correlation between IQ scores and possession of the deleterious genetic variant in her study. She does address this point in an interview with the Wellcome Trust. However here too she provides a misleading interpretation of the actual results of her study. Thapar claims (correctly) that the association was strong in the group with an IQ less than 70, but does not acknowledge that the correlation between IQ and possession of large CNVs stills exists even when this group is removed. See sub-section vii) below for a full discussion.

What these statements to the press show is that some at least in the academic community are so wedded to a certain narrative that they will tell stories. The Wellcome Trust, who part-funded the study, took up and amplified Professor Thapar's stories to the world. [12] The Wellcome Trust initially headlined their press release about this research, which they helped to fund, with the headline "Study finds first direct evidence that ADHD is a genetic disorder". They subsequently changed the headline for the article to: "Study finds first direct genetic link to ADHD". This can be confirmed by using the Wayback machine [13], an Internet service which keeps previous copies of web pages, and comparing this with the current page [12]. Whether this change (from something which is not true to something which could be defended) was made in response to this author's email correspondence with Dr Nigel Williams one of the genome study authors, and, separately, with the Press Department of the Wellcome Trust we cannot say. In many ways though the damage was done and the headline which makes the fictitious claim of “genetic disorder” was

widely repeated in the medical press around the world as a “scientific discovery”.

Unfortunately the misleading claims by Professor Thapar have been taken up by people who deserve to be better informed by publicly funded scientific research. One ADHD support group, Adders.org, put out a release saying:

> This is indeed extremely welcome news of clear evidence to confirm that attention-deficit/hyperactivity disorder (ADHD) is indeed a brain development disorder with closer links to autism than was previously thought. I hope this will be a welcome relief to the many families who have to face criticism and ridicule on a daily basis, when trying to explain the behaviour of their ADHD child. I hope also that many adults with ADHD, will feel much better knowing that their condition wasn't something to do with their upbringing or diet. Extremely low self esteem is probably the biggest common factor in those diagnosed with ADHD, both children and adults. Now we can point to proof that it is a neurodevelopmental disorder. Let us hope that this leads to a better understanding and treatment for children and adult sufferers alike. [14]

The headline on this piece was:

> It's Official! ADHD Is A Genetic Disorder [14]

The a genetic disease
enome study does not “confirm” anything even remotely close to the wishful thinking on display here. Possibly Professor Thapar is telling these people what they apparently want to hear, but it isn't science. Adders has received drug company funding [15]. The attentive reader will note a nexus of drug companies, The Wellcome Trust, an ADHD support group and the stories told by Professor Thapar. (As a side-note it does appear that diet can be linked to “symptoms of ADHD” [16]. Adders is embarrassingly eager to leap at the “genetic” narrative).

It seems that Professor Thapar has a “theory” about “ADHD” and eager to prove her “theory” made much more out of the actual findings of the genome study than they can in fact support. Here is Professor Nigel Williams, one of the lead researchers on the study, also talking to the Wellcome Trust:

> These findings are testament to the perseverance of Professor Thapar and colleagues to prove the often unfashionable theory that ADHD is a brain disorder with genetic links. [12]

This statement confirms that Professor Thapar is indeed persevering in trying to “prove” her theory. So persevering in fact that she didn't let the actual results of her study get in the way.

The explanation has been offered to this author in private email correspondence, with Dr Nigel Williams who led the study, that the large CNVs identified by the study should be understood as a “risk factor” which could determine the chances that someone might “get ADHD”. This is a welcome correction of Professor Thapar's wild claims, and indeed the statements Dr Nigel Williams made to the press were more accurate than those of Professor Thapar. The report of the study carried by Channel 4, for example, shows Dr Williams making it clear that the study showed a statistical correlation and not an explanation of a “disease”:

> Children with ADHD have a significantly higher rate of missing or duplicated DNA segments compared to other children and we have seen a clear genetic link between these segments and other brain disorders. [17]

However even this statement is potentially misleading. 76% of the “ADHD” young people in the study *did not* possess a large CNV. They are completely in the clear. They do not possess the factor in question. Dr Williams may perhaps be thinking of the rate calculation which we discussed above. If you divide the overall number of CNVs in a group by the number of people in the group then the ADHD group as a whole has a higher 'rate' of CNVs than the control group. Using this statistical method enables Dr Williams to make general claims about the ADHD group (in his study) as a whole and treated statistically. But in this statistical working of the empirical results individuals become nothing more than statistical abstractions. The empirical result was that in the ADHD group 14% of individuals possessed one or more large CNVs. And in the control group the figure was 7%. Looking at the results directly without the benefit of statistics makes it clear that the majority of young people in the ADHD group did not possess a large CNV. This makes it clear that the genetic factor identified in the study cannot be an explaining factor in the majority of cases. The statistical method makes it easier to generate the genetic narrative. But the public should be aware that this is a mathematical abstraction.

<u>iii) “ADHD” is still a construct however often they use the word “disease”</u>

“ADHD” is a term which has meaning in a psychiatric system of classification. Its “validity” as a genetic or biological disorder can be bolstered by findings which produce statistical correlations between groups of people so labelled and various physical factors. It is not a term of the same order as say “Cystic Fibrosis”, or lung cancer, terms which always refer to a physical condition. Someone does not “have” ADHD in the same way that they have Cystic Fibrosis. In the latter case the “having” refers in each and every case to something physical which the person really has,

which can be tested in a laboratory and is experienced by the person. None of these apply when a person is said to “have” “ADHD”.

ADHD was brought into being by being defined in the 1987 edition of the Diagnostical and Statistical Manual of Mental Disorders, the handbook of the American Psychiatric Association. There were forerunners. “Hyperkinetic reaction of childhood" was introduced in the 1968 edition. ADD (Attention Deficit Disorder) was introduced in the 1980 edition. As is the case with other psychiatric disorders ADHD is defined in terms of behaviour observed by a psychiatrist. Reports by parents and teachers can be taken in evidence. (Contribute to helping the psychiatrist make a “diagnosis”). Since many of the check-list points which form the “diagnosis” relate to compliance with adult instructions the system is one which contains the potential to be fundamentally unjust. Who is to define, for example, when not finishing chores (one of the DSM-IV criteria, see Appendix i)) is reasonable and when it is a “sign” of a “disorder”? Do all parents have the same expectations around “chores”? There is no scope within the diagnostic system to investigate whether the chores that are not finished were a reasonable demand. This is a sinister system which, like 18th century *lettres de cachet*, allows family members to put others away for non-compliance.

The attempt to find a biological basis for “ADHD” is a game of catch-up. The Cardiff genome study found that 14% of the “ADHD” subjects had one or more large CNVs, compared to 7% in the general population. The response of the authors of this study when questioned as to why they were making claims about a “genetic disease” on the basis of just 14% of their sample possessing the identified genetic factor is that the other factors have simply “yet to be identified”. For example; the Guardian reported:

> Although this finding was limited to 16% of all the children with ADHD, they say it is highly likely the rest have other genetic variants that have not yet been identified. [5]

See also this report in the Daily Mail [10] which reports the same argument. (The 16% figure in the Guardian report seems to relate to the statistical method of presenting the results. This divides the total number of CNVs found by the number of people in the group to produce a statistical “rate'” figure. The statistical figure produces a slightly higher figure than the clinical figure which counts the number of individuals with one or more large CNVs (14%). 15.6% has been rounded up here to produce 16%).

Right now only 14% of the “ADHD” group had the significant genetic variant tested for and studies testing for other genetic variants have yet to be carried out. If the “condition” is the result of a range of genetic factors then it is *still* a construct. It is a label which is then back-filled by statistical studies to give it some “validity”. Any correlation at all is taken as providing that validity. At the moment there is no evidence for the other genetic factors which the authors confidently expect to

be identified. The claim that in time other genetic correlating factors will be found to fully or nearly fully explain “ADHD” is unscientific. It is a proposition which cannot be falsified. This is akin to claiming that pink giraffes exist somewhere on the planet. Such a proposition could in theory be proven, by finding one. It cannot, however, ever be disproved. Genome wide association studies work by identifying and counting genetic factors of some kind and contrasting the count between the control group and the target group. To prove that other genetic factors are not involved would involve producing a list of all possible candidates and testing for each one. That list is open-ended. Those who make the claim that “the other genetic factors exist but have yet to be found” are not making claims which can be be tested. In making these claims about other proposed genetic factors which will be found the genetics lobby is privileging its own role. It is inherently unlikely that the fabled “other genetic variants” will be found in anything even remotely approaching 100% of an ADHD group. Similar claims have long been made for schizophrenia, yet genetics research in this area has yet to produce anything like a clear-cut list of genetic variants which are always associated with a diagnosis of “schizophrenia”. It is a reasonable hypothesis that *some* other genetic correlates will be found. It is not scientifically plausible to say that “the rest” will *all* be found to have a genetic factor.

Even in its own terms the genetic story is too simple. Even when there is evidence of a genetic correlating factor the determinant can be an interaction between genes and environment. (I.e. a person could have this genetic variant but if their environment were different they would not manifest the behaviour). Furthermore; there are a range of possible environmental causes for inattentive/impulsive behaviour which may or may not interact with a genetic predisposition. For example; diet [18], use of stimulant drugs, maternal abuse of drugs, lead poisoning, and mild closed head injuries. [19]  There is also a statistically significant association between the mother's consumption of fish during pregnancy and the likelihood of her child being seen as exhibiting ADHD behaviours by a teacher.  (More consumption of fish reduced the chances of an ADHD diagnosis). [7]  Given the range of possible correlations it becomes clear that even as a category of psychiatry “ADHD” is doubtful.  A construct which can be linked in some cases to environmental factors such as fish oil, IQ, maternal drug abuse, head injuries and to a genetic factor (though not to any one specific missing or damaged gene) is not a “genetic disease”.

The Cardiff researchers claim that showing a statistical link between possession of an ADHD label and a certain kind of genetic damage shows that ADHD is “is not purely a social construct”. But this is not the case. ADHD is “purely a social construct”. It does not exist in nature. It was brought into being in DSM-IV by the American Psychiatric Association in 1987. If the Committee hadn't decided to include it it wouldn't “exist”. What the Cardiff researchers mean is that this “social construct” can be correlated, statistically, to some genetic factors. Therefore it isn't a complete phantasy. They are saying that the behaviours that fall under an “ADHD” umbrella in some cases have a genetic background. To anyone who knows that there is a correlation between biology and behaviour this is no surprise. Nonetheless ADHD is a “social construct”. It doesn't need to exist. Other sets of

behaviours could be identified and labelled and linked to genetic factors but are not. The institution of psychiatry has created the label “ADHD”.

It has been demonstrated that in some instances of ADHD labelling the whole thing is in the imaginations of school-teachers and psychiatrists. It has been demonstrated that young people who are young for the class are more likely than their peers to receive an ADHD “diagnosis”. This finding has been repeated in three separate studies. [20][21][22] This finding shows conclusively that the “ADHD” label is not 100% linked to genetic factors, as the Cardiff researchers apparently propose. Unless, that is, we are to suppose that genetic variations are not evenly distributed across all birth dates. (Which would suggest a belief in astrology). The Cardiff researchers' dream of “the rest having other genetic variants” has *already* been comprehensively disproved. We look at these studies in more detail in sub-section vi) below.

The authors of the Cardiff genome study have assumed that something called ADHD exists. They referred to “children with ADHD” and “participants with ADHD” no less than 27 times in their paper. But this supposed ontological, actually existing, condition was not established before the genome study and was not established by it. The Cardiff researchers offer statistical correlations not accounts of disease pathways. They have researched correlates between possession of a label (which was awarded without a biological test) and a genetic factor. They have not researched a physical condition called “ADHD” as they appear to believe they have done.

iv) Removing stigmatization

In statements to the press the authors of the genome study claimed that the findings would “help overcome the stigma associated with ADHD “. [12] This claim makes Professor Thapar and her colleagues appear in the role of knights in shining armour riding to the rescue. Part of the reason for making this claim may be to counter the obvious criticism of the study that it has no clinical purpose. Its purpose, as we have discussed above, appears to be political. It appears aimed at developing the narrative that “ADHD is a genetic disease”.

The argument about stigmatization seems to be that there is a stigma arising from a perception that inattentiveness/impulsivity is due to bad parenting which the parents “of ADHD children” suffer from. Unless the Wellcome Trust Press Office has mixed up the quotes the implication therefore is that the stigma which Professor Thapar thinks she has helped removed is the one that apparently attaches to parents. It is not, as one might have expected, the one which attaches to the young people who actually have to carry the label:

> We hope that these findings will help overcome the stigma associated with ADHD," says Professor Anita Thapar. "Too often, people dismiss ADHD as being down to bad

> parenting or poor diet. As a clinician, it was clear to me that this was unlikely to be the case. Now we can say with confidence that ADHD is a genetic disease and that the brains of children with this condition develop differently to those of other children. [12]

In other words; the children really are bad. Faulty goods to the core. The parents cannot be blamed. There is some faulty reasoning here though. If only 14% of the subjects in the study possess the relevant genetic characteristic how, rationally, does that in fact remove the stigma from these parents? Assuming for a moment that such a stigma exists and it is the job of geneticists to remove it. Does a stigmatised ADHD parent say “well, there is a 14% chance that my child has a genetic variant which is associated with inattentive behaviours so you can't stigmatise me”? It is totally absurd.

The proposal by Professor Thapar about “removing stigmatisation” suggests some polarised thinking. The thinking appears to be that “ADHD” is either down to “genes” or “bad parenting”. Professor Thapar seems to think that the public believe 'it' is down to “bad parenting” and hold the parents of ADHD children in low esteem as a result. She then, with her genetic study, is showing the public, on behalf of ADHD parents, that in fact “ADHD is a genetic disease” and thus 'it' is not down to “bad parenting”. It seems the basic dynamic is a proposal that ADHD parents who are being “stigmatised” are being invited to improve this with the comforting thought that their children really are genetically faulty. In fact though it does not appear to be the case that the public holds in large numbers to this “bad parenting” “causing ADHD” narrative. At least the comments threads when the online press covers ADHD stories tend to divide into those expressing scepticism about the label, a minority who think that the problem is a lack of discipline in modern society and a few strident believers in “ADHD”. The “bad parenting” narrative appears to be largely a creation of Professor Thapar. Possibly some of the parents she meets in her clinical practice (Thapar runs an ADHD clinic) really do report that they feel that other people blame them for being “bad parents”. But, shifting the blame onto the children, is not a real solution to such feelings. It should be possible to reassure parents that they are not “bad parents”, if indeed they aren't, without pinning a “faulty” sticker onto their child.

The serious stigmatisation in the ADHD story relates to the experience that ADHD labelled young people have. Once labelled they are marked out as different from their peers. The ADHD label may well make it harder for them to make friends. Being labelled as different is not an easy experience for a young person. That is why discipline systems in schools have historically used just such marking out as different as a technique of punishment. (Dunce's cap, ADHD label).

Finally, it should be added that if there is a stigma associated with the diagnostic category of ADHD the simplest way to resolve the problem would be to stop labelling young people. Thapar is blind to the role that psychiatric labelling plays in creating ground for stigmatization.

v) More effective treatments

Apart from the “removing stigmatisation” claim the other claim which Professor Thapar makes for her study is that it will lead to “more precisely tailored treatments”:

> These aren't the sort of findings that will lead to a test for ADHD. We already have that - the best method for diagnosis at present is to ask the right sorts of detailed, careful questions. But this type of research might help us to refine our diagnoses or define meaningful subgroups. Most importantly, the results can help us understand the causes and biology of ADHD, which can suggest how it might be treated. At the moment, we only have a limited range of treatments available; but if we can understand what is happening in the brain during the development of ADHD, we might be able to develop more precisely tailored, more effective treatments. [23]

Once again though Professor Thapar is confused. If there is no test for ADHD, and she is at pains to explain that her study will not lead to a test, on what basis would these new “treatments” be distributed? You cannot rationally suggest that a treatment should be given “for ADHD” based on a survey which found 14% possessed a certain trait and at the same time declare that no test which could establish whether or not someone was in that 14% be developed. A “treatment” developed on the basis of this study but then administered to all young people with an ADHD label would be like playing Russian roulette with 5 bullets in a 6 chambered revolver. Five young people would get a treatment they didn't need, with all the “side-effects” (see Section 3) v)) that might entail, for each one who got the “precisely tailored” treatment.

Possibly one reason why Thapar is keen to emphasise that her study should not lead to a test “for ADHD” is were it to form the basis of a test the numbers “diagnosed” would drop overnight to 13.6% of their current levels. (Not even the 15.6% produced by the rate method; see sub-section i) above).

The genome study has not shown that the identified genetic variant is other than correlated statistically to possession of an ADHD label. It has not shown a causal pathway. And in fact the evidence from the study is that in the 14% with the statistically relevant genetic variant IQ plays a significant part too. Is Thapar planning to “treat” low IQ? Even leaving those considerations aside Thapar does not explain what kinds of “treatments” are available for chromosomal duplications and deletions. The “more precisely tailored treatments” are not specified. Is she proposing gene therapy for young people identified as “having ADHD”? That would surely imply a test? In fact; it is more likely that talk of these mysterious “more precisely tailored treatments” is simply fiction.

In general terms *statistical* studies which show correlations between a physical factor and a certain behaviour trait cannot lead to treatments. Highlighting difference statistically is not medically useful. Again, then, the question of the purpose of this kind of study (statistical correlations at the genetic level) is raised. In the absence of any other clinical derivatives from this kind of study the suspicion must be that the main purpose is to develop the biological narrative about “ADHD”, which in turn is aimed at legitimizing drugging. Certainly the narrative about “While the exact cause of ADHD is as yet unknown, it is generally accepted that it is likely to be biological in nature” is often found in close proximity to the narrative about “chemical imbalance” and in turn to the narrative about “the benefits of the drug.” (See for example this folksy leaflet about ADHD from Gateshead Council: [24]).

vi) Teachers' perceptions

Research has indicated that teachers' perceptions play a role in leading to an “ADHD” “diagnosis”. It has been shown by three separate studies that age in class is a significant risk-factor in being labelled “ADHD”. We mentioned these studies in sub-section iii) above. Two of these studies were published in the September 2010 issue of the Journal of Health Economics [20] [21]. The other was published in the Canadian Medical Association Journal in 2012. [22] The three studies have independently confirmed that there is a significant correlation between ADHD diagnosis and age in class. All three studies also showed that this correlation extended to likelihood of being drugged. The results of these studies present a very different narrative to the narrative about “ADHD” being developed by Professor Thapar. They conclusively show that, contrary to the claims apparently made by the genome study authors [5], it will not be the case that a fully “genetic explanation” will ever be produced to account for the ADHD label.

The correlation with age was significant. For example in Richard L. Morrow *et al.* 2012. [22] the study found that boys who were born in December were 30% more likely to receive a “diagnosis” of ADHD than boys born in January. Boys born in December were 41% more likely to be given a prescription for a “medication” (drugged) than if they were born in January. The figures for girls were 70% more likely for the “diagnosis” and 77% more likely for drugging. The reason is that birth month determines which year group a student joins in school and thus whether they are young for the class or old for the class. Those young for the class were likely to be “misdiagnosed” as “having” “ADHD”. Their age appropriate behaviour is misread as “symptoms of ADHD”. In Todd E. Elder 2010 the finding was that young people in the fifth and eighth grades were “nearly twice as likely as their older classmates to regularly use stimulants prescribed to treat ADHD”. [21] This latter finding is on a par with the strength of the genetic correlation found in the Cardiff genome study.

Speaking about his paper “The importance of relative standards in ADHD diagnoses: Evidence based on exact birth dates” [21] Dr Todd Elder, Assistant Professor of Economics at Michigan State University commented:

> If a child is behaving poorly, if he's inattentive, if he can't sit still, it may simply be because he's 5 and the other kids are 6. [25]

The findings from the two studies published in the Journal of Health Economics studies are summarised here. The italics are mine:

- ADHD diagnoses are driven by subjective comparisons across children in the same grade.
- The youngest children in school are twice as likely to use Ritalin as older children.
- *Teachers' perceptions are the mechanisms that drive these relationships.*

Todd E. Elder 2010 [21]

- Rising rates of ADHD have lead to the concern that ADHD is often misdiagnosed.
- We find evidence of medically inappropriate ADHD diagnosis and treatment in school-age children.
- Children younger than classroom peers have significantly higher rates of ADHD.
- *Age relative to peers directly affects a child's probability of being diagnosed with ADHD*.

William N. Evans, Melinda S. Morrill, Stephen T. Parente. 2010 [20]

Recall at this point how behaviour in class is a critical part of the ADHD diagnosis and how teachers play a role in the “diagnosis”. NICE specifically recommends this:

> While universal screening of the school population is not recommended, teachers may benefit from receiving some training to help them spot children who are suspected of having ADHD in order to initiate referrals and to implement support packages at the earliest possible stage. [26]

and

> Tier 1 professionals (including healthcare professionals and teachers) working in settings where children at high risk of ADHD might present should consider the possibility of ADHD. [27]

and

> A diagnosis of ADHD should not be made solely on the basis of rating scale or observational data. However rating scales such as the Conners' rating scales and the Strengths and Difficulties questionnaire are valuable adjuncts, and observations (for example, at school) are useful when there is doubt about symptoms. [28]

The studies cited were conducted in America and Canada. However given the role of teachers in "spotting" "children with ADHD" recommended by NICE it seems likely that a similar picture would be found in the UK too.

It is interesting to note that two of these three studies were published in September 2010, in the same month that the genome study was published. The genome-wide association study, according to its lead author Dr Nigel Williams, showed "Children with ADHD have a significantly higher rate of missing or duplicated DNA segments compared to other children" [17]. The papers, which were published in the Journal of Health Economics, showed "Children younger than classroom peers have significantly higher rates of ADHD" (William N. Evans *et al*.) [20] and "ADHD diagnoses are driven by subjective comparisons across children in the same grade" (Todd E. Elder. 2010) [21]. The genome study gained, as we have seen, considerable media traction. But there were no headlines screaming "Studies show that ADHD depends on teachers' perceptions".

<u>vii) IQ as a covariant</u>

The Cardiff genome study was a genome-wide association study. In studies of this kind which seek to identify a single specific factor which is linked to the trait being studied it is sometimes necessary to identify other factors, which could be the actual explaining element. These other factors are called co-variants. To obtain the evidence of a link between the being tested factor and the trait in question co-variants need to be controlled for. A simple example illustrates this point, as follows. When constructing a study to evaluate the effects of smoking on life-span by comparing a group of smokers with a group of non-smokers it might be necessary to control for alcohol consumption too. The study might show that smokers are more likely to die 10 years earlier than non-smokers, but if it is the case that the group of smokers also consume twice as much alcohol as the control group then we could not be sure that it was not the alcohol consumption rather than the smoking which

was linked to the shortened life-spans. To show that smoking was the relevant factor alcohol would have to be identified as a co-variant and the test group would have to be selected to have the same alcohol consumption patterns as the control group. This is a standard practice for these kinds of studies. ADHD is known to be associated with low IQ. (On average). The IQ data for the ADHD group in the genome study confirms this. (The average IQ score for the whole ADHD group was 86). This raises the question as to whether the genome study should not have treated IQ as a co-variant and controlled for it. Without controlling for IQ it can be argued that IQ rather than the genetic factor is the explaining factor for the ADHD behaviours.

I put the point to Dr Nigel Williams, one of the authors of this study, that IQ should have been treated as covariant. His response was to direct my attention to a paper "Why IQ is not a covariate in cognitive studies of neurodevelopmental disorders" (Dennis M. *et al*. 2009). [29] This paper argues firstly, in a section titled “The Historical Reification of General Intelligence”, that IQ is not a measure of anything. It is simply a measure with a questionable political history that one assumes, wrongly, to be a measure of something objective. Given that the ADHD narrative is built-up with reification upon reification there is considerable irony in being referred to a paper which presents an argument about reification in the context of defending ADHD. However, the covariate paper accepts that this argument about IQ not being an objective measure may not be 100% convincing. After all IQ probably measures something (even if it is not a single “thing”). The argument then moves on to a more complex discussion about whether IQ should be treated as a covariant in studies of “neurodevelopmental disorders”, such as ADHD. (Like all psychiatric papers relating to “ADHD” these authors too talk about the psychiatric label ADHD as if it refers to something which exists). The argument is that lower than average IQ is known to be a factor in learning difficulties and “neurodevelopmental disorders” in general and so to try to control for this factor would be to create an artificial group and would likely distort the findings from the study. The authors write:

> To the extent that IQ represents the same processes as the construct of interest, then controlling for IQ removes variability in the outcome measure that is directly related to the construct of interest. [30]

and

> Covariance analysis using IQ is usually predicated on the hypothesis that IQ “causes” the difference on a correlated variable (e.g., memory). When there is an inherent IQ difference between groups and the IQ difference is not separable from the level of the independent variable to which the patient belongs, the causal mechanism cannot be determined. The group difference in IQ remains a potential explanation for group differences on other cognitive measures and cannot be ruled out through statistical adjustment or explained away statistically, regardless of whether IQ is significant as a covariate or whether the differences on the dependent variables are significant. [31]

What these authors are saying, as applied to the case of ADHD studies, is that to control an ADHD group for IQ (perhaps by selecting an ADHD group with an average IQ of 100) would be to create an unrepresentative ADHD group. For this reason they suggest that the ADHD group should not be controlled for IQ. This advice appears to have been followed by the authors of the genome study. This means, though, that it will not be possible to say that it is not the low IQ which is causing the differences between the groups. As Dennis M. *et al*. say: “The group difference in IQ remains a potential explanation for group differences on other cognitive measures and cannot be ruled out through statistical adjustment ...“. In the case of the genome study this means that the authors cannot say that the large CNVs are the explanatory factor for the ADHD behaviours. Inattentiveness/impulsivity (“ADHD”) is confounded with low IQ and the exact causal relationships cannot be indicated by a study of this kind.

Had the authors of the genome study ignored this advice and controlled for IQ as a covariant they would have found much less of a difference in the rate of possession of large CNVs between the ADHD group and the control group than they did. Quite possibly they would have found no difference at all. The following table shows the different groups and the percentage of individuals in each group who possessed one or more large CNVs. This data is all contained in the study.

| **Group** | **Average IQ** | **Percentage of people with one or more large CNVs** |
|---|---|---|
| ADHD group with IQ < 70 | 60 | 36% |
| All ADHD group | 86 | 14% |
| ADHD group with IQ >= 70 | 89 | 11% |
| Controls | 100 [32] | 7% |

The table shows that in the overall ADHD group possession of one or more large CNVs is strongly correlated to IQ score. Notice also how the chances of possessing one or more large CNVs falls as IQ rises in the ADHD group. This data suggests very clearly that had there been an ADHD group with average IQ of 100 the percentage of young people in that group with one or more large CNVs would have been less than 11%, possibly quite close to the 7% in the control group. In which case no claim at all about “a genetic link to ADHD” [12] could have been made. These considerations make it clear that possession of an ADHD label is strongly related to IQ.

All this presents a dilemma for the authors of the genome study. If they control for IQ it seems likely based on the data from the study that they will not be able to make a claim for a “genetic link”, or if they can, one with only very slight statistical significance. If they don't control for IQ they will get the

correlation between an ADHD label and possession of the identified genetic variant they want, but cannot claim it is not also linked to IQ. They want to avoid controlling for IQ but still make claims that say that the identified genetic factor is directly related to the ADHD label and the relationship is not mediated by IQ.

The solution to this dilemma was effectively a ruse using the tried and tested psychiatric technique of dividing people into categories and making claims based on those categories. The ADHD group was divided into those “with intellectual disability” and those “without intellectual disability”. Professor Thapar explains in her interview with The Wellcome Trust:

> Also emerging from the study was the finding that the increased rate of CNVs in ADHD was not related to intellectual disability. “A proportion of people with ADHD do have intellectual or learning disabilities, so it could be that the CNVs we found are related to IQ, not to ADHD," she explains. "But we've shown that these CNVs are not just found in people with learning disabilities." [23]

It is true that the study showed that there was a higher rate of CNVs in the ADHD group “with learning disabilities” (IQ < 70) than in the general population. However it is *also* true that the ADHD group without “learning difficulties” had an average IQ score significantly lower than the general population (89). Professor Thapar does not mention this. This is probably because this points to precisely the result she is trying to avoid reporting. There is a strong correlation between inattention/hyperactivity, low IQ and possession on one or more large CNVs throughout the study. Thapar attempts to box the question away by creating an artificial group, based on a category of psychiatry, of those “with learning disabilities” in whom she can acknowledge the link. But the link is still present in the remaining group. However you divide up the data the study cannot show that IQ is not a factor. In fact Thapar is attempting precisely what Dennis M. *et al*. were at pains to point out cannot be done: “The group difference in IQ remains a potential explanation for group differences on other cognitive measures and cannot be ruled out through statistical adjustment or explained away statistically”. [31]

These observations open the way to an alternative interpretation of the study. It may be that it is IQ and not inattentiveness/hyperactivity which is linked to possession of one or more large CNVs. Young people who have a lower than average IQ struggle more in class. They appear “inattentive” because they are not following the lesson. They appear “hyperactive” because, bored, they start acting up or trying to find distractions. This interpretation is as consistent with the empirical findings of the genome study as the idea that large CNVs lead directly to inattentiveness and impulsivity. Possibly more so. On this basis “ADHD” is simply a label for people with low IQ who struggle in large classes. If this is the case then the “clinical implication” is quite clear. Unrealistic demands are being placed on young people with below average IQ. Rather than drug them to try to force

them to meet those demands the demands should be adjusted to what they can manage. This explanation is perhaps too simplistic. Nonetheless it is supported in broad terms by the study data and points towards one “solution” to the problem of hyperactive young people in school. And a solution which does not involve drugs.

In summary, the Cardiff genome study cannot determine if the genetic variant studied causes inattentive/hyperactive behaviours or whether it is linked to low IQ and the behaviours are a secondary effect. In her comments Professor Thapar attempts to create an impression that the correlation between large CNVs and an ADHD label is independent of IQ. The study authors want to do this so as to produce the “ADHD is a genetic disease” narrative or at least the “genetic link” narrative. But the actual data in the study directly contradicts this claim.

viii) Gender bias

The fact that IQ was strongly statistically correlated to the genetic factor studied, as well as ADHD behaviours, was not the only finding in this study which was not amplified to the press. The study reported that in the “ADHD” group there was no difference between boys and girls. Both genders carried the chromosomal duplications and deletions equally:

> In each of the ADHD and control samples, the rates of CNVs did not differ between male and female participants (data not shown; results available from NMW). [33]

This is a very interesting finding indeed. It is nothing short of astonishing that this finding found no prominence at all in the summary of the study or in Professor Thapar's round of press interviews.

In reality boys are much more likely than girls to be “diagnosed with ADHD”. It is difficult to obtain accurate figures for this. The NHS for example tracks the numbers of prescriptions issued (because of cost) but does not track who they are issued to. In general terms though there seems to be a consensus that boys up to nine times as likely to be “diagnosed with ADHD” as girls. [34] In the ADHD group selected for the Cardiff genome study there were more than six times as many boys as girls.

What does this result tell us? It more or less shouts at us that ADHD is largely to do with teachers in classrooms and parents in homes finding it harder to manage male young people than girls. It tells us that ADHD is what its critics say it is: more to do with the behaviour management requirements of schools than anything biologically “wrong” with young people so labelled. This is the case even if some biological correlates can be shown.

If the proposal is that “ADHD is a genetic disease” then the irrefutable corollary is that there is an absolute massive over-diagnosis of boys relative to girls. It might be argued that the gender imbalance in ADHD “diagnosis” or labelling reflects an “under-diagnosis” in girls. The NICE ADHD Guideline authors attempt just such a cynical escape with reference to the gender disparity in ADHD labelling. [35] Saying that girls are being “under diagnosed” however still leaves unexplained the *disparity* in rates of “diagnosis”. Whether you want to say that boys are “over-diagnosed” or that girls are “under-diagnosed” the disparity in diagnosis remains to be explained. If the biological factors are equally spread out between boys and girls then the explanation for the disparity has to be social or political.

(The suggestion might be made that the genetic factors which the authors of the Cardiff genome study propose exist but are yet to found are sex-related. However; this author has not seen the suggestion that any genetic correlation to inattentiveness/hyperactivity which may be found will be a sex-related genetic factor. As far as he is aware no one claims that “ADHD is an X chromosome disorder” like haemophilia).

Why did the study authors and the Wellcome Trust not lead with a headline that the study showed evidence of massive over-diagnosis of boys? That a statistical correlation was found associating some members of an ADHD group with a genetic abnormality is not surprising. The news is the finding that the link cuts completely equally across the sexes. This shows that the ADHD “diagnosis” is far more influenced by social and political factors than by “genetic” ones. Given how the diagnosis is made; referrals by parents and schools based on “disruptive" behaviour this is not remotely surprising. When the authors of the Cardiff genome study trumpeted “not purely a social construct” the unpublicised subtext was “almost entirely social construct”. The Cardiff genome study ignores the evidence that suggests that the construct is linked to social factors while amplifying the (much smaller) findings that do link the construct, statistically, to genetic factors. (As we have discussed above the ability to find correlating biological factors does not mean that the label is not a “construct”).

<u>ix) Quantitative genetic studies (twin studies) and the problem with the “genetic explanation”</u>

Professor Thapar is a believer in the genetic basis “of ADHD”. She wants to say with confidence that “ADHD is a genetic disease“. This is however an act of faith. In her interviews with the Wellcome Trust about the findings of the genome study Professor Thapar cites twin studies as existing evidence of the genetic “contribution”:

> We've known for many years that ADHD tends to run in families, so there is likely to be a genetic contribution," says Professor Thapar. "Over a decade ago, we studied identical and non-identical pairs of twins, and showed that ADHD is indeed highly

heritable, as people who have close relatives with ADHD are more likely to develop it themselves." [3]

ADHD twin-studies do not in fact provide clear-cut evidence for hereditary. Twin studies work in a somewhat complex way. Essentially they take a set of non-identical twins and produce a figure for the similarity of "symptoms of ADHD" (or whatever trait is being studied) between the twins across the set. (For example; if one twin "has ADHD" what are the chances the other twin also "has ADHD?"). Then they take a set of identical twins and produce the same figure for that group. Then they compare these figures. Non-identical twins are expected to share about 50% of their DNA in common and identical twins 100%. Therefore any greater similarity in the trait being studied in the identical twin set is attributed to genetic factors. For example; in a group of non-identical twins if one twin "has ADHD" there may be a 50% chance of the other twin also "having ADHD". In a group of identical twins if one twin "has ADHD" there may be a 75% chance of the other twin "having ADHD". The higher figure in the identical twin group is ascribed to their having a higher proportion of genes in common. This shows that the trait being studied has a genetic component. The higher the difference between the two figures the more genetic the trait is taken to be.

There are though a wide range of problems with these kinds of studies. A good review of the problems with twin studies has been published by the American Psychological Association. This paper, "A second look at twin studies", (Lea Winerman 2004) [36] reviews four assumptions which underlie twin studies. The most well-known and controversial of these is known as the "equal environments assumption". The assumption is that parents of identical twins will parent them (on average over a set) in the same way as parents of non-identical twins do their twins. If this is not the case, for example if parents of identical twins in general treat their twins both in the same way to a greater extent than parents of non-identical twins do then *this* might explain the greater similarity in the identical twin group, not the greater percentage of shared genes in this group. There is some evidence that this is the case. This assumption is therefore contested.

Another problematic assumption in twin studies from the point of view of "ADHD" is the assumption that people marry, or partner with, people who are different to them as often as with people who are similar to themselves. This is known as the "random mating assumption". It is on this basis that it is assumed that non-identical twins have 50% of their genes in common. This is an assumption. In fact the evidence is that people tend to marry others with similar traits. This is particularly so for intelligence. Intelligence is a key factor in "ADHD". Professor Thapar's own study shows this. Thus "ADHD" twin studies in particular should take account of non-random mating. If twin-study researchers have not taken this factor into account they may be claiming a greater genetic effect than the evidence warrants.

Another problem with twin studies is that in general they only consider the additive genetic

mechanism (mixing of genes producing a 'blended' and proportional result) and not dominant genetic mechanisms. In the latter case one gene “trumps” another; it is not a question of a blended effect. The mathematics of twin-studies is based on the additive genetic mechanism.

A fourth assumption which some twin studies make is to assume that a trait is the result of either genes or environment. They underestimate the complexities of gene-environment interaction. In some cases at least the propensity of “having” a given trait is a factor not of environment or genes but genes plus environment. A study which analyses the effect in terms of either a genetic influence or an environmental influence is over-simplifying. For example developing certain kinds of “inattentive” behaviours may be a factor of having a certain kind of genetic damage and attendance at a certain kind of school. If a twin study has not considered this and made sure that school attendance was equal in both sets of twins the study will be inaccurate.

Finally, it is worthwhile to notice that the base “data” for twin studies on “ADHD” is a subjective interpretation of behaviour not a physical fact. In some twin studies at least the behaviour is recorded by parents and/or teachers. This adds a further subjective element to the reported data which cannot be controlled for. The assumption will be that parents and teachers perceive and record in the same way for identical and non identical twins. But this may not, in fact, be the case.

In general twin studies are an example of how the use of over-simplified mathematical models superimposed on reality can be used to generate narratives.

The authors of the NICE ADHD Guideline are aware of some of the limitations of twin studies. They specifically mention the equal environment assumption and accept that it can be contested:

> A systematic review of 20 population twin studies found an average heritability estimate of 76%. In most cases, heritability in these studies is estimated from the difference in the correlations for ADHD symptoms between identical and non-identical twin pairs, as reported by parents and teachers: with the correlation for identical twin pairs in the region of 60 to 90% and for non-identical twin pairs being half or less than half of this figure in most studies (Faraone, 2005). Under the equal environment assumption for the two types of twin pairs, heritability can be estimated as twice the difference in the two sets of correlations.
>
> The assumption of ‘equal environment’ for identical and non-identical twins can be questioned. If it were not valid, then the estimated effect of genetic influences would decrease and that of shared environmental influences would increase. Even if this were to be the case, however, it would not argue against the validity of the disorder. It is not in doubt that twins’ scores are highly correlated – the level of ADHD symptoms in one child predicts that in the other. This tendency to run in families supports the idea that it

is a coherent syndrome, whether the reasons are genetic or environmental. [37]

(They also acknowledge the problem of complex gene-environment interactions).

The NICE authors hold to an account of “ADHD” which allows for “multiple genetic and environmental factors” [38]. It is not necessary for this pure form of the narrative to insist on a genetic explanation or link. Thus they are free to report some of the doubts around twin-studies. The account which privileges genetics and which insists on a “genetic disease” or “genetic link” can be understood as a strand within the wider ADHD narrative. This strand is less critical of twin-studies.

Twin-studies then tend to use a number of questionable assumptions. There is an argument that most or all of these assumptions can be controlled for and that more complex studies can take them into account. This. however, is not always done. Whether or not the twin-studies which Professor Thapar refers to have controlled for some of the variables mentioned above we cannot say. She made her claim about twin-studies in an interview and it is not referenced. Even if the studies she is referring to have managed to control for the equal environments assumption some of the other problems with twin-studies cannot readily be resolved. For example; it would be impossible to reliably produce a figure which took account of the variations caused by people partnering with people who are like themselves.

In any event, however these studies have been managed, it is not something called “ADHD” which can be shown (or not) to be heritable. Strictly speaking a diagnostic category of psychiatry is not a heritable characteristic.

The genetic narrative exaggerates the slender correlations that do exist between genes and impulsivity/inattention. It tends to assume causality and downplay the possibility of more complex environment-gene interactions. It assumes that more genetic correlations will be found but the evidence is that a purely genetic explanation for impulsive/inattentive behaviours will never be established. Indeed, as we have seen, there is compelling evidence that just being young for the class can get a young person “diagnosed” “with ADHD”. However; perhaps the main problem with the genetic narrative is that it lacks a medical application. Simply establishing that a percentage of young people “with ADHD” possess a certain genetic trait does not provide a basis for any kind of medical treatment. A medical intervention requires a) a test and b) a model of a disease pathway and c) an explanation of how the proposed treatment modifies the disease pathway. The genetic narrative on ADHD provides none of these things. The purpose of developing the genetic narrative is to support drugging. This is because if people can be persuaded that “ADHD is a genetic disease” then they are more likely to accept a biological intervention i.e. drugging.

x) Summary

The authors place on their study not a summary of their empirical findings but a political statement. They say that their results "suggest that ADHD is not purely a social construct". This is somewhat misleading. Their study has shown that 14% of young people in an ADHD group possess one or more large CNVs. For the other 86% the study has produced no evidence that *their* "ADHD" (possession of the label) can be correlated to anything genetic. For 86% of the sample group therefore the study does not suggest that *their "*ADHD" is anything other than a "social construct" in the sense in which the authors mean it. I.e. a label with no genetic correlation.

For the Cardiff researchers "ADHD" is a statistical category. Their claim about "not just a social construct" is true for the statistical category. But the term "ADHD" when it is ordinarily applied is not a statistical category. It is a clinical label attached to an individual by a psychiatrist in a consulting room. When "ADHD" is used in this way there is no biological test. In clinical terms the best the Cardiff study can say is that for any given young person with an ADHD label the probability that they will have one or more large CNVs is 14%.

The Cardiff genome study does not support the claim that: "Now we can say with confidence that ADHD is a genetic disease and that the brains of children with this condition develop differently to those of other children". [12] A process of inflation has occurred here.

The Cardiff genome study does not show a correlation between possession of the deleterious genetic variant in question and inattentiveness independent of IQ score. In the words of Dennis M. *et al.:* "The group difference in IQ remains a potential explanation for group differences on other cognitive measures and cannot be ruled out through statistical adjustment or explained away statistically, regardless of whether IQ is significant as a covariate or whether the differences on the dependent variables are significant." [31] The data presented in the study could equally well be interpreted as showing that the causal relationship is between IQ and the genetic variant. It is quite possible to construct an explanation based on the material in this study which would associate the large CNVs with IQ and see the ADHD behaviours as a secondary socially determined development. The authors do not develop this line of interpretation. On the contrary Professor Thapar sought to obscure the relationship between IQ and large CNVs which was present throughout the study.

The study found that the CNVs (duplications or deletions of genetic material) which were found tended to be in loci previously associated with "autism" and "schizophrenia". Scientifically this is probably the most interesting finding from the study. It was to be expected that some genetic correlation could be found. It is however news that this was in this loci. This at least helps to identify an area on the human genome which is associated with higher mental functioning. This does contribute to real scientific knowledge. But it was not this detailed and factual finding which

was amplified to the world. This may be because this finding does not help to construct the ADHD narrative. The finding that genetic variations and damage in a certain loci on the human genome are associated with problems with higher-order mental functioning, the ability to do mathematics and reasoning ability etc. is too general for that. Such a finding might lead to compassion. It does not lead to deliverable treatments. (See Section 5) i)). Psychiatry's preference for categorisation such as “learning difficulties”, “intellectual disability”, “ADHD”, “autism”, “schizophrenia” requires that you show correlations which link specific factors with the specific categories. It is clear that a specific aim of the study was to: “prove the often unfashionable theory that ADHD is a brain disorder with genetic links”. [12] The study aimed to support the biological strand in the ADHD narrative, not illuminate an area of human experience.

## 2) The Multi Modal study

The study was published as “A 14-Month Randomized Clinical Trial of Treatment Strategies for Attention-Deficit/Hyperactivity Disorder” in the Archives of General Psychiatry in 1999. [1] In keeping with other commentators we will refer to this study as the MTA study.

1) Introduction

The MTA study was sponsored by the US NIMH (National Institute of Mental Health). The NIMH is a US government agency concerned with research into mental illness. The MTA study was one of the largest and longest running ADHD trials. It makes a significant contribution to the ADHD narrative. When the NICE Guideline authors looked for studies they could accept to review for comparing drugging with behavioural interventions the MTA study had more subjects than all the others combined. (Section 4) iv)).

The MTA study gave different “treatments” to 4 groups of “ADHD children” and compared the results. The comparison was based on a statistical treatment of questionnaires completed, in the main, by teachers and parents. Classroom observers completed questionnaires for some measures and the “ADHD children” for one and their peers for another. The questionnaires were used to measure “ADHD symptoms” and some other factors such as “social skills” and anxiety/depression. On some scores “medication” outperformed the behavioural programme in the study.

This is summarised:

> For most ADHD symptoms, children in the combined treatment and medication management groups showed significantly greater improvement than those given intensive behavioural treatment and community care. [1]

Parents and teachers agreed that “medication” was more effective than the behavioural approach for the “symptom” of inattention. Parents but not, apparently, teachers agreed that “medication” was better than the behavioural programme at reducing the “ADHD symptom” of hyperactivity. The neutral classroom observers did not report a “benefit” for the “medication” regime over the behavioural approach in terms of classroom behaviour. The young subjects themselves did not report a benefit for “medication” on the score they were consulted on. There was some evidence then that methylphenidate can potentially be “superior” to a behavioural programme at improving attentiveness. This is in fact not news. Stimulant drugs (methylphenidate is a stimulant) are effective at improving attentiveness. At high dose, as in the MTA study, they may do this better than

a behavioural programme. Though it is significant that this score was not supported by the neutral classroom observers. The MTA study was conducted over 14 months. A follow-up study conducted at 36 months failed to confirm the initial results. (Sub-section vii) below). The MTA study does not consider the merits of drugging young people aged 8 (the average age of subjects in the study) to achieve improved attentiveness.

The MTA study is, from a scientific point of view, deeply flawed. The difficulties include: a) the imposition of a medical vocabulary of “symptoms”, “treatment”, “medication” etc. on operations which relate to “children’s” behaviour, rather than any medical condition, b) the use of interested parties such as parents and teachers to record the claimed “reductions in symptoms”, c) with one exception, the exclusion from the scoring of the young people's views, d) results which failed to support the drugging position were not given any weight in the results; only the positive results were reported, e) the failure of the blinded and neutral classroom observes to report a pro drugging result was not given the consideration it should have been, f) the fact that stimulant drugs were only tested on “ADHD” young people, disguising the fact that they have the same effect of improving attentiveness for all young people, and g) the usual reifying language of “ADHD children” is used. All the subjects are assumed to “have something”.

The MTA study clearly aimed at generating a result which can be used to add to the ADHD narrative a claim that “medication is better than behavioural interventions at treating ADHD”. On this point it just managed to scrape home. For the “symptom” of inattention two sets of raters, parents and teachers, produced a result in favour of “medication”. This result though was for the specific MTA “medication” regime and the specific MTA behavioural intervention programme. The former is unlike a typical out-patient “medication” regime. (As we shall see, it used a much higher than normal dose). The latter may be different from any other behavioural intervention. For this reason it is not valid science to make claims based on the results of the MTA treatment groups about “medication being superior to behavioural treatments” *in general.* While more cautious commentators (such as the NICE Guideline authors) are on the whole careful to acknowledge that the MTA treatment groups were specific to the study, other narrative builders are less careful. Thus we move from the specificity of the MTA study to more general claims about it having been “shown” that “medication is more effective than behaviour therapy alone at treating ADHD symptoms”. (See Section 5) ii)). Because of the specificity of the “treatment” regimes to the MTA study a finding that the MTA “medication” group outperformed the MTA behavioural group is without clinical application. But it appears that the aim of the study was to create the basis for narrative statements about “medication being superior to behavioural treatment” in *general*. The MTA study *only* works if it is misused.

That stimulant drugs are effective at improving attentiveness, at least for short-term use, is incontestable. This is not news. This is exactly what they do. The reality of ADHD drugging is that young people who are more inattentive than the average for their class are being given stimulant

drugs to reduce problem behaviours associated with variable attentiveness. Statements about the "superiority of medication management over behavioural treatment for ADHD symptoms" obscure this reality by presenting "symptoms" and the "treatment" as apparently self-evident "medical" facts. However; this is linguistic trickery, not empirical science. Describing a behaviour which belongs to a diagnostic category of psychiatry as a symptom and describing the somewhat haphazard control of that behaviour by drugs as a "treatment" involves re-purposing both the word "symptom" and the word "treatment".

The MTA study compared "medication" with a particular behavioural intervention. Other approaches which exist to care for young people who may be identified as having below average attentiveness for their age group were not explored.

ii) The construction and findings of the study

The MTA study compared 4 different "treatment" approaches "for ADHD". These were:

- A behaviour modification programme.
- A "medication" programme (stimulant drugging).
- Community care. This meant the usual treatment as an out-patient. Typically comprising a mixture of some stimulant drugging and some behaviour training.
- Combined treatment. The MTA stimulant drug programme and the MTA behaviour modification programme combined.

The "medication management" system which was used in both the medication only programme and the combined programme was, in the words of the MTA study authors, "carefully-crafted". First, methylphenidate, (Ritalin), was tried. If this failed to produce the desired change in behaviour dextroamphetamine or other drugs were used. For the initial methylphenidate titration a range of doses were tried and the "best" one for each young person was chosen by a "team of experienced clinicians". "Best dose" meant the one that produced the best response on the teacher and parent measurement scales that formed the main assessment in the study. Thus doses were fine-tuned to get the best possible results for drugging. There were monthly visits at which the doses for all drugs could be further fine-tuned if necessary.

The behavioural intervention programme was one specially put together for the MTA study. It included elements of parent training, teacher training and a summer camp for the students.

The "ADHD" young people (average age 8.5) were divided into the above four treatment groups. Over a period of 14 months the groups were measured against six criteria:

(1) ADHD symptoms were measured with inattention and hyperactivity-impulsivity sub-scales of parent- teacher-completed SNAP [2] ratings. (SNAP is an acronym denoting the names of the instrument's developers. One of the authors of the SNAP system is Dr James Swanson who was also one of the MTA study authors. The current version of SNAP, SNAP-IV is based on the DMS-IV definition of ADHD). SNAP is a check-list of behaviours. “Measured” means that a parent or teacher reports on their child or student's behaviour against a check-list of possible behaviours, rating them from not at all to very much. Behaviours “measured” include items such as “often is forgetful in daily activities”, “often fidgets with hands or squirms in seat”, “often argues with adults”, “often acts 'smart'” and “sometimes for at least a week has inflated self-esteem or grandiosity”. This is not “measured” in a scientific sense. Nor is “acting smart” or not sitting still in class a symptom of anything. This system could be called the symptom reduction scoring system.

Classroom observers who were blind to what “treatment” group a student was in also monitored for ADHD “symptoms” and “aggression” in class.

(2) oppositional/aggressive symptoms were measured with a parent and teacher SNAP oppositional-defiant disorder sub-scale;

(3) social skills were measured with a parent- and teacher-completed sub-scale from the Social Skills Rating System (SSRS)

(4) internalizing symptoms (anxiety and depression) were measured with an internalizing sub-scale from parent- and teacher-completed SSRS and children's self-ratings on the Multidimensional Anxiety Scale for Children (MASC)

(5) parent-child relations were measured with 2 composite scales from a parent-child relationship questionnaire

(6) academic achievement was measured with 3 sub-scales from the Wechsler Individual Achievement Test (reading, maths, and spelling)

[1]

In reporting their results the study the MTA authors stated:

a) The ADHD “symptom” of inattention was reduced more in the “medication” group than in the

behaviour “treatment” group according to both parents' and teachers' ratings. For the “symptom” of hyperactivity “medication” was more effective (at reducing the “symptom”) than the behaviour treatment according to teacher's ratings. However there is confusion here. The text clearly states that hyperactivity was reduced according to teacher’s ratings. But the table in which the data is presented clearly indicates that it was parents and not teachers who rated “medication” better than the behavioural intervention for the “symptom” of hyperactivity. It seems more likely that the table is correct since this is where the actual figures are presented.

b) The combined programmed also “outperformed” the behavioural programme for the ADHD “symptom” of inattentiveness on both parent and teacher ratings. In addition it also “outperformed” the behavioural intervention on parents' oppositional defiant behaviours scoring, internalizing symptoms (anxiety and depression) and Weschler Achievement Test reading achievement score. Both the text and the tabular data report that it was parents and not teachers who found the combined programme “superior” to the behavioural intervention only programme for the “symptom” of hyperactivity. [1]

c) That the combined programme scored better than the community care programme on 5 out of the 6 criteria.

The main claim then in this study is that the “medication” programme outperformed the behavioural programme at reducing “ADHD symptoms'. However, for the “symptom” of hyperactivity only one of the main measurement groups supported the finding. The report is confused about whether this was parents or teachers. Are they trying to confuse the reader? Are they trying to disguise the awkward fact that this result was only supported by one out of three measuring groups? It was not supported by the neutral classroom observers either.

The authors do not highlight the finding but the data shows that the neutral classroom observers did not report that “medication” was “better” than the behavioural programme for ADHD symptoms in the classroom. This is significant because this was a group of observers who were blind to what “treatment” any one young person was on. In addition they had no interest in the outcome and thus were more likely to give reliable results. This finding should be significant, for a study claiming to be following the standards of normal randomised clinical trials. However the unfortunate failure of the neutral group of raters to support the desired outcome is silently dropped from the summary of the results.

A secondary claim was that the combined programme outperformed the behavioural only programme not just on ADHD symptoms but also on parent measured oppositional defiant scores, parent measured anxiety/depression scoring, and reading. For anxiety/depression symptoms the young people themselves do not appear to have rated that the combined programme improved anxiety/depression better than a behaviour programme. The better score for “reading” was not

matched on the other two academic criteria, spelling and maths. (According to Breggin the claim for reading can be contested on statistical grounds. [3]) And, again, it does not appear to be the case that teachers rated the combined programme “better” than the behavioural programme for hyperactivity, at least according to Table 5.

The results then were patchy. Multiple groups of raters were used. But there was not a single measure on which all groups of raters agreed that “medication” achieved “superior” results to the behaviour programme. By reporting only the positive results and dropping from their summary the rating groups which failed to obtain a result the MTA authors are creating a misleading picture of the results of their study. If you ask 10 people to compare two products, one expresses a preference for the first product, but the other nine express no preference either way would it be correct to describe this as a robust finding in favour of the first product?

The MTA study *assumes* that reductions in symptoms demonstrated using the symptom scoring method are a good. In a sense this is valid. The “symptoms” are the signs “of ADHD” from DSM-IV. So reducing them means that someone's ADHD has been “reduced”. Nonetheless since “ADHD” is not a biological illness from which anyone suffers it remains to be explained how reducing “symptoms” is of benefit to the young person. The MTA study does not attempt such an explanation. This is characteristic of the ADHD narrative as a whole. The question of the value of obtaining a “reduction in symptoms” - by drugging - is obfuscated by precisely this false language of “symptoms”, “treatment” and so on.

iii) This is not science

In the MTA study the measurements were in the main carried out by parents and teachers. In the case of “ADHD symptoms” this was done using a questionnaire designed by one of the MTA study authors who is a noted pro-drugging enthusiast. [4] With the exception of the academic tests the base “data” in the MTA study is data which depends on interpretations of human behaviour by humans. This is not empirical, physical, data such as measures of heartbeat or temperature. It does not have the same degree of reliability as such data. For example; “inattentiveness” is open to interpretation. Can the MTA study authors be sure that it was not the case that the kind of behaviour produced by drugs was seen as more “attentive” by teachers whereas the kind of behaviour which resulted from a behavioural training course was not seen as more “attentive”? Does a student who sits still get marked up for attentiveness, though, in reality, they may be in a drug stupor? Whereas one who blurts out questions may get marked down even though he is attending to and trying to engage with the lesson? What was actually being measured? Ultimately it may come down to the kind of behaviour sought out by teachers. Can the MTA study authors be sure that when parents reported on “inattention” they meant the same thing as teachers? No. The MTA study is attempting to quantify base data which is subject to significant degrees of

unconscious bias before it gets to the stage where it is quantified. For this reason alone the figures, graphs and tables produced in the report do not offer even remotely the kind of certainty that their mathematical formulation suggests they possess.

Almost all the “measurements” were carried out by parents and teachers. It was not just that the measurements were subjective, interpretations of human behaviour by humans. It is further the case that the people doing the measuring were highly interested parties. Parents and teachers play an active role in the “diagnosis” of ADHD. They are part of the story. There were two exceptions to this reliance on interested parties as raters. Classroom observers, who were blind to which “treatment” any young person was on, “measured” classroom behaviour. The young people themselves were asked to report on their anxiety/depression levels. Tellingly, in both these cases the result of “superiority” for the “medication” based programmes over the MTA behavioural programme was not maintained. These facts are not highlighted in the results section of the MTA report. From a clinical perspective this is the wrong way around. From a clinical perspective the most important findings would be those of a) the subjects themselves and b) any neutral raters. The results of untrained parties with an emotional investment in the outcome would be handled with caution. That they are prioritised here tells us something about the nature of “ADHD”.

The young people in the study were assigned randomly to one of the four treatment groups. “Medication” was a treatment option and since all the participating parents had to agree in advance to random assignment it follows that *all* the parents involved were potentially favourable enough to medication to accept it as a possibility. When it came to it 17 refused or, more likely their parents refused on their behalf, to be drugged. Possibly only those who fully accepted the “benefits” of “medication” continued into the study. At any event the people doing the rating simplicity accepted that “medication” as a safe treatment. This would have introduced bias into the study.

31% of all study participants were on “medication” prior to the study (Table 3). The figure for those who were on a formal behavioural programme is not given in this table. This high percentage already on “medication” also creates the possibility of bias in the results. Interestingly, the drop-out rate was higher in the “medication” group than in the behaviour group, one of several facts present in the MTA study which is not favourable to the pro-drugging conclusion reached and which does not find its way into the summary of results.

The claims for the “superiority” of “medication” for ADHD “core symptoms” relate to the two ADHD “symptoms” of inattention and hyperactivity. But in reality these are not “symptoms” at all. A “symptom” in medicine is a change in the body which is noticed by the patient and which is biologically associated with a disease. An example of a symptom is a running nose as a symptom of having a cold. The virus and the body's response to it results in the medical symptom. Usually a medical symptom is something that to a greater or lesser degree the patient suffers from. But “ADHD symptoms” are something else altogether. ADHD “symptoms” are in fact a range of

behaviours. DSM-IV refers to “signs” not “symptoms” and explicitly defines ADHD in terms of “disruptive” behaviours which are “inappropriate for developmental level” (See Appendix i)). “Symptoms” are a sort of secondary reification based on DSM-IV.

By using the word “symptom” the MTA study authors are engaged in a conjuring act. The public understand that a “symptom” points to a disease which it is a symptom of. Thus if they are informed that “symptoms have been reduced” they may be led to believe that that is a desirable result. But this is a false use of language, not a medical reality. ADHD “symptoms” are not “symptoms” at all. They cannot be, because there is no biological disease which they can be symptoms of. Recall that the NICE ADHD Guideline authors concede: “The diagnosis of ADHD does not imply a medical or neurological cause”. [5] What is being reduced is “disruptive” behaviours.

There is a strange anomaly in the MTA study. If only a behavioural programme was being used the authors could have talked freely about behaviours being modified. But once they involve “medication” it becomes necessary to talk in terms of “symptom reduction” to mask the reality that drugs are being used to modify behaviour. But now they end up with the absurdity that the behavioural intervention too has to be described as “reducing symptoms”. Of course behavioural interventions do not “reduce symptoms”. They modify behaviour.

The MTA study also uses the words “medication” and “treatment”. Both of these words also create a false impression. The general public would understand a medical treatment to be a treatment *of* something. One is being treated for measles, flu, jaundice, a broken leg. But when “ADHD children” are treated they do not in fact have anything which is being treated. “The diagnosis of ADHD does not imply a medical or neurological cause”. [5] You cannot “treat” something which doesn't exist. “Medication” is the corollary of “treatment”. The Latin root of the word “medicine”, which is shared with “medic”, is from a word meaning to heal. The common understanding associates a medicine with a therapeutic or healing effect. Drugs given to young people with an ADHD label however do not have a healing effect. At best they affect brain chemistry to increase the amounts of a chemical substance associated with improved attention. This is not equivalent, for example, to an antibiotic which reduces the prevalence of a harmful bacterium in the body. In the end *“*ADHD” is a classification category of psychiatry relating to a problem behaviour of young people. The use of medical terminology masks that disruptive behaviour is being reduced with drugs.

The people who developed the MTA study (and others of this kind) do not as a rule consider how being part of a study effects the behaviour of those studied. The view of “children” involved is essentially the same as if they were laboratory rats whose responses would not be altered by being part of a study. Studies involving rats do not need to consider what effect being observed might have on the rat. A rat probably behaves much the same way whether or not it is being

observed by a human with a clip-board. But this, a lack of awareness of being monitored, is not the same for people. People will be aware that they are being monitored and this may well effect their behaviour. In the MTA study young people (“children”) are given drugs or involved with their parents in a behavioural programme, and then observed by people holding (as it were, or maybe even literally) clip-boards. This might be their class-teacher or their Mum or Dad. Their behaviour is likely to be effected by the knowledge that they are being observed by another human being as part of a study, especially perhaps when they have been told it is a study about “their ADHD”. For this reason studies of this kind have the problematic that inferences cannot be directly drawn about the behaviour of populations in non-study contexts. The exact effect of study participation cannot be determined and controlled for. The “clinical” posture which disregards this factor is both heartless and bad science at the same time. Furthermore, it discourages, possibly even excludes, the kind of warm relations between parent and child that might be all that is needed to “solve” the problem of the young person's behaviour. The MTA study embodies a certain specific kind of power relations. One the one hand the “children” are objectified. They “have” something. They are drugged and studied. Not consulted. On the other hand certain groups of people are enjoined to take up a special kind of subjectivity. The subjectivity that comes from observing and measuring other people. These acts of “measurement” produce certain forms of “knowledge”. In the MTA study the parents and teachers are being groomed to adopt a “clinical” posture towards their own children and their students. The structure of the MTA study funnels the problem into the kinds of theoretical frameworks which psychiatrists and psychologists use and thus towards the professional services which they offer and benefit from. Other solutions are excluded.

The MTA study authors reported that “medication” was superior to their behavioural programme “according to 2 different data sources“. They mean parents and teachers. Thus they bury the fact that the clinically more important group of raters, the neutral and blinded classroom observers did not report such a result. (They also try to bury it seems the fact that it was just parents who rated “medication” “better” than the behavioural programme for hyperactivity). How can we explain this use of interested parties to claim a result and the attendant ditching of the results of the neutral and blinded raters? In the world of ADHD an “ADHD” “diagnosis” does not come about because a young person has complained of feeling unwell. When a young person is “diagnosed” “with” “ADHD” and drugged it is his parents who have taken him to the psychiatrist or paediatrician and asked that something be done. (As we shall see there is anecdotal evidence that schools are putting pressure on parents to do this in some cases). The idea of ADHD drugging is to reduce the “disruptive” behaviours which are the “signs” “of ADHD”. It is in this light that we can understand the reliance of the MTA study, and most other pro drugging studies, on parents and teachers as raters. The test of ADHD drugging is whether it works for parents and teachers. There is no clinical problem to solve. The reliance on interested parties, in fact the end customers for the product, confirms that we are in the realm of a customer satisfaction survey.

iv) It's a setup

The current “wisdom” in the ADHD world is that:

> Although no cure exists for the condition, symptoms can be reduced by a combination of medication and behaviour therapy. [6]

These two “treatments” are those that are delivered by clinical psychologists and psychiatrists. That ADHD cannot be cured is clearly good news for any company or individual who makes money out of treating it. In fact the best cure for ADHD would be not getting “diagnosed” in the first place. The MTA study feeds into and supports this approach of “treating” ADHD with both “medication” and behavioural interventions. These were the only two approaches tried in the MTA study. Other approaches which would manage the problem of disruptive young people, without making it a “clinical” problem and “treating” them are not envisaged. Before the MTA study even starts the problem has been framed in a certain way. A way which is of potential benefit to certain professional interest groups.

The particular behavioural programme adopted by the MTA study was based in part on the work of an R. A. Barkley, author of *Defiant Children: A Clinician's Manual for Parent Training*. [7] One of Barkley's methods is based on commands and punishments. The method involves following up a command with a threat of punishment. This is how the NICE Guideline authors describe this approach:

> From the six included trials, there was one comparison involving a teacher-led intervention named ‘giving effective commands’ (Barkley, 1997), which consists of the teacher giving the child a command once and, if necessary, proceeding to a warning where the child is informed of the consequences of not carrying out the command; in cases where the child does not comply, the threat is carried out. [8]

The reader will note the emphasis on commands, compliance and “threats” towards “the child”. It is possible that had a different and more positive, caring, behavioural programme been used in the MTA study it would have “outperformed” the drugging regime for attentiveness. However, in practice there is no doubt that stimulant drugs are powerful agents for improving attentiveness at least in the short-term. Stimulant drugs might indeed “beat” any behavioural programme in the short-term. Nonetheless, in terms of the two-horse race competition set up by the MTA study authors it remains a theoretical possibility that had a different horse represented the behavioural method then a different result could have been obtained for attentiveness.

The “medication” regime in the MTA study was, in the authors own words, “carefully-crafted”. Each subject was individually titrated with methylphenidate to achieve the “best” possible “result”:

> Cross-site teams of experienced clinicians blindly reviewed graphs portraying parent and teacher ratings of responses to each of the 4 doses and by consensus selected each child's best dose. [1]

The “best” result was the one that produced the best scores on the raters scales. Everything possible was done to give the “medication” horse the best possible chance to shine. In subjects who did not respond to methylphenidate dexamphetamine or other drugs were used instead. (At the end of the study only a few subjects had been switched to drugs other than methylphenidate or dexamphetamine). Monthly medication visits monitored the responses and doses were continually adjusted for best “results”. Reductions in dose were only allowed to reduce side-effects.

Attendance at the behavioural programme in the MTA study was patchy. For example attendance at the parental component was just 77.8%. [1] The behavioural programme tapered off before the end of the study. In contrast, the “medication” regime appears to have been run at full strength with teams of clinicians monitoring and changing the “doses” throughout the study. Every possible advantage seems to have been given to the “medication” “treatment”. At any event this just emphasises how it is not possible to compare a “medication” regime with a behavioural intervention in a clinical sense. Decisions about the level of “dosing”, the type and length of the behavioural intervention, and so on, are all arbitrary. The MTA study never could have been a serious piece of clinical research.

The MTA subjects on the “medication” only programme who were receiving methylphenidate were being dosed with 37.7 mg daily. For those on the community care programme, that is the normal outpatient circumstance, the average daily dose was 22.6 mg, very significantly less. MTA subjects were “dosed” 3 times a day. Community care subjects receiving drugs were “dosed” on average 2.3 times a day. The “medication” regime in the MTA study was *nothing like* the typical experience of “medication” in normal outpatient settings.

The MTA study compared one specific behavioural programme with a very highly engineered and optimised regime of high-dose stimulant drugging completely atypical of usual out-patient experience. The MTA study did not compare current typical “treatments” but an artificial set produced just for the MTA study. The MTA study then *cannot* be used to justify “medication” in general over behaviour treatments in general. Given that the study was designed so as to be able to make just such a claim there is only one unavoidable conclusion. The primary purpose of the MTA study was to provide the raw materials for propaganda. It was designed to enable people to say *in general*:

> These drugs have been shown to be more effective at treating ADHD symptoms than behavioural therapy alone... [9]

Even though such a claim cannot be made on the basis of the MTA study *whatever its results.*

The above quote is from a paper by a Wellcome Trust ADHD researcher. We will discuss this paper in Section 5) ii). Here it is cited to show that the MTA study is used in the ADHD narrative to generate propaganda.

The MTA study only recruited "children with ADHD" as participants in the study. However; the fact is that stimulant drugs improve attentiveness and concentration in *everyone*. This is why, for example, US fighter pilots use them. [10] Had the same tests been carried out on a group of young people without an ADHD label, or a group where no attention was paid to any psychiatric labels, it is likely that a similar result would have been obtained. Stimulants are good at improving attentiveness; possibly better than a behavioural programme, at least in the short-term. This is all that the MTA study has shown. (Though the lack of corroboration from the neutral classroom observes must call even this result into question). The claims about stimulant "medication" being a "more effective" "treatment" "for ADHD" than a behavioural programme depend entirely on the fact that the study only studied "ADHD children". It is the way the study is set up (ADHD children v. normals), and not empirical science, which generates the narrative about "better treatment".

v) Stimulant drugs may improve concentration but should they be be given to "improve" behaviour?

Parents, but not teachers, rated the "medication" programme better than the behavioural programme for the "symptom" of hyperactivity. This is according to Table 5. [1] As we have seen, however, the text says it was teachers, not parents. This is an inexplicable confusion in the paper. The tabular data perhaps provides the more reliable report and we assume this to be correct.

Excessive use of stimulant drugs leads to a state of drug exhaustion. Breggin refers to the evening crash in his summary of the harmful effects of stimulant drugs. [11] The UK government advice to young people about the dangers of amphetamines explains that:

> The high is generally followed by a long slow comedown, making you feel really irritable and depressed. [12]

The "come down" is a well-known effect of stimulants and amphetamines. The subjects (8 year

olds) in the MTA study had been “dosed” at breakfast, lunchtime and then again in the afternoon. By the evening they were probably experiencing the inevitable “evening crash” effect. Their parents duly marked them down as “less hyperactive”. But this cannot be described as a “benefit” resulting from a “treatment”. Breggin points out that the use of parents rather than trained clinicians as raters is likely to increase the chances that negative drug effects may be misinterpreted. [3]

The most consistent result the MTA study can produce is that both parents and teachers rated (high dose) “medication” better than the MTA behavioural programme for inattentiveness on the symptom reduction scoring system. If we ignore the fact that this was not corroborated by the neutral classroom observers we are left with a claim that high dose “medication” was superior to the behavioural programme at improving attentiveness over a 14 month period. But what the MTA study does not do is explain why it might be a good idea to drug young people so as to make them slightly more attentive than they would be otherwise. The MTA study, like the ADHD narrative as a whole, avoids discussing this actual value question. Results such as “superior in benefiting ADHD symptoms”, “marked reductions in symptoms over time” and “offered greater benefits” seem to be intended to appear as self-evident goods. But ADHD “symptoms” are not medical symptoms. Young people do not suffer from them as they might say, from the symptoms of measles (fever, dry cough etc.). In the MTA study the “symptoms” which have been reduced are disruptive behaviours defined by psychiatry. Precisely because they are not medical symptoms there is no self-evident good to be achieved by reducing them. Rather than explain the value of reducing disruptive behaviours with drugs, psychiatry tries to mask the reality of what it is doing by adopting a pseudo-clinical language of “marked reductions in symptoms over time” etc. However; nothing in the MTA study establishes any kind of a medical reason for the practice of using powerful and toxic drugs to keep young people glued to their seats when remaining in seat is expected.

Nor is there any discussion in the MTA study about the fact that one “treatment” is accompanied with side-effects and the other has none.

vi) Side-effects in the MTA study

Side-effects of “medication” were monitored for those on medication. The MTA study reports:

35% no side-effects
49% mild side-effects
11% moderate side-effects
3% severe side effects

[1] (Rounded to whole numbers).

When evaluating medical treatments accepted medical practice is to weigh up the benefit accrued to the patient against any additional suffering caused. For example this is how the UK's Medical and Healthcare products Regulatory Agency (MHRA) explains it:

> Do the advantages outweigh the dis-advantages of taking the medicine?
>
> Does the medicine do the most good for the least harm for most people who will be taking it?
>
> Are the side effects acceptable?
>
> [13]

Medical science as articulated by the MHRA above demands that the final recommendation takes account both of “advantages” and disadvantages of a treatment. The fact is that behavioural interventions do not cause *any* side-effects. Young people on behavioural programmes do not experience stomach aches or psychosis. They do not have trouble sleeping. They do not experience a slowing down of normal growth. (See Section 3) v) for a summary of the harms done by ADHD drugs). There are no risks (even very slight ones at the margins of statistical significance) of suffering a fatal cardiac event as the result of attending a behavioural programme. But behavioural programmes still “reduce ADHD symptoms”.

The MTA authors acknowledge the “side-effects” associated with drugging but nowhere is there any attempt to draw up a cost-benefit matrix which would measure the advantages of “medication” minus the harms it does against the advantages of behavioural training minus the harm it does (none, medically). Such a calculation would inevitably lead to a recommendation for behavioural programmes rather than “medication” to “treat ADHD”. This is probably why it is not done.

Side-effects reporting in the MTA study was done by parents. The young people, who were on the

drugs and who would have experienced the “side-effects” do not appear to have been consulted unless their parents consulted them. Some parents may not have bothered. Pro-medication parents can be blind to the “side-effects” experienced by their children. (Parents who might be especially alive to these side-effects will have excluded themselves from this study). Sometimes young people will not tell their parents about, for example, night-time hallucinations. They may not have the words to tell. The young people are aware that their parents have put them on drugs. This may make it difficult for them to tell their parents of negative consequences. The young people may not realise or be able to clearly formulate that their discomfort is a result of the drug. For a range of reasons young people may not to tell their parents about side-effects. Side-effects will therefore be under-reported in the MTA study.

The MTA study authors, predictably, claimed that the side-effects may have been over-reported:

> These figures may overestimate side effects, because 6 of 11 reported severe side effects (depression, worrying, or irritability) could have been due to nonmedication factors. [1]

The MTA study did not use an “untreated” control group. They are free to speculate that the side-effects were not due to methylphenidate. But it is just speculation. In fact, being irritable and depressed are well-known “side-effects” of stimulant drugs. See for example UK government advice to young people about amphetamines. [12] Methylphenidate is not an amphetamine but is a stimulant and shares a similar effects profile. [14] See also Peter Breggin's summary of the “side-effects” of methylphenidate and other ADHD drugs. [15]

The side-effects rating system used in the MTA study was the Pittsburgh Side Effects Rating Scale [16]. On this scale mild means the side-effect is present. Moderate or severe means that “impairment of functioning or social embarrassment” was caused. 14% of the young people being “medicated” on the MTA study therefore became impaired as a result of their “treatment”. 63% experienced at least one of the items in the ratings system with or without impairment. The Pittsburg Rating Scale includes these points:

- Tics
- Buccal-lingual movements (jaw-clenching for example)
- Picking at skin
- Worried/Anxious
- Dull, tired, listless
- Headaches
- Stomach-aches
- Crabby, irritable

- Tearful, sad, depressed
- Socially withdrawn
- Hallucinations
- Loss of appetite
- Trouble sleeping

Most of these “side-effects” appear non-trivial. Even if not to the extent of “impairment of functioning” is it still acceptable to render an eight year old “tearful, sad and depressed”, “worried/anxious”, insomniac or hallucinatory in order to gain an improvement in behaviour only somewhat better than one which could have been obtained on a behavioural intervention programme? (And, on some measures, not better at all).

Despite the fact that 63% of the young people in their study (average age 8) experienced side-effects the MTA study authors claimed that this was not a problem:

> In contrast to frequently expressed concerns, children given combined treatment and medication management tolerated medication well, including a third dose given in the afternoon. [1]

In 2009 the European Medicines Agency (EMA) published a review of all preparations of methylphenidate. [17] The EMA acknowledge that reported adverse events for methylphenidate include:

> Most frequently reported psychiatric adverse events of interest from spontaneous reports were abnormal behaviour, abnormal thinking, anger, hostility, aggression, agitation, tic, irritability, anxiety, crying, depression, somnolence, aggravated ADHD, psychomotor hyperactivity, emotional disorder, anger, nervousness, psychotic disorder, mood swings, morbid thoughts, obsessive-compulsive disorder, personality change/disorder, restlessness, confusional state, hallucinations, lethargy, paranoia and suicidality. [18]

Adverse events are those that are reported by clinicians observing the actual result of giving a drug to their patients. The side-effects listed in the MTA study are well-known effects of taking methylphenidate. Beyond documenting them the MTA authors don't seem to think they matter very much. But if a young person presented to a doctor as suffering from stomach-aches, hallucinations and insomnia *that* would normally be regarded as medical problem. While fixing “disruptive” behaviour, which is not a medical issue, methylphenidate causes health problems in many young

people who take it.

vii) Only in the short-term

The MTA study showed a superior result (symptom reduction system) for its “medication” programme over its behavioural programme according to some but not all of its rating groups. The original MTA study lasted 14 months. Some of the researchers involved in the original study continued to work with the same subjects and the same methods. Their results were published as the “3-Year Follow-up of the NIMH MTA Study”. This is Jensen *at al*. 2007. [19] This study found that:

> At 3 years, 485 of the original 579 subjects (83.8%) participated in the follow-up, now at ages 10 to 13 years, (mean 11.9 years). In contrast to the significant advantage of MedMgt+Comb over Beh+CC for ADHD symptoms at 14 and 24 months, *treatment groups did not differ significantly on any measure at 36 months*. [19] *Emphasis added.*

Dr William Pelham was one of the original MTA researchers. He was also involved in the follow-up study programme. He told the press:

> The children had a substantial decrease in their rate of growth so they weren't growing as much as other kids both in terms of their height and in terms of their weight. And the second was that there were no beneficial effects – none.
>
> I think that we exaggerated the beneficial impact of medication in the first study. We had thought that children medicated longer would have better outcomes. That didn't happen to be the case. There's no indication that medication's better than nothing in the long run. [20]

This appears to be a significant finding. It comes from a direct follow-up to a major NIMH (US National Institute of Mental Health) sponsored study which was supposed to have demonstrated the “superiority” of “medication'” over behavioural interventions once and for all. Was Dr Pelham right to say that the “medicated children” did not have “better outcomes”?

Matters are slightly more complex than the convergence of scores alone would tell us. As Jensen *et al*. point out there are other possible explanations. The main one is that the original MTA treatment groups were not maintained. After the original 14 month MTA study ended participants (in practice their parents most likely) were free to choose what kind of “treatment” they received.

The original MTA “treatment” regimes were no longer available. As a result of this, at 36 months there had been some movement within the groups. Jensen *et al*. detail this:

> Medication use changed substantially over time, however. Thus, during the 24- to 36-month assessment interim, the percentage of children with high use decreased to approximately 71% for Comb and MedMgt, remained relatively steady at 62% for CC, and increased to 45% for Beh. Despite this convergence in use rates across groups by 36 months, medication use rates and total daily doses continued to differ significantly at 36 months (Table 2). [19]

Thus there was significant convergence of “treatment” across the original MTA study groups. Jensen *et al.* explain how this factor can explain the convergence of scores and the loss of the “medication advantage” from the original MTA study:

> Thus, differences in the intensity or quality of treatment (or lack of treatment) during the 14- to 24-month post study interim may have resulted in the loss of some of the 14-month difference. [19]

This is a valid explanatory comment on their own findings. However, as Jensen *et al*. 2007 acknowledge “”medication use rates and total daily doses continued to differ significantly at 36 months”. And yet the scores still converged:

> No significant differences were found among the originally assigned treatment groups on any of the variables in this table at 36 months. [19]

There was complete convergence on scores but only a drawing together of “treatments” which continued to “differ significantly at 36 months”. Some young people were still being much more heavily “medicated” than others. But still the scores converged. The inescapable conclusion is that in the longer run the “medication advantage” over a behavioural treatment does wear off, at least to some extent. This is exactly what we would intuitively expect. It is known that people develop tolerance to stimulant drugs. The drugs will thus probably be less effective in the long-term.
The MTA follow-up study calls into question whether the “medication advantage” found, on some measures, in the original MTA study would have been sustained at 36 months even if the original “treatment” regimes had been maintained. The result of the MTA follow-up study was a total disaster for the ADHD community. The US National Institute of Mental Health which sponsored the original MTA study now spins these results like this:

The study also showed that these benefits last for as long as 14 months. [21]

Some of the MTA authors tried to recover the position. The main effort was a secondary evaluation of the data study, Swanson *et al*. 2007 [22]. This study re-analysed the data to show that "medication" still "reduced symptoms", absolutely (compared to base-line), even over 36 months, for at least some young people. This was a retreat to the basic "medication reduces ADHD symptoms" position. The evidence from their own study, which should surprise no one, that the "medication advantage" wears off over time was not followed up. As with any other finding from an ADHD study which does not support the main ADHD drugging narrative, it was hastily discarded. And the effort was redirected to finding new data to support the "scientific evidence" for ADHD drugging. We review Swanson *et al*. 2007 in Section 4) v) where we discuss how the NICE Guideline authors use it to limit the damage from Jensen *et al*. 2007.

viii) The flawed MTA study - a summary

The MTA study is not a scientific study. Its conclusions have no scientific value. The following summarises some of the problems with this study.

1. The "data" on which the comparisons between the different modes of "treatment" were based were not empirical measurements. They were subjective assessments of the behaviour of young people. Ratings criteria such: "Often is forgetful in daily activities", "Often loses temper", "Often is spiteful or vindictive" [2], for example, are not objective measures like, for example, measures of blood pressure or heart rate. No amount of presenting the "data" in tables and graphs and applying methods of statistical analysis can disguise the fact that the foundations are based on subjective interpretations of behaviour.

2. The people doing the measuring, providing the "data", were people who are part of the ADHD story. People who are involved in a situation cannot be relied on to provide unbiased reports. The blinded classroom observers, the only group of measurers who might reasonably be supposed to be detached and unbiased, did not produce a score which favoured drugging over the behavioural intervention. This finding should be given significantly more weight than the results from the parents and teachers. Indeed the ratings from the parents and teachers are of no scientific value and should only have been included as ancillary data.

3. In the MTA study words like "diagnosed", "symptom", "treatment", "benefit" and "improvement" are used in such a way that an uncritical audience hearing about such a study through the media may mistake these for scientific medical terms. In the MTA study as in the ADHD narrative as a whole these terms have been misappropriated. In is nonsensical to talk about "symptoms" of a

diagnostic category of psychiatry which “does not imply a medical or neurological cause” [5]. In the MTA study as in the wider ADHD narrative use of clinical medical terms in connection with a practice which is neither clinical nor medical serves to disguise the real nature of what is going on.

4. The only objective result (in the sense of something which can be objectively measured) from this study is the slighter better increase in reading scores with the MTA combined regime compared with the MTA behavioural programme. The better score for reading was not matched on the other two academic criteria, spelling and maths. The reading result can, according to Breggin, be contested on statistical grounds. In any event; simply showing that giving eight year olds stimulants can slightly improve their reading scores is not, perhaps, a reason to do this.

5. Follow-up research to the MTA study, conducted by the MTA authors, Jensen *at al*. 2007 [19], showed that in the longer term (that is 36 months) the higher scores (symptom reduction system) for “medication” over behavioural interventions found in the original MTA study (14 months) were not maintained. Pro-drugging groups such as the US NIMH and the authors of the UK's official Guideline on “managing” ADHD (See Section 4) v)) are left to spin and claw their way out of this highly awkward finding.

6. All of the students in the MTA study appear to have been in school. The MTA study thus reifies (treats as if it were an absolute fact of nature when it is in fact a matter of social policy and current practice) the prevalent system of mass schooling in the industrialised world. A condition which appears when measured against the mores of a specific social institution is not an objective condition. The MTA study, framed as it is as a “clinical” endeavour, participates in the project of excluding a social policy solution to the problems of inattentive young people in school. The way the study is constructed already excludes many of the more positive and humane solutions to the problems of inattentive young people in school which could be tried.

7. From a medical perspective a proposed treatment should be weighed up in terms of its benefits and side-effects. In the MTA study the relative advantages (“symptom reduction” claim) of “medication” over a behavioural intervention are considered but not their relative harms.

8. The voices of the young people themselves are absent from the MTA study. The young people were consulted on just one of six measures. That of anxiety/depression. On this score they did not report that “medication” was “superior” to the behavioural programme. There is no endorsement from the actual “patients” therefore for the claimed results of the MTA study. This is a reminder that “ADHD” is about adult convenience not patient well-being.

9. The study reported that parents but not teachers reported that “medication” scored better (symptom reduction system) than the behavioural intervention for hyperactivity. (Table 5). We discussed how this may well be because stimulant drugs induce a lethargic reaction as the drug

effect wears off towards the end of the day. The well-known “evening crash” effect. By the evening when they were assessed by their parents they may have been suffering from drug induced exhaustion. A negative consequence of taking the drugs may have been reported as a “benefit”. Because the young people were not properly consulted (see above point 8)) such effects are not likely to be discovered.

10. Because the “treatment” regimes in the MTA study are unique to it and, at least in the case of the “medication” regime atypical of normal outpatient regimes, no results can be extrapolated from the MTA study to the wider clinical scene. The MTA study cannot provide the basis to make claims about “medication being superior to behavioural treatment” in general. This is a structural feature of the study and applies whatever results were obtained. Any claims made on the basis of the MTA study about the general picture have an element of propaganda about them.

11. The MTA study is a customer satisfaction survey. In as much as it masquerades as science that is a hoax.

ix) Ethics and the MTA Study

The authors of the MTA study claimed that their study did not include a control group (that is a group receiving no “treatment”) because that would have been “an ethically unacceptable option for an ADHD study of this length”. [1] This is despite the fact that there is no biological test “for ADHD” and no one who is “diagnosed” “with” “ADHD” has been identified as having a medical illness or condition. Any one group of “ADHD children” is essentially arbitrary. Assignment to such a group depends to a large extent on whether parents choose to have their children assigned to it. Many young people are inattentive relative to the average for their age-group and survive without ill-effects without being “treated”. It might be unethical not to treat, for example, measles, as part of a study. It is not unethical not to “treat” someone simply because they have been assigned to a “diagnostic category of psychiatry”. We can add that a behavioural intervention cannot be described as a “treatment”. Summer camps, parenting skills classes for parents, and classroom aides improve behaviour. They don't save lives. Those on the behavioural programme were therefore, in any medical sense of the word, not being “treated” as such. The argument that it would not have been acceptable to have included an untreated control group is therefore fallacious. Had such a “no treatment” control group been included it would have been possible to assess (symptom reduction scoring system) how “medication” and an ADHD behavioural programme compared against no “treatment”. This may have produced an unwelcome result for the MTA study. However; the main reason that no control group was used was probably propaganda related. It is essential to continue to spread the message that “ADHD” requires “treatment” and including a no treatment control group would have exposed the fact that no “treatment” is a perfectly valid option. No harm results from no “treatment”.

The purpose of the MTA study was to generate pro-drugging propaganda. To do this it subjected 8 year olds to an intensive regime of head-aches, insomnia, stomach-aches, growth-loss, hallucinations, anxiety and depression. The MTA authors claim that they didn't include a no treatment control group for “ethical” reasons. The ethical problem is perhaps the reverse of that proposed by the MTA study authors.

## 3) The drugs

i) The drugs

Currently in the UK there are a small number of drugs used for “ADHD”. The following table shows the drug, the company which produces it and the chemical substance which it actually is:

| **Drug sales name** | **Company** | **Main Chemical substance** |
|---|---|---|
| Strattera | Lilly | Atomoxetine Hydrochloride |
| Dexedrine | GlaxoSmithKline | Dexamfetamine Sulphate |
| Dexamfetamine | Sold generically | Dexamfetamine Sulphate |
| Concerta | Janssen Pharmaceuticals, Inc | Methylphenidate Hydrochloride |
| Equasym | Shire US, Inc | Methylphenidate Hydrochloride |
| Ritalin | Novartis | Methylphenidate Hydrochloride |
| Methylphenidate Hcl | Sold generically | Methylphenidate Hydrochloride |

*Notes*:

a) Methylphenidate hydrochloride is a cocaine-like substance. It is listed as an addictive Schedule II drug by the US Drug Enforcement Agency. It is a stimulant drug pharmacologically similar to amphetamines and cocaine. [1]

b) Dexamfetamine Sulphate is a stimulant of the amphetamine family.

c) Strattera is the only drug in the list which is not a stimulant. The actual chemical substance is atomoxetine hydrochloride. Strattera was originally researched as an anti-depressant. [2]

d) Based on 2013 data for England the majority of ADHD drug prescriptions are for methylphenidate hydrochloride in some form, with atomoxetine second and dexamfetamine sulphate third. [3]

e) The licensing of drugs in the UK is carried out by the Medical and Healthcare Products Regulatory Agency (MHRA). Some drugs are licensed at a European level by the European Medicines Agency. In the UK dexamfetamine and methylphenidate are not licensed for use on adults. Atomoxetine is licensed for use on young people and adults who “who had symptoms of

ADHD as children”.

In the UK doctors can and do prescribe drugs “off-license”. For example the drug Adderall may occasionally be prescribed though it is not licensed for ADHD at all. Adderall is a mixture of four amphetamines.

f) In the US methamphetamine (brand name Desoxyn) is also used as an ADHD “treatment”. This is exactly the same substance which is also sold on the street as crystal meth, a substance commonly held to be absolutely lethal for young people to use. Desoxyn does not appear to be available on the NHS, based on 2013 data. [3].

ii) The myth of the paradoxical effect

There is a myth around giving stimulant drugs to young people ("with ADHD") known as the “paradoxical effect”. According to this myth there is something special about the brains of “young people with ADHD” that makes stimulant drugs which are bad for everyone else good for them. This myth is necessary to avert the suspicion that dangerous drugs are being given to young people which may harm them. While the ADHD lobby does not seem to actively promote the myth these days it remains a necessary but unspoken part of the narrative. The authors of the NICE Guideline reference it but avoid taking a firm view:

> The question of a paradoxical effect of stimulants on people with ADHD has been raised but is not well studied. For example, do stimulants have an impact on the same processes and in the same way in all people, whether they have ADHD or not? [4]

As we have already seen, the NICE Guideline authors concede that “The diagnosis of ADHD does not imply a medical or neurological cause”. [5] You cannot plausibly discuss the biological effect of a drug on someone and at the same time say that they don’t have a biological condition. The point of posing this fake and entirely disingenuous question is to spin the myth out for a while longer while avoiding actually making an unsustainable and refutable direct claim for it.

The "paradoxical effect" claim has its origins in some 1930s research by psychiatrist Charles Bradley who noticed that while giving “disruptive” children an amphetamine (Benzedrine) treatment for headaches their concentration improved. This was the original basis for prescribing stimulants to young people for inattention. Bradley was enthusiastic:

> There appeared a definite 'drive' to accomplish as much as possible during the school period, and often to spend extra time completing additional work. Speed of

> comprehension and accuracy of performance were increased in most cases. [6]
>
> It appears paradoxical that a drug known to be a stimulant should produce subdued behaviour in half of the children. [7]

Thus stimulant drugs as a “treatment” for inattention and hyperactivity were “discovered” by accident. There was no research which identified a biological process and which showed how the medicine modified that process so as to promote health or reduce symptoms (in the actual sense of the word symptoms).

The "paradoxical effect" claimed by Bradley was simply a convenient conjecture. What Bradley noticed is a description of what happens when you give anyone amphetamines. They become “driven” and somewhat obsessive. There was no “paradoxical effect”. We can also notice the rather frank claim about the benefit of the treatment. It made the “children” “subdued”. Since the 1930s psychiatry has had to be less open about the actual reason for stimulant drugging.

The idea that there was a “paradoxical effect” has continued in the ADHD discourse since the time of Bradley. It was well known from the Second World War onwards (at least) that amphetamines improved concentration in all people, not just hyperactive people. Nonetheless the psychiatric profession apparently persisted in using the myth of the "paradoxical effect" until a study in the 1970s established that there is a general effect to improve concentration and reduce impulsiveness in all young people and adults with or without an ADHD label. [8] It is absurd that a study was needed to establish for the psychiatric profession that stimulants improve attention and focus even for “normal children” as well. This is an example of a misuse of science which is prevalent in much of the ADHD literature; claiming that the obvious is not known until it has been “established” by a “study”. The US military would not have been using amphetamines in WWII and Vietnam if they did not improve concentration, to name just one official use.

The nearest this author has seen to a scientific account of how stimulants (methylphenidate in this case) may effect “people with ADHD" differently from people “without ADHD”, is in the paper Dopamine Activity in Caudate and Preliminary Evidence of Limbic Involvement in Adults with Attention-deficit/Hyperactivity Disorder, (Volkow *et al*. 2007) [9]. Volkow *et al*. 2007 was not attempting to establish the “paradoxical effect”. It was investigating possible links between dopamine production and inattention and exploring the theory that dopamine production is limited in an ADHD group. In the study methylphenidate hydrochloride (Ritalin) did not induce the same level of increase in dopamine activity in the “ADHD group” as in the control group. Thus, in this study it was found that young people "with ADHD" showed a "blunted response" to methylphenidate.

One of the possible flaws in this study is that some of the ADHD subjects may have had some, limited, previous exposure to Ritalin. This means that this finding of a "blunted response" could be argued to have been due to this previous exposure since people develop resistance to drugs. A second weakness in Volkow *et al*. 2007 concerns the small sample size. Just 19 adult subjects "with ADHD" and a group of 24 adult controls were used. But, leaving aside these weaknesses in the study, a "blunted response" is not a "paradoxical effect". A slightly less pronounced effect is not the same matter at all as some kind of reversed effect whereby what is harmful for one person magically becomes beneficial to another.

The key point is that stimulants effect all people in the same way even if a finding can be produced with a dividing study that there is a statistical association between a somewhat increased resistance to stimulants and possession of an ADHD label. It is the same effect going in the same direction. There is nothing "paradoxical" about it.

The "paradoxical effect" claim is an appeal to pre-rational magical thinking.

Volkow *et al.* 2007 also provides an example of the lack of certainty in this putative science of brain chemistry. They cite a number of earlier studies which produced results which stand in contradiction to theirs. The mechanisms by which methylphenidate "works" to reduce the "symptoms of ADHD" are not clear.

<u>iii) There is no scientific or medical basis at all for the prescription of stimulants to impulsive/inattentive young people</u>

There is no coherent scientific explanation for how stimulant drugs are supposed to work. Unbelievable though it might sound every day in the UK young people are being given powerful drugs which effect the central nervous system without the pharmaceutical companies who make them or the psychiatrists and paediatricians who prescribe them being able to offer a clear explanation of how they work.

The authors of the NICE Guide are not completely certain about methylphenidate:

> Methylphenidate is a CNS stimulant. While the mechanism by which it reduces symptoms in ADHD is not completely clear, it is believed that it increases intrasynaptic concentrations of dopamine and noradrenaline in the frontal cortex as well as subcortical brain regions associated with motivation and reward (Volkow et al., 2004). [10]

Volkow *et al.* 2004, (an earlier study by the same author of the Volkow *et al.* 2007 paper we discussed above), did indeed associate methylphenidate induced dopamine increases with enhancing the "saliency of an event". However, in Volkow *et al.* 2004 methylphenidate *only* increased dopamine levels when the subjects were *also* given an interesting task to do. Giving the subjects methylphenidate and a neutral task did not result in increased levels of dopamine:

> Methylphenidate, when coupled with the mathematical task, significantly increased extracellular dopamine, but this did not occur when coupled with the neutral task. The mathematical task did not increase dopamine when coupled with placebo. [11]

NICE report that Volkow *et al*. 2004 found that methylphenidate increases dopamine levels. But they failed to mention that this was found only when the subjects were also given a challenging task to do. The actual result points the way towards educational strategies around making educational tasks more interesting. Volkow *et al.* 2004 conclude:

> These findings support educational strategies that make schoolwork more interesting as nonpharmacological interventions to treat ADHD. [11]

A paper which points the way towards educational interventions is used by NICE to promote drugging. As we shall see in the next section, such selective handling of the material in their "evidence base" is not at all unusual for the authors of the NICE ADHD Guideline.

As we saw in the last section Volkow *et al*. 2007 are candid enough to admit that different studies have produced different results. For example Volkow *et al*. 2007 found a "blunted response" to methylphenidate in the ADHD group. However; an earlier study found exactly the opposite. The earlier study (Rosa-Neto *et al*. 2005) [12] found that there was a positive correlation between "ADHD symptoms" and methylphenidate induced levels of dopamine. In Rosa-Neto *et al.* 2005 more "symptoms" meant more receptivity to methylphenidate, not less. Volkow *et al*. 2007 discuss possible reasons for these contradictory findings, including the fact that the subjects in the earlier study were young people and those in their study adults. However; it is striking that these findings were diametrically opposed. The situation with methylphenidate, dopamine, and inattentiveness is thus both far less certain and more complex that the NICE authors would have us believe with their "not completely clear".

The manufacturers of Ritalin, the original preparation of methylphenidate, are somewhat more cautious than the NICE authors about how their product "works":

> There is neither specific evidence which clearly establishes the mechanism whereby Ritalin produces its mental and behavioural effects in children, nor conclusive evidence regarding how these effects relate to the condition of the central nervous system. [13]

It is not just methylphenidate about which there is no certainty about what it is actually doing to the brain. This is the NICE ADHD Guideline authors explaining what is known about atomoxetine hydrochloride (Strattera), the one non-stimulant drug used to “treat” “ADHD” in the UK:

> Its precise mechanism of action in the treatment of ADHD is not clear but it is thought that it works by selectively inhibiting the pre-synaptic noradrenaline transporter thus inhibiting noradrenaline reuptake. [14]

No certainty there either. ADHD drugs studies “measure”, usually using parents and teachers as the raters, reductions in “ADHD symptoms”. They do not ask the young person how they feel. The behaviours of the subject are measured but he is, typically, not consulted. This lack of consultation means that there is little concern for how the drug may be “working” to achieve its effect. The change in behaviour may be caused by a positive drug effect or a negative drug effect. The positive drug effect might be, for example, that the drug facilitates the increased production of a chemical in the brain which helps with attention. A negative drug effect might be for example that the effect of taking stimulant drugs throughout the day leads to an inevitable evening “come-down” effect. The young person is suffering from drug exhaustion and is simply too tired to “argue”, “act smart” etc. Because of the exclusive focus on observed behaviours a negative drug effect is likely to be regarded as “positive” if it achieves the desired change in behaviour. Omitting real consultations with the “patients” i.e. the drugged young people from the studies and focussing entirely on parent and teacher ratings of behaviour means that this kind of misinterpretation of negative drugs effects as positive results is extremely likely. The “clinical” posture complete with clip-boards, check-sheets and statistical algorithms is less rather than more scientific.

ADHD drugging relies heavily on Victorian notions that authorise parents and teachers to speak for the “child”. The decision to administer drugs is made by parents and teachers. The definition of “improved” is made by psychiatry and measured by teachers and parents. The “child” has very little role in this other than as a mouth to swallow the drugs and an objectified little being whose behaviour can be recorded and assessed.

ADHD research studies which show some kind of brain abnormality or difference in “children with ADHD” are all based on averages across groups and statistical comparisons with the normals. In any one “clinical” case there is no test “for ADHD”. Which, anyway, “does not imply a medical or

neurological cause". [15] Therefore when a doctor prescribes drugs "for ADHD" she has *no idea* what is going on in her patient's brain. The prescription of these powerful drugs is based on a guess. The "chemical top-up theory" which is essentially the theory behind this guess states that "children with ADHD" have reduced dopamine levels and explains that the drugs raise the levels of dopamine. But since there is no diagnosis for ADHD and since it is all a matter of statistics some, at least, with the label will have perfectly normal brains - with no abnormality and no chemical deficiency. What will happen to their brains when they are given their top-up? This will be the equivalent of "healthy young people" taking stimulants such as amphetamines or cocaine. According to information published regularly by the government about the dangers of taking stimulants, they will be very seriously harmed. [16]

Let us imagine that a test for dopamine shortage or resistance to methylphenidate was developed and prescription of stimulants was only in these cases. It *still* wouldn't follow that prescribing methylphenidate would be a good idea. To assume that this would be a good idea requires a purely mechanistic view of a human being and the human brain. The implicit model in this process is that the human brain is like a bucket. If the level of a certain chemical in the bucket is 1% below the average level for people ("children") of that age we should just top it up. Obviously human beings are much more complicated than this. The brain is complicated. The drugs can change one variable in the brain but there is no understanding of the whole and we cannot, therefore, be sure that changing this one variable with drugs is a suitable "treatment". It may be that in the case of a young person with increased resistance to methylphenidate and/or reduced levels of dopamine their brain produces just the right amount of dopamine for their particular brain structure. We don't know that this is not the case. The dopamine top-up theory is a convenient *folk-truth*, not science. It is difficult not to see it in terms of the marketing objective of selling more pharmaceutical products and raising the stock price of certain US pharmaceutical companies.

iv) The ADHD drugs market is large and growing

The table below shows the net ingredient cost of all drugs used to "treat ADHD" on the NHS in England alone over the last few years (that is not Wales, Northern Ireland or Scotland):

| **Year** | **Number of prescription items dispensed '000 s** | **Net ingredient cost £ million** |
|---|---|---|
| 2004 | 434 | 14 |
| 2005 | 486 | 19 |
| 2006 | 562 | 23 |

| | | |
|---|---|---|
| 2007 | 655 | 26 |
| 2008 | 699 | 29 |
| 2009 | 744 | 31 |
| 2010 | 804 | 34 |
| 2011 | 861 | 39 |
| 2012 | 937 | 42 |
| 2013 | 1020 | 45 |

*Notes*:

a) Figures have been rounded to nearest whole number.

b) Source: NHS Information Centre [17]

c) The table summarises figures for British National Formulary category 4.4 excluding the chemical entity Modafinil which does not appear to be used to treat ADHD. Some of the drugs may have been used for “conditions” other than “ADHD”; for example Dexamfetamine Sulphate can be used to treat narcolepsy. Conversely some other drugs may have been used to “treat ADHD”.

d) These figures do not include drugs administered in hospitals.

e) These figures do not include those issued for private prescriptions.

f) A prescription item is an item indicated on a prescription form, for example a bottle of pills.

The figures show that there has been substantial growth in the ADHD drugs market over the last 10 years. The market in England alone for all ADHD drugs in 2013 was £45 million. This included £32 million for methylphenidate. [17] This figure excludes private prescriptions and drugs administered in hospitals so the true size of the market is actually larger.

The market for ADHD drugs in the US is vast by comparison. The US “medicates” more young people with methylphenidate per capita than the UK. The ratio was 1.25:1 in 2003 [18]. The population of the US is approximately approximately 6 times greater than that of England. As a very rough estimate this produces a figure for the US market for methylphenidate alone, (not the other ADHD drugs such as the amphetamines), of at least £240 million in 2013. This is compatible with the figure of US consumer sales of Ritalin (branded methylphenidate) in 1995 being USD 349.3 million, approximately £222 million, provided by marketing consultancy IMS America, quoted

by Dr Peter Breggin. [19]  One study gives a figure for the total worldwide market for all “ADHD medications” in 2003 as being USD 2.4 billion. [20]

Marketing drugs “for ADHD” then is very big business. The market is expanding. In England the market has grown year on year at a steady rate since 2004.

The majority of the cost of ADHD stimulant drugs in the UK will be met by the taxpayer. Doubts have been raised about how much control the NHS exercises over this expenditure. The story of dexamfetamine is a case in point. In 2009 dexamfetamine had been supplied to the NHS as the branded drug Dexedrine at £0.11 per pill. [21] In March 2010 a company called Auden McKenzie took over the license for Dexedrine. The MHRA then granted Auden McKenzie a change in the terms of their license to sell the generic dexamphetamine. [22] In 2011 generic dexamfetamine was supplied to the NHS at a cost of £0.58 per pill. [23]  This had risen to £0.68 per pill in 2013. [3] That is a rise from £0.11 per pill for branded Dexedrine in 2009 to £0.68 per pill in 2013 for the generic version. A rise of 600% for precisely the same substance. It is not possible to be certain that all the generic dexamphetamine bought by the NHS in England in the period 2011 to 2013 was supplied by Auden McKenzie as the NHS does not record the manufacturer of generic drugs which it purchases. Either way; the rise in cost is striking. In 2010 Auden McKenzie featured in a Daily Mail report about companies profiteering from drug sales to the NHS. [24]  In 2011 branded Dexedrine was still available to the NHS at £0.11 per pill. [23] But doctors were prescribing the generic substance dexamphetamine which was five times more expensive. Did they just assume that the generic would be cheaper and not check? Surely someone should have noticed and alerted doctors? In 2011 alone the actual loss to the NHS caused by this situation was approximately £2,000,000.00. Since this situation was completely avoidable the conclusion has to be that the NHS is not exercising strict budgetary control.

v) The drugs are extremely harmful

Documented and typical side-effects of stimulant drugs include:

- Insomnia
- Depression
- Nervousness
- Abnormal movements (Tics)
- Headache
- Stomach ache
- Weight loss
- Growth suppression
- Mania, psychosis and hallucinations

- Evening crash
- Cardiac complications (rarely)

This list to was put together by the ADHD critic Dr Peter Breggin from clinical trials. [25] Drug advocates tend to downplay the seriousness of the “side-effects” but there is no essential dispute that the above are the “side-effects” of stimulants. These side-effects are acknowledged by manufacturers of ADHD drugs. The manufacturer of Ritalin acknowledges a similar list of side-effects and states that insomnia and nervousness are the most common. [13] The pro-drugging MTA study described a similar list to the above (Section 2) vi)). And the MTA follow-up study found evidence of growth suppression. [26]

The most used ADHD stimulant drug in England is methylphenidate. This substance is similar to amphetamines and cocaine. Ritalin is one form of branded methylphenidate. The US Drug Enforcement Agency (DEA) comments on Ritalin:

> Ritalin is a Schedule II stimulate, structurally and pharmacologically similar to amphetamines and cocaine and has the same dependency profile of cocaine and other stimulants. [1]

In the following we compare the advice the UK government gives about amphetamines on the “Ask Frank” website aimed at young people [16] with the “adverse effects” of Ritalin as indicated by the manufacturer [13]:

> ASK FRANK: Speed (the 'street' name for amphetamines including dexamphetamine) can lead to agitation, panics or even a psychotic episode.
>
> RITALIN: Treatment emergent psychotic or manic symptoms, e. g., hallucinations, delusional thinking, or mania in children and adolescents without a prior history of psychotic illness or mania can be caused by stimulants at usual doses.
>
> ASK FRANK: Depending on how much you’ve taken, it can be difficult to relax or sleep.
>
> RITALIN: Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening.

ASK FRANK: Speed [amphetamine] puts a strain on your heart, so it's definitely not advisable for people with high blood pressure or a heart condition – users have died from overdoses.

RITALIN: Sudden death has been reported in association with CNS stimulant treatment at usual doses in children and adolescents with structural cardiac abnormalities or other serious heart problems

ASK FRANK: Speed makes people feel wide awake, excited and chatty

RITALIN: Ritalin is a mild central nervous system stimulant.

Both Novartis, the manufacturer of Ritalin, and the UK government are describing the adverse effects of taking the same type of drugs. The advice given by the British government about the use of amphetamines applies equally to young people given amphetamines, or similar substances, by a psychiatrist. There is no special magic that means that because a young person has an ADHD label attached they are suddenly immune from all these well-known harmful reactions to amphetamines and similar drugs. It is purely magical thinking to believe that amphetamines and other stimulants are harmful when taken voluntarily for recreation (or for self-medication) and are benign (a “treatment”) when prescribed by a psychiatrist. Yet the only way of balancing the two official UK narratives about stimulant drugs and young people is to subscribe to just this kind of primitive, magical, thinking.

In England another stimulant drug used “for” “ADHD” is dexamphetamine. Dexamphetamine is a member of the amphetamine family. In this case we can expect an even more direct correspondence between the effects of the drug and the lists of the hazards of amphetamines as described by the government. It is exactly the same substance.

Insomnia, nervousness and growth loss are typical reactions to ADHD stimulants for young people taking them. They not rare occurrences. In the MTA study, for example, 63% of “medicated” subjects reported “side-effects” such as insomnia and “Worried/Anxious”. (See Section 2) vi)). It is hardly surprising that sleeplessness is common. Stimulants keep people awake. Nor is it surprising that growth loss is common. Stimulants suppress appetite. (And may also interfere with growth hormones). [27] The pharmaceutical industry which now sells amphetamines and similar substances for “ADHD” has previously marketed amphetamines for appetite suppression to help with dieting and to keep people awake. What is an effect of the “medication” and what is a “side-effect”? The answer is that this appears to have more to do with changing marketing requirements than with medical science. Clinical researchers such as those who conducted the

MTA study lend their “scientific” credibility to these commercial re-purposing operations. It could be said that their role is to wrap the drug sales in scientific packaging.

One unfortunate pattern in ADHD drugging is that young people who are started on stimulants may end up taking a stack of drugs to combat the “side-effects” of the stimulants. One case study from the NICE Guideline provides an example. Parent E describes giving their son melatonin to counter the insomnia induced by methylphenidate. [28]

One of the MTA follow-up studies reported “Significant growth suppression”. [26] Novartis, the producer of Ritalin, admits that there may be a slowing of growth “without evidence of growth rebound during this period of development”. [13] The reference to a rebound relates to a claim by ADHD promoters that the retardation in growth is often reversed when the young person comes off “medication”. However, even where such a “rebound” (after “medication” is stopped) occurs it is not a natural process. It can hardly be healthy for young people to grow in drug modulated stop-start episodes. Furthermore, it may be that the growth loss reported as a result of long-term use of methylphenidate is not simply due to appetite suppression. There is some work to suggest that methylphenidate disrupts the normal cycle of growth hormone in the body itself. [27]

Strattera is a relatively new drug used to “treat” “ADHD”. It has been on the market since 2004. The chemical substance is atomoxetine hydrochloride. Atomoxetine hydrochloride is not a stimulant. The selling point of Strattera therefore is that the risk of its escaping onto the black market, for illegal use, is reduced. From 2004 to 2013 its use by the NHS in England has grown by about 700%. During this period prescriptions for methylphenidate have risen by about 230%. Prescriptions for dexamfetamine have fallen by about 30%. Strattera is thus gaining market share. Like the discovery of the beneficial effects of stimulants on concentration in “disruptive children”, the applicability of atomoxetine hydrochloride to treat “ADHD” was an accidental discovery. The drug failed as an anti-depressant and was re-purposed to “treat ADHD” “in children”. [2] Strattera's claim to be suitable “for ADHD” rests on the fact that a number of studies managed to show that ADHD “symptoms” were reduced by the substance. The authors of the NICE ADHD Guideline found 17 studies for Strattera which met their inclusion criteria. Of these, 16 were funded by Lilly, the company who makes Strattera. There was no funding data for the other one. [29] This then is not a medical-scientific process of discovery but a marketing launch. Indeed because there is no “medical or neurological cause” “for ADHD” there cannot be a medical-scientific process of research leading to a treatment, as there is, for instance for the HIV virus. All they can do is give the drugs to young people and count the reduction in symptoms, that is the reduction in the “disruptive” behaviours which constitute ADHD. A*nything* which reduces the “symptoms” / altered the behaviours would pass this test.

The average length of the 17 Strattera studies identified by NICE was 83 days. [29] ADHD drug

studies tend to be in the short-term but ADHD drugs are typically prescribed in the long-term. This means that negative effects which occur over the long term are unlikely to have been considered.

Like methylphenidate, Strattera has a long list of harmful side-effects. For Strattera there is a particular risk of suicidal thoughts and behaviour. [30] The authors of the NICE ADHD Guideline report that:

> In double-blind clinical trials, suicide related behaviours occurred at a frequency of 0.44% in atomoxetine-treated patients (6 out of 1,357 patients treated, one case of attempted suicide and five of suicidal ideation). The age range of children experiencing these events was 7 to 12 years. There were no events in the placebo group (n = 851). It should be noted that the number of adolescent patients included in the clinical trials was low (Eli Lilly and Company Ltd, 2008). [31]

It is not possible to know how many young people are being prescribed atomoxetine as the NHS does not keep figures for individuals being “treated”, just for the overall numbers of prescriptions issued. It is however, possible to use the figures that are available for prescriptions issued to extrapolate to likely numbers of individuals being treated. [32] If our estimates for atomoxetine hydrochloride of 56,500 are correct we can extrapolate directly, based on clinical trial “evidence” reported by NICE to a likelihood of 41 attempted suicides related to atomoxetine in 2013 in England and about 250 cases of suicidal ideation. (This assumes that the double-blind clinical trials reported by Lilly lasted less than a year which is highly likely). If the authors of the NICE ADHD Guideline use “clinical trial evidence” to make claims for “symptom reduction” surely they should also consider and report on the potential for suicide evidenced by the same “clinical trial evidence”? Is the “evidence from clinical trials” only taken seriously if it can be used to promote drugging? Or; do the authors of the NICE Guideline simply believe this level of drug-related suicidal behaviours in young people is an acceptable price to pay for reduced levels of “squirming in seat” and “getting up from seat when remaining in seat is expected”?

The suicidal behaviour predicted by the clinical trials reported by NICE has come to pass. We asked the UK's Medical and Healthcare Products Regulatory Agency (MHRA) for figures on “adverse events” reported for Strattera (atomoxetine hydrochloride) between 2003 and 2012 in the UK. [33] The figures include the following types of adverse events grouped together: intentional self-injury, self-injurious behaviour, self-injurious ideation, suicidal behaviour, suicidal ideation and suicide attempt. Overall 137 adverse events of this nature were reported during this period. 122 were in under 18s, 3 in people aged 18-24 and 9 where no age was supplied by the reporter. Looking at the detail it is possible to provide a break-down to some extent. There are 106 cases of suicidal ideation and 12 suicide attempts. (We have not counted suicidal ideation where there was also a suicide attempt). Of the 12 suicide attempts eight show “recovered/resolved” or

“recovering/resolving”, though in one case the status of “brain injury” is unknown. Two show “not recovered/resolved”. Two show “unknown”. It may be reading too much into the figures but “not recovering” from a suicide attempt would generally mean death. Reporting to this scheme is not mandatory so these figures will be an under-representation of the true extent of the suffering seen by doctors and psychiatrists. This may be the case by a large margin. Furthermore, many young people will suffer suicidal ideation or may self-injure in various ways without their parents or paediatrician or psychiatrist even becoming aware of it. Young people who self-injure often do so in secret.

It should be noted that these reports are of adverse events when a young person is on atomoxetine. The reports do not show that atomoxetine caused the suicide attempt in any one case. Nonetheless the reporting criteria is that there is a suspicion that there is a connection. Furthermore, the evidence from the clinical trials is that atomoxetine causes suicidal behaviours. This is the case because there were no events in the placebo group. We can reasonably assume therefore that some, possibly most, of the adverse events of suicidal ideation or suicide attempts monitored by the MHRA would not have occurred had the young person not been on atomoxetine.

In terms of the clinical evidence the NICE Guideline authors blandly commented:

> There is evidence suggesting that atomoxetine may increase side effects when compared with placebo and when compared with methylphenidate. [34]

The clinical trials quoted by the NICE Guideline authors *predict* that suicide attempts will occur. The adverse event reporting from the MHRA *confirms* that suicide attempts and successful suicides *have occurred.* This is real. Giving atomoxetine to large numbers young people will lead and *is leading* to suicides that would not have occurred otherwise. This is what the “clinical evidence” says. In terms of “side-effects” suicide is final.

In 2005 the US Food and Drug Administration (FDA) issued a “black box” warning for Strattera (atomoxetine) for posing a risk of causing suicidal thinking in children and adolescents. [35] A “black box” warning is deemed especially severe. It requires the manufacturer to give prominence to the warning. The manufacturers responded by saying:

> There were no suicides among children, adolescents, or adults on the medication during any Strattera clinical trials and there was no indication of an increased risk of suicidal thinking in the adult population. [36]

This is “spin” which neatly seeks to bypass the finding of increased suicidal thinking in young

people. The level of irresponsibility apparent in these comments from the manufacturer is staggering. While releasing a drug which is known to lead to suicidal ideation and suicide attempts in young people they simply look the other way. The Health policy bodies and the regulatory agencies all also appear to be looking the same way. That is, looking away.

In 2012 the US Food and Drug Administration issued another warning for Strattera (atomoxetine) in connection with a rare but potentially serious risk of liver damage. [37]

In general terms the manufacturer of Strattera advises:

> The most common side effects in children and teenagers include upset stomach, decreased appetite, nausea or vomiting, dizziness, tiredness, and mood swings. [38]

Which hardly sounds like much fun for a young person.

Good medical practice is that the advantages of taking a drug should outweigh the disadvantages. This is the stated policy of the UK's Medicines and Healthcare Products Regulatory Agency which states that the key questions which they ask when considering whether to license a drug are:

> Do the advantages outweigh the dis-advantages of taking the medicine?
>
> Does the medicine do the most good for the least harm for most people who will be taking it?
>
> Are the side effects acceptable? [39]

It is very hard indeed to see how those questions can have been seriously asked when the MHRA was considering issuing licenses for the drugs which are used to “treat” young people “for ADHD”. Most of the drugs used in the UK, including Strattera, Ritalin, Concerta (a preparation of methylphenidate), and dexamphetamine have been licensed by the MHRA.

<u>vi) It is about authority not medicine or science</u>

There is anecdotal evidence that schools in the UK are pressurising parents into “getting an ADHD diagnosis” and drugging their children. The Daily Mail interviewed one mother who reported that:

> His school told Andrea Ruben faced exclusion unless he took drugs to control his

> behaviour. [40]

Another case is reported by the Guardian:

> Take Leon. He insists he didn't want to start taking Ritalin. His mum didn't want him to, either. It was his last school that gave him an ultimatum: go on the drug and act with more respect, or leave the school. [41]

The already dis-empowered “child” is reported to the psychiatrist. The psychiatrist makes a “diagnosis”. The central point is that there is a deficiency. And it is in the “child”. The child “has” something. Everyone else can relax. The child certainly can't, because now he will be stuffed full of stimulants which prevent him relaxing.

In the ADHD world the focus in on making the behaviour of the young person align with the expectations of adults. It is taken for granted that this is a valid goal. The shift to a “clinical” interpretation and framing serves to avoid any requirement for those on the side of authority to change so as to meet the *actual* needs of the young person, as they are. Once a “child” is said to “have ADHD” then a formulaic “treatment” is ordered. This may be drugging or a behavioural programme. Even if the intervention is a parenting programme this is still constructed as helping the parents to manage their “ADHD child's” behaviour, for example: “to optimise parenting skills to meet the above-average parenting needs of children and young people with ADHD”. [42] This sounds sympathetic but is based on the usual reification; “the children and young people with ADHD”. Everything is predicated on the problematised “child”. Whatever it is it is *his* problem. This is still an objectification of the young person. The authentic relationship between adult and young person wherein actual needs might be met is obliterated under this manipulative framework. This location the problem *in* the child resembles moral narratives about “children” from the Victorian era. The “clinical” picture is superimposed on this essentially moral, and always potentially punitive, practice. The moral nature of psychiatry is expressed by Foucault:

> What we call psychiatric practice is a certain moral tactic contemporary with the end of the eighteenth century, preserved in the rites of asylum life, and overlaid by the myths of positivism. [43]

An interesting example of the moral themes underlying the ADHD narrative is provided by Singh 2007 with a paper “Taking Methylphenidate for ADHD. Clinical Implications of Ethical Concepts: Moral Self-Understandings in Children”. [44] Sing 2007 is a confusing paper. Singh appears to believe that she is taking issue with a certain strand in dialogues which are critical of the ADHD

drugging programme. These strands emphasise the “naturalness” of the individual and, apparently, promote an ethics of personal authenticity. Singh attributes the “natural” character of the “child” position to a writer D. Brock. Singh associates the point of view which emphasises the “uniqueness and individuality” of the self as a reference point for morality to an academic philosopher Charles Taylor. Singh believes that these views are used to develop an argument that ADHD drugging should not be allowed because it undermines these “innate dimensions” of the person. Singh however appears to have completely misunderstood the philosopher Charles Taylor. Singh writes that:

> The philosopher Charles Taylor (1991) describes an ‘ethics of authenticity’ as the self’s sense of its own uniqueness and individuality, and the desire to be true to this self (Abbey, 2000). [44]

In fact Taylor argues against this view. He criticises the idea that Singh outlines here about ethics being grounded in the “self’s sense of its own uniqueness” as being part of the “soft relativism” of contemporary culture. He argues that soft relativism “self-destructs”. It does not base choice on values with are given, beyond the self, e.g. from “the needs of my fellow human beings, duties of citizenship, or the call of God, or something else of this order...”. Thus while it can celebrate choice the choices this kind of personal ethics makes can only be trivial ones. [45] In as much as she is concerned about whether the “natural self” of the “child” is undermined by stimulant drugging this does not effect the thread of Singh's argument. However; it does mean that she has failed to put the argument onto a philosophical basis. If you want to argue against an “ethical” and philosophical argument that is critical of ADHD drugging you do at least need to find such an argument to argue against. Taylor doesn't provide it.

Singh sets out to show that in fact “childrens'” “sense of personal authenticity” is not undermined by stimulant drugs. She does this by showing, by using questionnaires with a small sample group of “children with ADHD”, that their moral judgements of their own selves are that they are “bad”. The drugs haven't caused this. They assess themselves as “bad” “despite medication”:

> Second, children’s moral conceptions of their authentic selves are characterized by persistent badness, despite medication. [44]

A key part of the argument depends on the idea that the “moral” judgements that a young person (aged 8 to 12 in her study) pass on themselves constitute their “authentic self”. If a young person says “I am bad” that means we can say “his authentic self is bad”. This notion that self-statements of this kind somehow determine the nature of what might be called an “authentic self” is difficult to apprehend. In this paper various concepts from the fields of genetics, psychology and philosophy

have been uprooted from their situation in their own narratives and elided together. To this already confused blend is then added a strong sense of “morality” which appears to be Dr Singh's own. In this morality being “bad” equates with “doing something wrong”. “Doing something wrong” appears to mean doing something for which a “child” might be told off by a parent or teacher.

The main thread in Singh's paper appears to be:

1. Statements “children” make about their “moral selves” can be taken as true statements about themselves. If Johnny says he is bad he is bad.

2. The “children” interviewed in the study said they were “bad” despite “medication”.

3. Therefore “medication” does not harm the “authentic selves” of children. They were “bad” before the “medication”.

4. Since “medication” does not harm the “authentic selves” of the “children” there is no reason to give them a break from the drugs at week-ends for them to be their “natural selves” as is apparently sometimes the case. One potential “clinical implication” therefore of Singh's work is that this practice should be stopped.

Dr Singh introduces her “empirical” research with a theoretical discussion in which she seeks to establish the principle that what “children” say about themselves in terms of moral self-evaluations can be taken as statements about their “authentic selves”. Having established this she then carries out interviews with 23 young people aged 8-12 all with an ADHD label and all being drugged with methylphenidate. The interviews were carried out using a “binary” method which presents binary alternatives such as being “on your tablets” or “off your tablets” and of course a binary morality of “good” versus “bad”. The idea is that the “children” say that they are “bad” even without the drugs. Thus it is proved that it isn't the drugs which make them “bad” (undermine their “personal authenticity”). Maybe drugs even make them good. The study fails at a theoretical level. This failure occurs in two principal ways. Firstly; Singh confuses her old-fashioned morality of the nursery where being “bad” means doing something which might make an adult “reprimand” a “child” with a philosophical discourse about authenticity. But the “authentic self” of philosophical discourse is not a moral self let alone one based on this nursery morality. “Authenticity” is not about doing what your parents tell you (or not). Singh's morality elides into ethics elides into concepts about “authenticity”. “Naughtiness” which is usually understood as a transient state of childhood is confused with the deeper philosophical discourse about authenticity. The “children“ may well be “naughty”. However, they may still have a “natural self” worth defending. Secondly; it is not the case that statements that someone makes about themselves necessarily can be taken to describe something called their “authentic self” or even their “core self”. People can make self-

statements which can be wrong. Just because some of the young people say they are “bad” doesn't mean that they are “bad”.  In the following we review Dr Singh's attempted argument.

Here is Singh explaining the “natural self” position:

> For example, Brock (1998) has argued that as a unique individual, a child’s ‘character, capacities and life history should be permitted to unfold according to its own nature’ (p. 62). [44] [46]

Having elided this concept with that of “authentic self” Dr Singh explains how she has determined that what a young person says about their themselves can be taken as statements about their “authentic self”:

> These assumptions of a core aspect of the self can be viewed as theoretically analogous to arguments for a genetic basis to personality and temperament in the field of clinical genetics. However, the genetic research on personality strongly suggests a gene–environment interaction: Genetic predispositions to temperament outcomes or to psychopathology can be triggered by environmental stressors; or the environment can have a protective function (Caspi et al., 2002). [44]

and

> If genetic predispositions interact with environmental factors to create distinct persons, then children’s conceptions of core or stable aspects of themselves, as reported in this study, can be viewed as the expression of an emerging or developing sense of the authentic self. [44]

Dr Singh believes that in the above she has justified using the self-statements she manages to produce from her sample of 8-12 year olds as true characterisations of the actual nature of their “authentic” selves. However; the problem here is that “authentic self” is a philosophical concept. Singh may believe she can map (“analogous”) ideas about personality from the fields of social psychology and genetics to this concept but she may be alone in this belief. She hasn't demonstrated or proved this mapping. On the whole the concepts belong in different types of discuses and cannot be so crudely mapped. In philosophy ideas about an authentic self are not “assumptions” as Dr Singh terms them. Or; if she wants to assert that philosophy is just non-empirical chatter which makes “assumptions” then she needs to demonstrate that. But, if she could do this, she would, to borrow a phrase from Charles Taylor, self-destruct her own argument which depends on the existence of an “authentic self”. This is all confusion and Dr Singh has not

established that persistently made self-statements are descriptive of the “authentic self” of a person. She could not because she is mixing discourses. Like most positivists Dr Singh has leapt from empirical science into the field of philosophical discourse, *collapsing* the philosophical concepts in the way. The starting point of positivism; that only empirically established facts have any meaning, is used to crudely destroy other discourses. Dr Singh can get a group of 8-12 year olds to say whatever she wants, but this is not a discussion about what constitutes the “authentic self” of a person. Nor even what constitutes the “authentic self” of a “child with ADHD”.

The “natural self” argument is a value argument. The argument is quite well put in the quotation from Brock provided by Singh we which cite above. The value claim is that whatever young people are in their natural state we should not interfere with that even if in certain areas they might have less “capacity” then the average for their peers. We should not try to fix “deficits” with drugs. This value claim remains even if a “child with ADHD” can be shown to view himself as “bad”. The self-statement about being “bad” does not mean that the young person *is* rotten in his core. It is just a self-statement. Singh's reductionist argument from genetics does not allow the possibility of a young person adopting a self-image which is not an accurate assessment of how they actually are. Even if self-statements are always defining of how someone actually *is*; Singh makes yet another assumption which is that “badness” should always be corrected. This is the imposition of her nursery school morality onto her “empirical” data.

With her amateurish forays into philosophy Singh has failed to provide the basis for the theoretical aspect of her paper. However; even the “empirical” case is fraught with problems. It is necessary for her argument that the statements young people in her study make are genuine and unfettered. However, it is unlikely that this is the case.

The sample included 20 boys and 3 girls. Thus confirming the extraordinary gender bias in ADHD “diagnosis”. Almost all ADHD studies have groups with a massive preponderance of boys. The usual response, if it it discussed at all, is to kick the problem into the long-term as a subject for “future research”. Singh follows suit:

> Boys make up 75–80% of ADHD cases; therefore it is more difficult to recruit girls into research on ADHD. The small number of girls in the current study (3) problematizes a gender analysis. However, the gender question may be particularly important to explore further, given that the developmental literature views gender as a critical component of self-understanding and self-appraisal (e.g. Gilligan et al., 1991). In order to adequately explore issues of gender, future research may need to oversample girls. [44]

Thus all the questions that should arise in connection with the gender disparity in the studies are

simply avoided.

Dr Singh based her “empirical study” on a series of interviews with a small group (23) of “children with ADHD”. In the interviews the young people were shown pictures and asked for their responses. For example one picture was “a standardized picture of a child being reprimanded by an adult”. The young people were aged 8-12 and all were dosed with “stimulant drugs”. The “interviews” were conducted in the homes of the young people and one parent was present in the home at the time of the interview. Dr Singh openly admits that some of the parents involved in this study drug their children at week-ends to stop them being “too naughty” - a phrase she appears to quote from the parents. Dr Singh is aiming to demonstrate something about the “moral self-understandings of children”. But, some at least of the parents involved clearly think their children are “naughty” and that methylphenidate is the answer. And at least one of these parents was present when the interviews were being conducted. This doesn't bode well for obtaining statements from the young people which are not simply reflections of what their parents have told them.

The interviews themselves were pre-loaded. The “children” were told (for example):

> All the children in these pictures have ADHD, like you, and they take Ritalin tablets to help them. [44]

This manipulative question prevents, or makes it extremely hard, for a young person to say anything other than Ritalin “helps them”. Dr Singh then discusses what the young people said about “medication”:

> They understood medication as something that helped them be good, and they were aware of, and worried about, the ‘bad’ part of them that could enjoy hurting or harming others. [44]

You cannot tell someone something and then present it as an “empirical” finding if they tell you what you have just told them.

It would take a brave and unusually independent 8 year old in these circumstances to state categorically that being on “medication” made them feel worse than not being on “medication”. Nonetheless out of twenty boys and three girls in the Singh 2007 “study”:

> One boy presented a reverse binary to the majority of the sample. He reported feeling happier *off* medication and sad *on* medication. [44]

From the interview snippets provided in the paper it is not in fact clear that the other 22 subjects did report being "*happier* " "on their tablet".

The paper is intended to demonstrate something about the "moral self-understandings of children". The questions were all framed in terms of what Dr Singh calls a "binary" format. They invited the young people to say how things were for them "on and off medication". To make sure that results were produced which enabled claims to be be made about "moral self-understandings" questions were framed in terms of "morality". For example here is an extract from Mark's interview:

> Another child, Mark, elaborates a similar narrative about the relationship of medication to his good/bad self:
>
> Interviewer: If you had taken your tablets and you hit someone and hurt them on purpose, would you be a bad person?
> Mark: Mmmm. It would be a bit of both.
> Interviewer: Bit of both . . . in what way?
> Mark: Bad, and good then.
> Interviewer: Oh, cos you said it makes you feel good when you hit someone?

and

> You're saying that there's a bad part of you that the tablets can't make good?
>
> Mark: Yeah, inside I might be evil. I need the tablets to make me good but they can't take away all the evil.
> Interviewer: So if I were to ask you what you think is the 'real' you – the bad part that the tablets can't make good, or the good part with the tablets . . .
> Mark: Well of course I'm not real with the tablets!
> Interviewer: So the real you is the bad you?
> Mark: I think so.
> Interviewer: How does that make you feel?
> Mark: Ok. [pause . . . 3 seconds] As long as I have the tablets! [44]

In the dialogue above we can note that the term "bad person" is introduced by the interviewer to Mark. Mark accepts the term. The interviewer then follows up the advantage with "You're saying that there's a bad part of you....". The concept of "real you" is similarly introduced to Mark by the interviewer. This is not looking very "empirical". And indeed if this really was an "empirical" study

about whether young people understand methylphenidate drugging as undermining their “authentic selves” it would appear that Mark has given a completely clear answer:

> Well of course I’m not real with the tablets! [44]

Naturally; this rather unambiguous answer does not appear to influence the results!

Singh started her paper with a discussion about authenticity and ethics. But she attempts to substantiate her thesis about methylphenidate not harming the “authentic selves” of “children” with a nursery morality about “doing something wrong”. Somewhat alarmingly Singh appears to believe that if an adult is “angry” with a “child” this means that the “child” has done “something wrong”. At least one of the interview questions is described a young person being shown a picture of an “angry” adult and the young person being asked if he could have helped his behaviour:

> [*In pictures where adult is angry and child has not taken tablets]*: Can this child help it that he did this? [44]

Simon is asked how he feels when he “has done something wrong”:

> Interviewer: When you’ve taken your tablets and you’ve done something wrong...
> Simon: Yeah?
> Interviewer: How do you feel inside?
> Simon: Bad.
> Interviewer: But when you haven’t taken your tablets, and you do something wrong...
> Simon: [interrupts] I feel good about it! [emphatic]
> Interviewer: Do you like that feeling of feeling good inside?
> Simon: Yeah. Wait. What do you mean by ‘good’? Do you mean doing something bad and I feel good inside?
> Interviewer: Yeah.
> Simon: No, I don’t like that. I feel bad about myself. [44]

Singh is troubled by Simon feeling “joy or glee in his had behaviour”:

> Simon does have experiences in which he feels joy or glee in his bad behaviour when not taking medication, but he also understands that these good feelings are not appropriate. [44]

Singh appears to credit methylphenidate with the power to instil moral feelings in Simon because he feels “bad” about his “bad behaviour” when he is on drugs but when he is not on drugs he feels, at least temporarily, “glee” about his bad behaviour.

Tommy is also offered as evidence that young people believe that methylphenidate helps them “behave”:

> Tommy: That’s me acting like a crazy monkey.
> Interviewer: You’re acting like a crazy monkey?
> Tommy: Yeah, like this . . . ahha ahhh [monkey sounds].
> Interviewer: So is that when you’ve taken your tablets, or when you haven’t taken your tablets?
> Tommy: Haven’t. That’s really fast.
> Interviewer: And how does it feel to be this crazy monkey?
> Tommy: Really quick.
> Interviewer: Really quick.
> Tommy: Ohhhahhha. Very movable, like that.
> [Pause 6 seconds]
> Tommy: Hmm. Do you feel like you can control this crazy monkey that you’ve become?
>
> Tommy: Not quite . . . well, you can’t really tell . . . if I’m going to be able to control it. Cos sometimes you can control it, and sometimes you can’t. Sometimes when my mother says, ‘Stop!’ I can do it, and sometimes when my mom says, ‘Stop!’ I carry on doing it.
> Interviewer: Why do you think that is, that you can stop sometimes and not others?
> Tommy: Cos I think your brain sends messages inside the body.
> Interviewer: So your brain says . . .?
> Tommy: Carry on because I don’t have any tablets.
> Interviewer: Oh, your brain says, ‘Carry on because you don’t have any tablets’.
> Tommy: Yes.
> Interviewer: Do the tablets stop your brain from making you behave that way?
> Tommy:Yes.

Singh interprets this as Tommy attributing to methylphenidate the power to give him self-control. An alternative interpretation would understand this in terms of drug dependency. Recall that Tommy may have been told by his parents that he is being given the drugs to control his “naughtiness”. In as much as he has come to believe that being “naughty” or “not naughty” is something which is

controlled by being on or off tablet he may allow himself to be “naughty” when he is “off tablet”. Thus he develops a dependency on drugs. Telling young people that it takes drugs to stop them being “naughty” will discourage them from learning how not to be “naughty” without drugs.

Dr Singh believes that the young people think that methylphenidate “makes them good”:

> They understood medication as something that helped them be good, and they were aware of, and worried about, the ‘bad’ part of them that could enjoy hurting or harming others. [44]

But she isn't completely sure about this:

> In fact, not one child in this study consistently attributed lack of control to a lack of medication. Within and across interviews, explanations varied from ‘I guess I just don’t care enough to stop’ and ‘I don’t want to listen’ to a lack of medication or insufficient medication. [44]

At any event it is clear that the moral dimension of the narrative produced by these interviews is something which has been imposed onto the dialogues by the parents and by the interviewers. One of the parents was present when the interviews were conducted. The young people have been told that methylphenidate “helps them”. The interviews consist of leading questions about “your real self” and being “a bad person”.  In these circumstances the possibility of the answers provided by the young people giving any objective truth outside of the constructed context in which they found themselves is absolutely zero. Somewhat strangely, Dr Singh appears to admit that her structured questions and the context in which they are asked determine the outcome:

> It may be that the experience of being identified as a problem child is itself enough to produce these responses in children. [44]

and

> These binary representations of the self/behaviour on and off medication make up one level of discussion in interviews with children. These representations are notable in that they appear to demonstrate a lack of cognitive sophistication in these children; the tendency to structure descriptions of the self and behaviour as unintegrated opposites is identified with the cognitive skills of 5–7-year-olds (Griffin, 1992; Harter, 1999). However, these representations should probably not be taken to reflect cognitive immaturity in these children; rather, they are better explained by the structure of the

> question children were responding to when taking their photographs... [44]

Singh appears to believe that the “experience of being identified as a problem child” could be controlled for in a future study. But it is difficult to see how a study could be conducted “with ADHD children” and at the same time eliminate the effect of being “diagnosed” from their experience.

Singh explains that it doesn't matter if the young peoples' “moral self-evaluations” are authentically their own or reflect what their parents have told them:

> Children expressed fear, sadness and loneliness in relation to all these worries: ‘I’m always in trouble because of how I behave and it makes me sad’. It is impossible, and probably not necessary, to know whether these worries are derived spontaneously out of children’s own sensibilities, or imposed by carers’ refrains about the potential implications of out of control behaviours. The important point is that for many children in this study, their jumpy insides and difficult-to-control bodies were a site of complex and ambivalent self-understandings. [44]

But, if your case depends on an explanation about “children's self-understandings” surely it does matter if what the “children” say in the interviews is what they think, or what their parents have told them?

This study revealed that some “children” talked about the “side-effects”:

> Some children discussed one further dimension of physical behaviour: *Side effects of medication*. Here too, photographs yielded binary representations: Children reported that when on medication they had little or no appetite, had trouble sleeping, had headaches or tummy aches. Children reported having no such troubles when not taking medication. [44]

This reporting by young people of the negative effects methylphenidate has on them does not seem to influence the “potential” clinical recommendations formed from the research which recommend more consistent dosing (extending “dosing” at week-ends and holidays and not just during the school-week) [44]. Approaches such as this while appearing to “consult” young people are not really doing so seriously. The young people are not asked the meaningful question “would you like to stop taking these tablets now?”, as a real question where if they answered “yes” then

they would stop being given the drugs. They are being asked for their views about a given situation, organised by all the adults around them which they know is going to continue whatever they say. Like all young people being abused by adults these young people will give adaptive answers. Such “consultation” exercises with “ADHD children” consistently produce the same answer. The “children” approve of their “medication” and voice a quiet, permitted, protest about the “side-effects”. That won't help them because clinicians like Dr Singh have already decided that methylphenidate has a “tolerable side-effect profile”. [44]

Singh (2007) is a travesty. The aim appears to be to counter an argument that even if some young people have certain deficiencies they should not be drugged because their “natural selves” have a value in their own right. Singh's method appears to be to “demonstrate” that the young people (in her sample) see themselves as “bad” “despite medication”. “Bad” is somehow equated to the concept of “natural self” or “authentic self” and the argument appears to be: because the young people say they are “bad” they are “bad”, in their “authentic selves”. Therefore they are already “bad”. Therefore “medication” cannot make them any worse. Therefore the argument not to drug them because it harms their “natural selves” fails. (In essence the argument appears to be that these young people are already rotten in their “core selves” so methylphenidate can't make them any worse). There is even a hint that methylphenidate can instil moral feelings in them. This claim is attributed to the young people themselves. For example:

> Mark views his tablets as having the ability to change him, to ‘make you good’ – but only ‘partly’ good, or ‘not all bad’. [44]

ADHD drugging does indeed curtail a certain set of “disruptive” (DSM-IV) or “naughty” (ADHD parents) behaviours. Indeed the “condition” is defined in terms of “disruptive” behaviours. All that Singh's empirical study has shown then is that ADHD drugging does indeed cause less “naughty” behaviours. Mark and his parents probably both agree about this. Once again though this is a circular argument. ADHD drugging has been shown to reduce the behaviours which constitute the ADHD diagnosis. But nothing objective outside of this discourse of psychiatry has been established. None of this has *anything* to do with a philosophical discourse about the “authentic self” nor with the value statement that even if young people have deficiencies there is an ethical or value case to value them as they are and not try to change them. Furthermore; how the effect of reduced “symptoms”, “disruptive behaviours” or “naughtiness” is achieved, whether through a positive and helpful drug effect or through a painful, discomforting and unpleasant negative drug-effect is a matter of indifference to positivists, psychiatrists and quite possibly to at least some ADHD parents. Leaving aside the unmade philosophical arguments we can accept that it may be that through improving short-term attentiveness methylphenidate can help young people reflect on their behaviour in a more focussed way. Thus, perhaps, they really do start to develop more appropriate thinking about being “naughty”. (That is, thinking which is in line with Dr Singh's moral

system). But even if this is accepted it does not follow that “medicating” is vindicated. Behavioural interventions (or indeed other types of intervention or response) may well achieve the same result without any of the side-effects that young people report from “medication”.

With quite amazing insouciance Dr Singh appears to believe that her small ADHD study has re-written the philosophical discourse about authenticity. But this depends on several arbitrary jumps in her argument by which a philosophical concern with an “authentic self” transmogrifies into statements young people aged 8-12 make about themselves in terms of a morality of obedience to parental demands. Statements which, Dr Singh concedes, may just reflect what their parents have told them.

The flavour of Dr Singh's study can perhaps be given by this question which the young people were asked:

> This doll has to take the same Ritalin tablets that you do. So when she takes them how does she do it?
>
> Can you tell me where the tablets go once she’s swallowed them?
>
> Is that where her problem is? Can you point to where the problem is that the tablets are helping? [44]

Can Dr Singh point to *where* the tablets are helping?

Absent from Dr Singh's paper is any discussion about what these young people need. It appears to be mostly a projection of a certain archaic and heavy-handed morality onto a group of “ADHD children” through leading interviews, who are not, in effect, consulted at all. As such it is characteristic of a general moral tone in the ADHD discourse.

A careful observation of the ADHD narrative shows that claims about actual benefits to the young people of taking the drugs are few and far between. In 2009 The European Medicines Agency produced a detailed report of the adverse events associated with methylphenidate. The benefits were explained in terms of a claim about “reducing the symptoms of hyperactivity” and “improving the quality of life”. [47] The claim about “improving the quality of life” is folksy, intangible, and untestable. On its information page about “ADHD” the NHS makes this claim:

> These medications are not a permanent cure for ADHD, but they can help someone with the condition concentrate better, be less impulsive, feel calmer, and learn and

> practise new skills. [48]

The cheery claim about young people “learning and practising new skills” by taking amphetamines, stimulants and even a failed anti-depressant is part of the folk narrative about ADHD. This doesn't really happen. As the authors of the NICE Guide concede:

> There is little evidence that stimulant medication alters the relatively poor long-term outcome for many of those with ADHD. [49]

The drugs can “reduce symptoms”. The “symptoms” are behaviours which are “disruptive and inappropriate for developmental level”. But the young person does not necessarily benefit from this reduction in his “disruptive” behaviours. The ADHD narrative rarely tries to even offer an explanation for how the young person himself benefits from being drugged. Dr Singh's paper is an interesting and rare piece of ADHD drugging promotion in that it steps outside the usual “clinical” framework wherein ADHD drugging is justified on the grounds that it “reduces symptoms”. Singh almost appears to be attempting to credit methylphenidate with the power to “make children more moral”. This is a somewhat surprising emergence into the open of the moral theme in ADHD drugging. However; Singh fails to do anything other than demonstrate that methylphenidate can indeed make young people a little less “naughty”. This is the moral version of the clinical framing. But it remains limited to the small self-referential circle that establishes that methylphenidate can control and manage the “disruptive” behaviours which are ADHD.  Methylphenidate does not in fact make people ontologically better.

vii) Summary

There is no medical or biological case for ADHD drugging. There is no test that identifies any kind of biological condition in any one young person “with ADHD”. The drugs reduce behaviours that are characterised as “disruptive”. There is no guarantee as to whether this is the result of a positive drug effect or a negative drug effect. Stimulant drugs effect all people in the same way. There is no special feature of the brains of young people “with ADHD” that means that drugs which are typically considered harmful for others are wonderful for them. The proposition that there is a “paradoxical effect” whereby stimulants drugs have some especial beneficial for “children with ADHD” is an appeal to purely magical thinking.

The ADHD narrative is redolent with a kind of old-school morality about “children” suffering to make them more “moral”. There is a Victorian copy-book morality about “children” where obedience and compliance to adult commands are the chief good. We saw how one ADHD researcher highlighted that when an “ADHD child” was on methylphenidate and he misbehaved he felt appropriately “bad” but when he was not on methylphenidate he enjoyed misbehaving. (The spiteful little devil). It is this “joy” (her word) in misbehaving that she apparently wants to eliminate. Most healthy people recognize that young people are mischievous and that enjoying being naughty is a normal part of growing up. Possibly this is especially so for some young people; perhaps even especially so for those with minor limitations in high-order mental functioning. This could be explained as a compensatory measure. Nothing more than a somewhat more intense version of what motivates virtually all young people to be “naughty” sometimes. Not a “disorder”.

The non-stimulant drug Strattera (atomoxetine) is increasingly used in the UK. Strattera has less potential for “abuse”. It does not have a recreational use. The price for increasing attentiveness without the attributes that make stimulants popular as a recreational drug seems to be an increase in suicidal behaviours in some users. Taking the evidence from clinical trials as reported by NICE, together with the data on adverse events recorded by the MHRA, we can say that a significant number of young people, some aged as young as eight, will (not may) feel suicidal as a direct result of taking Strattera “for” their “ADHD”. We have shown that there is solid evidence to believe that young people in the UK have in fact already committed suicide as a result of being on atomoxetine.

In reality the drugging agenda is a “moral” one. The drugs do curtail “disruptive” behaviour and “naughtiness”. However, the drugs are extremely harmful and there is no medical benefit to taking them.

One estimate for the total value of the global market for ADHD drugs is USD 2.4 billion. [20] ADHD drugging appears to be a collaboration between 19$^{th}$ century morality and 21$^{st}$ century greed.

## 4) NICE

i) Introduction

The National Institute for Health and Clinical Excellence is the government body in the UK which provides recommendations to healthcare professionals in the National Health Service with respect to the best treatments which can be provided for patients at a realistic cost. Part of this work involves sponsoring the production of Clinical Guidelines for treating certain conditions. NICE has established four centres to develop these Guidelines. The Nice Guideline on ADHD [1] was produced by the National Collaborating Centre for Mental Health (NCCMH). NICE pays the NCCMH to produce Clinical Guidelines. The NCCMH itself is a coalition between The Royal College of Psychiatrists and The British Psychological Society. It appears that the NCCMH itself has no legal existence. It is simply headed paper. The address of the NCCMH situates it in the offices of The Royal College of Psychiatrists. The Royal College of Psychiatrists and The British Psychological Society are the professional membership bodies for psychiatrists and clinical psychologists respectively in the United Kingdom. NICE commissioned the NCCMH to produce a Guideline on the "treatment and management of ADHD". The document was published in 2008. We review it here.

The two main recommendations produced by this group, as they relate to young people and "ADHD" are:

> Drug treatment is not indicated as the first-line treatment for all school-age children and young people with ADHD. It should be reserved for those with severe symptoms and impairment or for those with moderate levels of impairment who have refused non-drug interventions, or whose symptoms have not responded sufficiently to parent-training/education programmes or group psychological treatment. [2]

and

> In school-age children and young people with severe ADHD, drug treatment should be offered as the first-line treatment. Parents should also be offered a group-based parent-training/education programme. [3]

These recommendations allow for psychological services and psychiatric prescribing in equal measure. The two professional membership groups who produced the Clinical Guideline are thus well-served by its recommendations.

There is no official “clinical” category of “severe ADHD”. Thus, in effect, the recommendations allow individual practitioners to prescribe at will as “severe ADHD” is a matter of judgement. Furthermore; the recommendation which permits drugging for those “who have refused non-drug interventions” will in effect allow parents to choose drugging rather than behavioural treatment. This is because a behavioural intervention often involves a parent-training course. Parents simply have to say that they would prefer their child to be drugged rather than attend such a course. The recommendations thus grant considerable autonomy to psychiatrists and parents. If the preferences of parents and the arbitrary decisions of psychiatrists form the basis for treatment decisions we *cannot* be in the realm of “clinical guidelines” for a clinical condition as the authors claim.

In any event these are recommendations and are not binding on psychiatrists. Psychiatrists can and will prescribe drugs “for ADHD” regardless of the official recommendations.

The production of this Guideline does not appear to have impacted the steady year on year growth in the market for ADHD drugs. The number of prescriptions issued for drugs to “treat ADHD” has been rising steadily year on year in recent years and continues to do so. (See Section 3) iv)). Despite several hundred pages of publicly funded research and discussion the final recommendation appears to be shaped in such a way as to have as little impact on practice in the UK as possible. In effect individual psychiatrists can continue to prescribe exactly as they see fit.

There is a striking lack of evidence-based argument in this report. The authors have made relatively little attempt to show how their final recommendations can be derived from the material that they reviewed. For example, as we shall see, the only evidence to support a recommendation specifically for drugging in cases of “severe ADHD” appears to be a single study which re-analysed some of the original MTA study data. This study found, by secondary data analysis, enhanced “drug benefit” for those, who it was supposed retrospectively, would have met the stricter ICD-10 “diagnostic” criteria “for ADHD”. However, the NICE Guideline authors do not appear to make a direct link between this (secondary) evidence and their final recommendation. They cannot because they could not seriously propose to base UK policy on ADHD “medication” on a single secondary evaluation of the data type study. (Which does not in any event even imply the conclusion that “medication” should necessarily be used as a first-line “treatment” even in these cases). Thus at best the national recommendation appears to *possibly* rest on a single secondary evaluation of the data study. This tactic of argument by tangential reference is thoroughly characteristic of the entire document. It enables a vague sense of an evidence chain to be built up while avoiding establishing a position which could be criticised in any way. In any event the final recommendations effectively appear out of thin air.

ii) There is no biological test for ADHD

*a) ADHD is not a biological condition*

The NICE Guideline authors accept that “ADHD” is not a biological condition:

> The diagnosis of ADHD does not imply a medical or neurological cause. [4]

Nor is there a biological test “for ADHD”:

> There is no specific biological test for ADHD... [5]

In fact there no biological test, at all. Not just no “specific” test. This is weasel words, an attempt to mislead the public. They try to sow confusion again:

> It is recognised that defining neurodevelopmental and mental health disorders is a difficult process because of the overlapping nature of syndromes, the complexity of the aetiological processes and the lack of a ‘gold standard’ such as a biological test. [6]

A biological test is not a “gold standard”. It is the basis of a medical diagnosis. Whatever “ADHD” is it is not a medical condition. It is, according to the NICE authors, a “mental health disorder”. But then we are no longer in the realm of physical facts. We are in a realm where subjective interpretations, social institutions, power, budgets, government policies, decisions of private interest groups and the availability of pharmaceutical products and so on all collide to produce the “definition” of a “disorder”. The falsehood at the base of the ADHD narrative is the attempt to disguise the fundamental difference between a label which refers to a physical reality and a label which is a social and political production.

It is for this reason, that it is a political production, that the discussion about “ADHD” is precisely one about the social and political context in which it occurs, about power and about economic factors. We will see in the following how the authors of the ADHD narrative are keen to do everything they can to prevent the discussion about ADHD taking place in this field.

The following passage is an example of how the authors of the NICE Guideline attempt to pass off their “diagnostic construct” as being on a par with actual medical diagnoses:

> Although biological tests for ADHD do not exist, the diagnosis can be reliably applied

> when data capture tools such as standardised clinical interviews used by trained individuals and operational diagnostic criteria are employed (for example, Taylor et al., 1986; Schwab-Stone et al., 1993; SchwabStone et al., 1994; Epstein et al., 2005). [6]

The mystification lies in the “diagnosis can be reliably applied”. In fact the “diagnosis” does not have an objective existence as this idea implies. There is no objective condition outside of the definition which is the arbitrary creation of psychiatry. Once again we see that ADHD is only established by a circular argument.

*b) A “diagnostic* category*”?*

If ADHD is not related to a biological diagnosis what is it? It emerges that ADHD is a “diagnostic category” of psychiatry. This is the phrase used by the NICE Guideline authors. The Guideline authors devote a section to discussing whether the “diagnostic category” is valid.

In discussing whether “ADHD is a valid diagnostic category” the authors of the NICE Guideline firstly adopt a standard argument used to explain psychiatric “diagnostic categories”. The argument is that symptoms such as inattention and hyperactivity-impulsivity can be shown to occur together and at the same time can be distinguished from other “psychiatric disorders” such as “oppositional defiant disorder” as well as from the “normal spectrum”. Once the category is established, the authors effectively run an open competition inviting people from different disciplines to establish that it is possible to make statistically significant links to the category. This creates endless “research”, and therefore publishing opportunities for academics with expensive pieces of kit at hand. (MRI scanners, PET scanners and genetics laboratories). The statistical links are taken to affirm the “validity” of the category.

The NICE authors review evidence from MRI studies which show some statistically significant differences in brain functioning between ADHD groups and groups of normals. Many MRI scan studies fail to use “un-medicated” ADHD subjects and therefore cannot discount the possibility that the effects they find are due to exposure to stimulants. [7] The NICE authors concede as much in their review of one major meta-study (Valera et al., 2007):

> It was not possible to include or exclude the role of medication in the observed changes to brain volume and structure. [8]

The NICE authors also discuss the evidence from genetic twin studies which suggests a hereditary aspect “to ADHD”. The authors concede that without reliance on the equal environments

assumption the evidence from twin-studies for a genetic link to the label is inconclusive. We discussed the equal environments assumption in Section 1) ix). It is a key assumption on which most of the evidence from twin-studies rests. It is contestable.

> This tendency to run in families supports the idea that it is a coherent syndrome, whether the reasons are genetic or environmental. [8]

The NICE Guideline authors also refer to the evidence from molecular genetic studies. These are studies of the same kind as the genome wide association study which we reviewed in Section 1). The NICE Guideline pre-dates this study. Based on the studies available to them at the time they found very slender evidence of a small statistical link between identifiable genetic factors and possession of an ADHD label:

> As with all other types of risk factor associated with ADHD, the individual genetic variants associated with the disorder are neither sufficient nor necessary to cause it, but contribute a small increase to the overall risk for ADHD. [8]

The NICE Guideline authors also cite a study (McCann *et al*., 2007) which showed an association between food additives and increased levels of “ADHD symptoms”. [8]

The above is a summary of the main points but is not exhaustive. The reader is referred to Section 5 of the NICE Guidelines for the full presentation.

The NICE Guideline authors concluded:

> The review above identified clinical, genetic, environmental and neurobiological factors associated with ADHD or correlated with levels of ADHD symptoms in the general population that were sufficient to validate the diagnostic construct of ADHD. [9]

What the NICE authors mean by this exercise in “validation” is that they have established that it is possible to find various statistical links that correlate physical factors to the category of ADHD which they have created. The statistical links in most cases are small. Only a tiny percentage of young people in an ADHD group actually possess the significant trait. It is just that the ADHD group possess it slightly more than the normal group. As we have already discussed, (Introduction iv)), most ADHD studies subtract young people with increased inattentiveness from the normal group. Thus the ADHD group is in reality being compared not with the population norm but with another abstracted group; those with better than average

attentiveness. Inevitably this will help to produce “results”. Even leaving this problematic aside all these studies show is that the category does correlate statistically to some physical reality when the *averages* between two groups are compared. Many people will belong to the labelled group and not possess a single one of these statistically discovered links to the category. Statistical correlation based on comparing group averages does not establish a clinical disorder in a single individual. And thus this method does not establish a clinical condition.

Given that behaviour does correlate to biology many categories could be established in this way as being “valid”. The key question is: for what purpose has a specific category been “defined”, that is established as an official category of psychiatry? Why “ADHD” and not a category, say, for clumsiness? The development of the ADHD story itself may provide a clue. The “discovery” that stimulant drugs can be used to improve the behaviour of disruptive “children”, or at least make them more “driven”, (See Section 3) ii)) pre-dated the official definition of ADHD as a diagnostic category of psychiatry (Section 1) iii)). A category and “diagnosis” is required in order to facilitate the dispensation of the drugs.

The NICE Guideline authors say:

> The GDG [Guideline Development Group] recognised that ADHD is a complex heterogeneous disorder with a range of different aetiologies, including environmental, genetic and non-genetic neurobiological factors. [10]

On this basis more or less any labelling of any behavioural trait could be “validated” as a “disorder”. The reader should not be confused at this point about what has been “validated”. What has been “validated” is the “diagnostic category”. It has been “validated” as a statistically meaningful way of dividing people up into groups. No condition which any one individual has has been established. No medical causal pathway has been established. No physiological or biological “cause” of “ADHD”. At the heart of the ADHD narrative though is a kind of linguistic slippage by which the statistically valid label is used *as if it were* a valid scientific diagnosis referring to a biological fact in each and every individual so “diagnosed”.

*c) A continuum?*

According to the NICE Guideline authors “ADHD” is:

> best conceptualised as the extreme of a continuous trait that is distributed throughout the population. [9]

Conceptualising behaviours as being on a continuum of the normal population is an idea from the social sciences rather than psychiatry. It is therefore probable that this view was included in the report at the insistence of The British Psychological Society. It is at least a recognition that ADHD is not a “disease”. However, only quantified data can be understood as spread out along a line (forming a “continuum”). Thus the “continuum” model implies a quantification of human behaviour. Physiological data such as blood pressure can be directly quantified. But while behaviours such as “hyperactivity” can be quantified this is a secondary operation. The behaviour is first interpreted (usually in ADHD studies where the ratings are done by an involved party such as a parent or teacher), assigned according to a scale conceived of by the researchers, and then a score is placed on this. Social science studies of this kind already then involve a prior “subjective” step, or rather several steps, which medical science studies do not. These steps creates the possibility for all kinds of “bias” to enter into the sums.

The MTA study used just such a system to produce quantifications of “symptoms” to enable mathematical operations to be performed on the “data”. The system used in the MTA study asked parents and teachers to rate the young person's behaviours (such as “often is forgetful in daily activities” and “often loses temper”) on a scale consisting of: “not at all”, “just a little”, “quite a bit” and “very much”. These questionnaires were then turned into numerical “data”. [11] This is a typical methodological operation in the social sciences. This is the basis for the thinking about a “continuum”. It is not, even remotely, empirical science.

This kind of theorising is comfortable for social scientists. It poses a political problem however for the ADHD drugging narrative. If ADHD behaviours are just the “extreme” end of a continuum (not just one end but the “extreme” end) then it becomes hard to justify drugging. Where is the line to be drawn? The psychiatrists on the NICE Clinical Guideline committee made a come-back:

> This [understanding “ADHD” as a continuum of normal behaviour] highlighted the importance of defining what amounts to a significant impairment and ensuring that impairment is fully evaluated when applying the diagnostic criteria. [9]

This notion of “impairment” is key to the drugging narrative.

*d) An impairment?*

The concept of “impairment” is essential to the drugging programme. It enables ADHD interventions to be constructed as beneficent and caring. It is to help them with their “impairment” that “children” are drugged. “Impairment” is a key element of the DSM-IV check-list:

> There must be clear evidence of significant impairment in social, school, or work functioning. (See Appendix i)).

However; this concern with “impairment” does not appear to relate to the suffering, or not, of the young people. ADHD “symptoms” are said to improve when behaviours such as “squirming in seat”, “getting up from seat when remaining in seat is expected”, and “talking excessively” are reduced. The targeted behaviours relate to management problems and adult convenience. We have seen how the original discovery of the benefits of stimulant “medication” for disruptive young people praised the “drive” that it gave the young people in school-tasks and noted the “subdued behaviour”. (Section 3) ii)). The concern is with their *performance/non-compliance* not with their suffering/health. That is, the young person may “squirm less” as a result of being drugged but they don't go on to live a more fulfilling life. A concern with impairment would be plausible if young peoples' lives were being demonstrably improved by the drugs. But they aren't. The narrative admits as much:

> There is little evidence that stimulant medication alters the relatively poor long term outcome for many of those with ADHD (Weiss & Hechtman, 1993). [12]

*e) Conclusion*

There is no biological test for ADHD. Thus ADHD is not a physical condition. ADHD is a “diagnostic category” of psychiatry. Young people who exhibit certain behaviours *may* be designated as “having ADHD”. This is a man-made label. ADHD is not an objective reality. Psychiatry, officially, concedes this. However, having admitted as much, albeit reluctantly (no “gold standard”, no “specific biological test”), psychiatry immediately reifies the label, treating it as if it refers to something objective, something which exists. Phrases such as “children with ADHD”, “having ADHD”, and “with ADHD” are used frequently in the NICE Guideline document. In fact the phrase “with ADHD” occurs no less than 1008 times. The specific phrase “children with ADHD” 366 times. Thus the myth is spawned that there is a condition ADHD, something that “children” “have”. The myth is that people who've been placed in this category, actually *have* something. In fact they have just been placed into this category because a coalition of their parents, teachers and a psychiatrist or paediatrician have determined that they want to do this and that the behaviour of the young

person meets the criteria of the “diagnostic instrument” of DSM-IV or ICD-10, labelling systems of psychiatry.

iii) NICE discusses the “controversy” around ADHD

The authors of the NICE ADHD Guideline felt compelled to discuss what they call the “controversy” around ADHD. In this section we review how they set about this by examining some parts of the text in detail. The ADHD Clinical Guideline was produced for NICE by a coalition of The Royal College of Psychiatrists and The British Psychological Society. It will become clear that the text was never designed to be critically examined. The purpose of the document was not seriously to provide a “medical-scientific” case for ADHD interventions but to provide a political fig-leaf to justify existing practice.

In the following indented excerpts are followed by commentary.

> 5.3 THE VALIDITY OF ADHD AS A DIAGNOSTIC CATEGORY
>
> The use of the diagnosis of ADHD has been the subject of considerable controversy and debate and the diagnosis itself has varied across time and place as diagnostic systems have evolved (Rhodes *et al*., 2006). Points of controversy identified by the GDG [Guideline Development Group] included both specific issues, such as the wide variation in prevalence rates reported for ADHD and the possible reasons for these differences, and the nature of the aetiological factors that increase the risk for ADHD, as well as more complex broader sociological and philosophical issues. [6]

“Diagnostic systems” potentially sounds scientific and objective. In fact what is referred to are the tick-box systems of DSM-IV and ICD-10. These two main “diagnostic systems” are attached as Appendices. The reader can perhaps judge for themselves whether or not these behaviour check-lists are properly described as “diagnostic systems”. At any rate these are systems devised by psychiatry. They do not refer to empirical reality (as say a medical check-list aid to help a doctor diagnose measles might).

A system for labelling behaviour such as “ADHD” will obviously generate varying “prevalence rates”. The “prevalence rates” will depend on a range of factors. For example, the policy of the school board in a certain area, the availability of publicly funded drugs, the policies of public health bodies, the numbers of psychiatrists per head of population and who is allowed to make the diagnosis (in the UK for example only psychiatrists and paediatricians can make the diagnosis, in

the US a broader range of medical practitioners can; thus “prevalence rates” are higher in the US). Prevalence rates will also depend on which rating system is being used. According to the NICE Guideline authors, use of DSM-IV will produce more than twice as many “diagnoses” as ICD-10. [13] Other sources give a much higher rate for DSM-IV compared to ICD-10. Singh 2008 gives a figure of 3-4 times for cases “diagnosed” with DSM-IV than ICD-10 [14]. A “clinical category” which varies by 100% - 400% depending on which rating system is being used is not an objective category subject to some variation. Indeed the NICE Guideline authors appear to admit as much:

> Such a wide range in prevalence estimates is unlikely to reflect true differences in the numbers of individuals with ADHD in various populations. Polanczyk and colleagues (2007) made a systematic review of prevalence studies and concluded that the great majority of variability derived from the methods used, such as the way symptoms were measured and the exact definitions used. [13]

In contrast to this the prevalence rates for an actual disease, measles for example, will be an objective fact, whether or not “treatment” is available and regardless of the policies of the local school and health boards.

Despite this apparent admission of obvious social factors being involved in their “diagnostic category” the Guideline authors will not be giving consideration to what they call the "social scientific paradigm".

> Some of the complex areas of controversy relate to broader sociological and philosophical issues representing two conceptual paradigms, broadly characterised as medical–scientific and social–scientific. The latter perspective casts doubts on the utility and legitimacy of ADHD as a diagnostic category by emphasis on: the problematic nature of the meaning of ADHD, the social determinants of the behaviours that come to be labelled as ADHD, and the spectrum of human behaviour that results in indistinct boundaries of many medical diagnostic categories. While it is important to acknowledge the validity of the social scientific paradigm and its body of literature, in the context of the development of practical clinical guidelines, it is not possible to offer alternative processes for clinical assessment or treatment. It is accepted that the research literature reflects the dominant medical scientific paradigm and hence the nature of the evidence base. [15]

There is a further irony to this elimination of the “social scientific paradigm” from their investigation.

Any study which relies for its base data on quantifications of questionnaire data, rather than objective physiological measures (e.g. heart-rate) is a work of social science. As it turns out the majority of the pharmacological studies, including the MTA study, reviewed by the NICE Guideline authors rely in whole or in part on just such data. Typically they rely on parent and teacher completed questionnaires and surveys to measure the changes (as they perceive them) in behaviours, brought about by drugs. Some of these studies include an element of physical science, for example, measuring heart rate, but the main concern is with perceived behaviour changes which are recorded by parents and/or teachers. These studies use the methods of social science and psychology. There are in the domain of social science. It is thus more than ironic that the authors of the NICE Guideline dismiss the "social-science paradigm" as being outside their remit. The case for drugging depends entirely on material generated using the social sciences.

When the NICE Guideline authors say "It is accepted that the research literature reflects the dominant medical scientific paradigm and hence the nature of the evidence base" how is that to be understood? As we shall see (sub-section vii) below) the clinical trials which are used to produce the symptom reduction claims on which ADHD drugging is based, (not perhaps all of the "research literature", but a key part of it), are typically funded by pharmaceutical companies interested in marketing their products. The financial resources of these companies enable them to dominate the "research literature". The argument as presented amounts to saying that fiscal power can determine truth. One would possibly have expected slightly more from a report which claims to be "medical" and "scientific"?

> The gender ratio for children attending ADHD clinics is typically higher than in community surveys, raising the possibility of under-recognition in females.
>
> Studies of clinic-based diagnoses suggest that ADHD is nine times more common in males, although this gender imbalance is inflated to some extent by referral bias; epidemiological studies suggest that prevalence is only two to four times greater in males. [10]

The authors acknowledge the enormous gender difference in rates of ADHD "diagnosis". Surprisingly given how significant this is they do not discuss the matter in detail and it forms no part of their recommendations beyond advice to carry on as normal: "The evidence does not allow for a clear scientific consensus, so the practice is still to apply diagnostic criteria regardless of gender". [10] Since it is hard to argue that a label is "objective" in any sense when it is patently more linked to boys than girls, the gender disparity in ADHD labelling is an awkward one from the point of view of the ADHD narrative. The blasé suggestion by the NICE Guideline authors is that this awkward anomaly in the narrative might be fixed by simply "diagnosing" more girls. However; this suggestion

bypasses any discussion of why, currently, boys are far more likely to be “diagnosed” than girls. Such a discussion would move into the terrain of understanding ADHD as the product of social policy and practice; precisely the discussion the NICE authors are keen to dismiss as being out of “clinical” scope. But if the evidence shows that rates of ADHD diagnosis are influenced by social factors then avoiding that discussion is unscientific.

The NICE authors say that up to nine times as many boys as girls are “diagnosed” “with ADHD” in clinics. In general population studies two to four times as many boys as girls meet the diagnostic criteria “for ADHD”. The difference is explained as “referral bias”. The NICE authors do not appear to be unduly concerned that their “diagnostic category” can be so readily misused as to have a “referral bias” of up to 450%. Beyond a bland and unconvincing call for more research “to clarify the nature and prognostic implications of different presentations in boys and girls” [16] they do not apparently feel any need to explain it.

The ADHD narrative as it is cannot explain the gender differences in ADHD because to do so would be to admit that behaviour management and the expectations of adults play the central role in determining who gets the “diagnosis”. The authors of the NICE Guideline avoid or postpone the question. With their usual single-mindedness of purpose as concerns anything which contradicts the drugging narrative the question is dismissed.

> Can the diagnosis be made from rating scales only?
>
> Rather, it is important to complete a full evaluation including diagnostic clinical interviews with parents, children (especially older children and adolescents) and other corroborative evidence such as school reports. The use of rating scale data alone will generate both false positive and negative diagnoses and would remove the critical element of an in-depth appraisal of the entire clinical picture including onset, cause, associated developmental and mental health exacerbating and causal factors. [17]

The “diagnosis” is subjective. There is no objective test. It *cannot* therefore produce “false positives and negative diagnoses” because without an objective measure there can be no way of ascertaining which “diagnoses” were false positives. This is a fraudulent attempt to portray the “diagnosis” as objective.

We can note that what is described here is not a process of medical diagnosis. It is a judicial procedure. The “child” is interviewed. Witnesses are called. “Corroborative evidence” can be used. The “child” can be condemned on the “evidence” of their parents and teachers.

> Can the diagnosis be made on the basis of observation alone?
>
> Direct observation of an individual with ADHD, particularly older adolescents and adults, for short periods of time during assessment sessions may not demonstrate any obvious features of the condition. This should not exclude the diagnosis where there is a clear account of inattentive, impulsive or hyperactive behaviours in usual situations.
>
> The reason is that some people with ADHD can regulate their behaviour for short periods of time and because ADHD behaviours are typically reduced in situations where a person is engaged in an important task. The GDG advises that diagnosis should only be made on the basis of a full assessment.
>
> Summary statement: The diagnosis of ADHD should not be made on the basis of observational data alone. [18]

This is somewhat shocking. If a young person “with ADHD” doesn't confirm this by showing the “symptoms” is it not that they don't, in fact, “have ADHD”, but they are hiding it. This is eerily reminiscent of medieval methods for ascertaining who is a witch. Whichever way you go you will be condemned. Why a young person might want to “regulate their behaviour” in the psychiatrist’s office is not explained. Interestingly there is an implicit admission here that young people with ADHD behaviours can concentrate on tasks when they strongly motivated. A pity that this observation is not followed up into a serious “clinical” approach to “treating” young people “with ADHD”. The link between motivation and attention is evident in the material they review but is ignored by the NICE Guideline authors. This is because it does not support the drugging narrative. We saw (Section 3) iii)) how, in reporting a study into methylphenidate and dopamine the NICE authors simply cut out the main result of the paper they were citing. That was that methylphenidate only increased dopamine when associated with a challenging task.

The diagnosis may be made where there is a “clear account of inattentive, impulsive or hyperactive behaviours in usual situations”. Who will provide this “clear account”? Parents and teachers. One can see how this recommendation will be of benefit to psychiatrists in private practice. To issue an ADHD “diagnosis” (whose main purpose is to provide a legal authorisation to drug a young person) they do not even have to witness the DSM-IV behaviours themselves. They can rely on reports by the child's parents. Parents can have their troublesome children drugged at will. “Is your child impulsive?” “Oh yes.” “OK. Here is a prescription”.

> The GDG wished to evaluate evidence for the validity of the diagnostic category of ADHD and formulate a position statement on the use of the diagnosis. It is recognised that defining neurodevelopmental and mental health disorders is a difficult process because of the overlapping nature of syndromes, the complexity of the aetiological processes and the lack of a ‘gold standard’ such as a biological test. In this regard ADHD is similar to other common psychiatric disorders that rely on the identification of abnormal mental phenomena. Although biological tests for ADHD do not exist, the diagnosis can be reliably applied when data capture tools such as standardised clinical interviews used by trained individuals and operational diagnostic criteria are employed (for example, Taylor et al., 1986; Schwab-Stone et al., 1993; SchwabStone et al., 1994; Epstein et al., 2005). [6]

As we have already discussed, a "biological test" is not a "gold standard" of defining a disease. Outside of psychiatry it is a basic condition for establishing a disease.

In fact “complexity of the aetiological processes” is more disinformation. ADHD does not have “aetiological processes” as such. As the Guideline authors admit themselves:

> The diagnosis of ADHD does not imply a medical or neurological cause. [4]

But unless there is a biological condition there can be no aetiology. This is an example of how the narrative operates at two levels. On the one hand psychiatry officially admits that that the label does not refer to a biological “cause”. But soon afterwards they can't help themselves discussing just such processes. This is because they are *believers* in the biological model.

The MRI, PET scan and genome wide association studies, such as the genome wide association study reviewed above in Section 1), establish statistical correlations between possession of the label and physical factors. They do not, generally, establish evidence of aetiological (causal) processes. For example the genome study which we reviewed in Section 1) shows a correlation between possession of a certain genetic trait and an ADHD label in some cases but cannot establish that the genetic factor has a causal relationship to the ADHD behaviours.

To think purely in terms of (supposed) “aetiological processes” is to use a reductionist model. This reductionist model thinks about humans as biological units. It eschews thinking about people in human terms, beings with agency who live and work in a social context. This reductionist model de facto justifies pharmaceutical interventions. Because, if behaviour is understood as the product of biology it makes sense to modify it pharmacologically. On the other hand if behaviour is understood in terms of human agency and social context it makes sense to think about modifying it

with human interventions such as changes to the social context, discussion, negotiation. This is still the case even if we accept that biological factors often play a role in determining the range of behaviour which a person might be capable of. We can see why The Royal College of Psychiatrists wishes to promote a (fictitious) narrative about “complex aetiological processes”. It promotes the pharmaceutical interventions which they administer. However; this approach objectifies young people.

The claim that ADHD is like other mental health disorders which rely on identification of “abnormal mental phenomena” is simply made up. ADHD is defined in terms of behaviour not mental processes. The ADHD discourse is characterised by a more or less total lack of interest in the mental processes of young people. The NICE authors are trying to find a new place to hide their behaviour management category, this time in amongst the “mental illnesses”. But mental illnesses, whatever they are, do often relate to real human suffering. And the treatment might, at least sometimes, reduce that suffering. Whereas with ADHD the concern is with behaviours.

That the standard clinical interviews are reliable tells us nothing. Any set of behaviours could be grouped and given a label which could reliably be “diagnosed” on a repeat basis by different clinicians following the same standardised test. This establishes that clinicians are capable of consistently applying a behaviour check-list. It does not “validate” the “psychiatric category” other than in an internal self-justifying sense for the psychiatric profession. ADHD can only support itself by a circular argument.

> In keeping with most common mental health disorders, the distinction between the clinical condition and normal variation in the general population is difficult to define on the basis of symptom counts alone. This is because there is continuity in the level of ADHD symptoms between those with an impairing mental health disorder and those who are unimpaired. The distinction between ADHD and normal variation in the general population requires the association of a characteristic cluster of symptoms and significant levels of impairment. This is comparable to normal variation for medical traits such as hypertension and type II diabetes, as well as psychological problems such as anxiety or depression. Controversial issues surround changing thresholds applied to the definition of illness as new knowledge and treatments are developed (Kessler et al.,2003) and the extent to which it is acknowledged that clinical thresholds are socially and culturally influenced and determine how an individual’s level of functioning within the ‘normal cultural environment’ is assessed (Sonuga-Barke, 1998). In considering these issues, a key question is to define the level of ADHD symptoms and associated impairments required to trigger the use of this guideline. [6]

Here the authors try to give creditability to their behaviour category ADHD by equating it with the physiological and medical condition Type II Diabetes. Type II Diabetes is a medical condition arising from insulin deficiency. Both, the authors argue, exist on a “continuum”. Both become significant when “impairment” arises. However; the variation on a spectrum of Type II Diabetes is not the same kind of variation on a spectrum as that for ADHD. With ADHD a “symptom count” is produced by a process involving questionnaires and surveys, usually completed by interested parties such as parents or teachers. This “data” is then collated into a form in which it can be statically analysed. The “spectrum” has been constructed artificially. It depends on the kind of questions asked and on the reliability of the reporters. In the case of Type II Diabetes the measurement is direct, empirical and unambiguous. It is a physiological measure of glucose tolerance. The analogy between ADHD and Type II Diabetes therefore fails at this point. ADHD does not exist on a continuum in the same sense that Type II Diabetes does.

The analogy is also unsustainable in terms of “impairment”. In the case of Type II Diabetes the “impairment” may mean heart disease, kidney failure or other life threatening conditions. There is a significant reduction in life expectancy. The “impairment” in ADHD appears to mean that a young person functions less well as a school-child and/or as a well-behaved child at home. Their “impairment” is not something they suffer from. It is not a matter of medical urgency. In the ADHD narrative “impairment” means something different than the medical sense it has in terms of Type II Diabetes. The analogy breaks down on this basis too. The attempt to claim an equivalence between the medical condition Type II diabetes and the psychiatric label ADHD is another example of the way that psychiatry attempts to bolster its ADHD narrative by borrowings from other discourses.

> The GDG wishes to emphasise that psychiatric nosology is a dynamic and developing field and changes are to be expected as more data are accrued over time. [6]

Psychiatric nosology means the systems of classification developed by psychiatry. This is The Royal College of Psychiatrists giving themselves unlimited scope to adapt the definition to meet any changes in the “data” that may come up. The “data” they are referring to is the kind of material generated in studies such as the MTA study. Questionnaires designed by ADHD advocates, completed by parents (who at least believe in “medication” sufficiently to allow their child to be drugged and take part in a trial) quantified into “data” and then mined to produce pro drugging results. The “diagnosis” can shift with the times. The constant is not an illness (most illnesses are probably quite fixed over time) but psychiatry. Medical science responds to illnesses. Psychiatry imposes them.

What is this “diagnostic category” of psychiatry really about? One psychiatric contributor to the

NICE ADHD Guideline candidly admits:

> ADHD symptoms were designed for primary school children and an adult with ADHD is a child with ADHD who has gown up but continues to have problems. [19]

The symptoms were “designed” for “primary school children”. This clarifies both that the “symptoms” are in fact “designed” psychiatric definitions (not medical symptoms that anyone actually has) and that the “disorder” is something to do with non-functioning in school situations.

Psychiatric systems of classification are creations of the psychiatric profession. This exercise where the authors of the NICE ADHD Guideline “discuss” the “validity of ADHD as a diagnostic category” never escapes from being an insular discussion amongst psychiatrists. A solipsist discussion of psychiatry. ADHD will be what they decide it to be.

iv) How NICE uses the MTA study

The NICE Guideline authors consider “psychological” interventions versus pharmaceutical ones (drugging). [20]. This is the key, in fact only, question remaining within the limited frame of reference which promotes uncritically the notion of “ADHD” as a “clinical” problem and which compares only the standard “treatments”. This then was the decisive question for the The National Collaborating Centre for Mental Health, a partnership between The Royal College of Psychiatrists and The British Psychological Society. We knew that they were not, for example, really going to decide that “ADHD” is not a “valid diagnostic category”. As we have seen their final recommendation supports both drugging and behavioural interventions in equal measure. When faced with the opportunity to favour one over the other they have produced a Guideline which neatly recommends both in equal measure.

*a) Behavioural or “pharmaceutical” interventions?*

The NICE Guideline authors approach the question as to whether pharmaceutical or psychological interventions are more “effective” “for ADHD” by reviewing the existing studies. “Effectiveness” is defined in terms of the symptom reduction scoring system which is typically used in ADHD-drugging studies. The claim that “symptoms are reduced” is presented as a self-evidently worthwhile result. As we have discussed (Section 2) iii)) by importing the word “symptoms” into the narrative promoters of ADHD drugging seek to avoid a discussion of the value or ethics of what they are doing. They try to disguise their behaviour modification programme by appropriating a medical language of “symptoms” and “treatment”.

The NICE authors found 6 possible studies which met their inclusion criteria which tested drugging against behavioural training programmes. [21] The MTA study which we have reviewed in Section 2) above was the largest scale one. The MTA study had a total of 579 “participants” with approximately 120 - 140 subjects in each of the four “treatment” groups. The average number of participants in the other 5 studies was 53, with total numbers per study ranging from 30 to 86. The total across all these studies was 266. [22] The MTA study thus involved more participants than all the other reviewed studies put together. It is clear then that in answering the question as to whether pharmaceutical interventions are more “effective” for “treating ADHD” than psychological ones the MTA study is the primary source available to the NICE Guideline authors. The full assessment by NICE of the 6 studies is interesting:

> For both teacher and parent ratings of core ADHD symptoms and conduct problems at the end of treatment, stimulant medication delivers better outcomes than psychological interventions, with effect sizes in the small to moderate range. [23]

According to the MTA study authors there was no statistically significant benefit to medication” over the behavioural intervention when measured by teachers for the ADHD “symptom” of hyperactivity: Table 5 in the MTA study. [24] (As we have discussed in Section 2) ii) the MTA study text reports that it was teachers not parents who noted a reduction in “symptoms” but Table 5 shows that it was parents and not teachers who produced this score). According to the MTA study “medication” did not deliver better results (symptom scoring system) than a behavioural intervention for any domain other than ADHD symptoms including oppositional-defiance. “Medication management and behavioural treatment did not differ significantly on any other outcomes “. [24] The above claims by the NICE authors therefore cannot be derived from the MTA study. The NICE authors do not attempt to explain how their conclusion about pharmacological versus behavioural interventions differs substantially from the results of the study which (by number of participants) provided 2/3's of their data.

In another section the NICE Guideline authors summarise the MTA study more enthusiastically:

> At 14 months (MTA Co-operative Group, 1999a) the outcome strongly favoured careful medication (whether or not in combination with behaviour therapy);... [25]

It was only for the “ADHD symptom” of inattention that both parents and teachers concurred on a “medication advantage”. On the other ADHD “symptom” of hyperactivity, only parents produced better symptom reduction scores. This was the case whether or not in combination with behaviour therapy. When comparing the “medication” programme against the behavioural intervention there was no “benefit” to the “medication” programme on *any* of the non-ADHD domains: “Medication

management and behavioural treatment did not differ significantly on any other outcomes". [24] Furthermore; as we have discussed (Section 2) ii)) the favourable findings for "medication" over behavioural treatment were not supported by the neutral classroom observers on an ADHD "symptom" measure nor by the young people themselves when self-scoring for anxiety/depression. It is therefore significantly misleading to summarise these results as "strongly favouring". It would appear that this passage in the NICE Guideline was authored by a team even more in favour of stimulant "medication" than the team who concluded that the MTA study showed "effect sizes in the small to moderate range".

In the above passage the NICE Guideline authors refer to "careful medication". This is misleading. The "medication" programme on the MTA study was not "careful" (or not "careful" at all from one perspective). It was a "carefully-crafted" regime. It was put together specially for the study. It used a dosage significantly higher than in typical outpatient settings and a thorough titration regime designed to optimise the "benefits" (higher symptom reduction scores) in each individual case. The MTA study compared this highly unusual and optimised "medication" regime against a behavioural programme unique to the MTA study. In the real world doses of methylphenidate will be lower and there will be a huge range of different behaviour programmes. Scientifically then no conclusions can be drawn from the MTA study about whether "medication" is "better" in general than a behavioural programme in a typical outpatient setting. This is one of the many fatal flaws in the MTA study. (Discussed in Section 2) iv)). The NICE ADHD Guideline authors are apparently aware of the particular nature of the "medication" regime in the MTA study and are at pains to disguise it with another linguistic manipulation. "Carefully-crafted" becomes "careful".

Generalising findings from a specific research context to a more general context needs to be undertaken carefully. If the conditions in the wider context do not match those of the research environment such an extrapolation cannot, scientifically, be made. The "medication" regime in the MTA study was nothing like that which is typically encountered in an out-patient clinic. The data from the MTA study itself shows this. The MTA subjects on the "medication" only programme who were receiving methylphenidate were being dosed with 37.7 mg daily (at treatment end-point). For those on the community care programme, that is the normal outpatient circumstance, the average daily dose was 22.6 mg (at treatment end-point). [24] This is very significantly less. The MTA study does not on any basis provide a basis for making the claim that "medication" is superior to a behavioural treatment in general terms. The NICE Guideline authors appear to believe that general conclusions can be drawn from the MTA study. However; scientifically this cannot be done.

Furthermore, the NICE authors reported that the MTA study showed that when the MTA behavioural intervention was compared against community care the results (symptom scoring system) were about equal:

> A further tentative inference from the data gathered at the end of treatment is that the

> intensive MTA behavioural intervention may have had similar effects to routine medication because the majority (66%) of the community care group received medication for ADHD and the behavioural intervention group did not differ significantly from the community care group for end of treatment outcomes. It must, however, be noted that the absence of a statistical difference between the groups does not prove that there is no difference between the effects of the behavioural intervention and continued community care. [26]

This can be confirmed in Table 5 in the MTA study. [24] This seems clear. When the comparison was made between the MTA behavioural intervention and normal out-patient care (which often involves “medication”) the scores were about the same. Normal out-patient care is what young people will (by definition) receive. These results have far more clinical relevance than comparisons using the atypical and enhanced medication regime of the MTA study. On this basis if we were to accept the methodology of the MTA study it seems clear that it could be said to provide strong, even compelling, evidence for replacing typical “medication” based out-patient regimes completely with behavioural interventions. In terms of “symptom reduction” the effects would be the same and there would be less side-effects. Naturally, neither the MTA authors nor the NICE authors follow-up this obvious conclusion from the “clinical evidence”.

It becomes clear from the above that the NICE Guideline authors are manipulating the already manipulated results of the MTA study. In place of science and “evidence-based medicine” we have manipulation piled upon manipulation. The aim is to produce a narrative favourable to drugging.

However, the NICE Guideline authors are careful and somewhat adroit in their use of the MTA study. Rather than using it to claim that “medication” is better than psychological interventions they settle in the end for the claim that the MTA study (and others) shows that “medication” and psychological interventions are “about equal”:

> While there is no evidence that psychological interventions are favoured over stimulant medication for any outcome, or at any time point, it is also the case that medication does not appear to be strongly favoured over psychological interventions. [27]

This paves the way for a recommendation which says that both “medication” and psychological interventions are suitable, and the decision should, in effect, be left to parents and individual psychiatrists.

Atomoxetine is being used increasingly in England. However, all of the 6 studies which the NICE Guideline authors used to compare pharmacological interventions with behavioural ones used

methylphenidate as the main drug, not atomoxetine. [22] In fact then atomoxetine has not been tested in comparison with behavioural interventions at all. But the recommendations are produced which will apply equally to atomoxetine and methylphenidate. Essentially, methylphenidate has become “medication” and the complexity that other drugs are used has been ignored. Again; this is not remotely “evidence-based”. (The NICE authors might argue that atomoxetine has been shown to have comparable symptom-scoring reduction counts to methylphenidate [28] and so by extension comparisons between methylphenidate and behavioural interventions will apply to atomoxetine v. behavioural interventions. That kind of argument would be a tenuous position for a case allegedly based on “clinical evidence”).

Rather than critically discuss the MTA study the NICE Guideline authors make careful use of it to support their predetermined outcome.

*b) Is there any “clinical evidence” for the recommendation that drug treatment should be used as a “first-line treatment” for those “with” “severe ADHD”?*

> Drug treatment is not indicated as the first-line treatment for all school-age children and young people with ADHD. It should be reserved for those with severe symptoms and impairment or for those with moderate levels of impairment who have refused non-drug interventions, or whose symptoms have not responded sufficiently to parent-training/education programmes or group psychological treatment. [29]

Do the NICE Guideline authors offer any “clinical evidence” to support the recommendation that “medication” is particularly suitable for those “with severe ADHD”? There is one reference in the Guideline which may be behind this recommendation. [29] This is to Santosh *et al*. 2005. [30] Santosh *et al*. 2005 was a secondary evaluation study based on the MTA study. The NICE authors report that Santosh *et al*. 2005 showed that “medication” achieved a “greater decrease in symptoms” for “the more severe subgroup meeting criteria for hyper-kinetic disorder” [29] The reference to this paper does not directly appear in the review of “clinical evidence” (Section 11.3.4) where pharmacological versus behavioural “interventions” are compared nor in the “Clinical evidence summary” (Section 11.3.5) of this review. It appears in a subsequent and follow-up section entitled “Further considerations with respect to the treatment of ADHD – additional evidence from the MTA study”. (Section 11.4.1). In their overall concluding section on comparing psychological versus pharmacological interventions the NICE Guideline authors do not refer again to Santosh 2005. (Section 11.6). This means that they make no link between the recommendation (Section 11.7) that “medication” is a suitable “first-line treatment for those with severe symptoms” and the Santosh 2005 study. However it seems apparent that the purpose of citing Santosh *et al*. 2005 is to support this recommendation. No other evidence is offered to support this conclusion in

Section 11.3.4 where the “clinical evidence” is reviewed. All this means that this recommendation is not “evidence-based”. The tactic seems obvious. The reference to Santosh 2005 is floated out because it seems to support the preferred recommendation, but it is not claimed to do so directly, so it cannot be argued against. This is not how science proceeds.

What if anything does Santosh *et al*. 2005 actually show? Santosh *et al*. 2005 was a secondary evaluation study based on the MTA study. These kinds of study re-sift data from existing studies. By cutting up the data in different ways it is often possible to engineer results which are more supportive of a given position. Santosh *et al*. 2005 reviewed the MTA study data and based on the original questionnaire material they identified a sub-group who would (they assert) have met the criteria for the more “stringent” ICD-10 ADHD diagnosis. (The test used in the main MTA study was DSM-IV). Santosh claims that:

> The superiority of medication to behavioural treatment was greater for children with HD. [30]

(HD stands for hyperkenetic disorder, or the ICD-10 ADHD diagnosis). This means that on the MTA scoring system a greater score difference on some measures between the “medication” group and the behavioural intervention group can be demonstrated for this (abstracted) ICD-10 group than for the DSM-IV group as a whole.

The Santosh *et al*. study inherits all the methodological flaws of the MTA study on which it is based and which we have discussed extensively in Section 2) above. Furthermore, it should be noted that the Santosh *et al*. study used, according to the NICE Guideline authors, the data from the end of the treatment time of the original MTA study (14 months) [28]. Santosh *et al*. is thus subject to the same problems as for the MTA study as a whole posed by the MTA follow-up study Jensen *et al*. 2007. Jensen *et al*. showed that the “medication” superiority of the MTA study did not hold up in the longer run. This NICE Guideline authors concede this:

> However, the benefits of stimulant medication over psychological therapies for core ADHD symptoms and conduct problems in general do not appear to be sustained at later follow-up assessments (3–6 months, 7–12 months and 13–24 months after the end of treatment). [23]

The NICE Guideline authors do not admit however that it is likely that the fall-off of the “medication advantage” will also apply to the Santosh 2005 study. This point was made by the well-known critic of ADHD excesses, psychiatrist Sami Timimi. Writing for the Guardian, Professor Timimi comments

that the recommendation for “medication” for those with “severe symptoms” is not evidence-based:

> Yet Nice concludes that medication should be used as a first-line treatment in "severe" ADHD, citing only one reference in support of this.
>
> Even this reference is fundamentally flawed, as it refers to data from a large trial comparing medication and behavioural treatments, which concluded that the more severe subgroup showed a larger decrease in symptoms with medication than with therapy after 14 months in treatment.
>
> However, after 36 months, this research project found no superiority in outcome for medication over behaviour therapy, even in those with more severe symptoms. At the same time, it found that children exposed to medication for the longest periods were now significantly lighter and shorter than their peers. [31]

In addition it should be noted that the MTA study used methylphenidate. The claims in Santosh *et al*. 2005 therefore relate to methylphenidate. Santosh *et al*. 2005 cannot therefore be used to make a claim about other drugs. In England “drug treatment” is just as likely to mean atomoxetine as methylphenidate. Somehow this “research” (secondary evalaution study) appears to have been expanded to draw conclusions about “drug treatment” in general. This is not even vaguely scientific. It has no connection with “evidence-based medicine” whatsoever.

The NICE authors include amongst their final recommendations the recommendation that “healthcare professionals should advise parents or carers and the child or young person about the benefits and superiority of drug treatment in this group” [32]. A single secondary evaluation of the data type study does not establish “clear benefits”. Furthermore; even if taken as being a determining study Santosh *et al*. 2005 does not justify “medication” in this group (“severe ADHD”) either. They will still suffer from all the side-effects of the drugs. And their “symptoms” would still be reduced by a behaviour programme.

Interestingly, one of the co-authors of the Santosh *et al*. *2005* secondary evaluation study was Professor Eric Taylor, lead author of the NICE ADHD Guideline and a leading ADHD-drugging advocate in the UK. Possibly his involvement with this secondary evaluation of the data study may account for its prominence in the NICE ADHD Guideline?

The above disccssion relates to the sections of the NICE document where the authors compare pharamceutical verus behavioural interventions, (Section 11). However the same claim about a supposed “superior benefit” for those “with severe ADHD” is also made in the section which deals with pharmaceutical interventions, (Section 10):

> If a group parent-training/education programme is not effective for a child or young person with severe ADHD, and if drug treatment has not been accepted, discuss the possibility of drug treatment again or other psychological treatment (group CBT and/or social skills training), highlighting the clear benefits and superiority of drug treatment in children or young people with severe ADHD. (Section 10.18.3.4) [33]

Reviewing the sections in the NICE Guidleline where the authors summarise the "clinical evidence" for atomoxetine (Section 10.8.5) and for methylphendiate (Section 10.6.7) there is no mention of evidence for a superior effect in those with more "severe symptoms". One would expect in a work of "evidence-based" medicine that a recommendation would follow from research. Since this claim about "the clear benefits and superiority of drug treatment in children or young people with severe ADHD" has not been evidenced in this section of the document (at least in the summaries for the two main drugs), which deals with evidence from drug trials, it appears likely that it has been imported from the material relating to comparing pharmaceutical and behavioural interventions (Section 11.4.1), and even from there in a section presented outside of the main research pathway entitled "Further considerations ... additional evidence from the MTA study" which reviewed a secondary evaluation of the data study of one trial. It would appear that this claim about "clear benefits and superiority of drug treatment in children or young people with severe ADHD" has been added to the pharmacological section as a subsequent edit. It would appear that someone has been cooking the books.

The MTA study was not designed to assess the absolute efficacy of a pharmaceutical treatment. There was no untreated control group. Therefore Santosh *et al*. 2005 cannot be used to make any claims whatsoever relating to pharmacological treatment as compared with no treatment. This makes the presence of this claim, in the section assessing the absolute "efficacy" of pharmaceutical treatments, all the more extraordinary. Yet this claim about "the clear benefits and superiority of drug treatment in children or young people with severe ADHD" appears central to the final recommendations of the NICE report.

*c) Discounting the harms*

Having decided that:

> While there is no evidence that psychological interventions are favoured over stimulant medication for any outcome, or at any time point, it is also the case that medication does not appear to be strongly favoured over psychological interventions. [27]

the NICE authors conclude:

> Accordingly the decision about whether to use a psychological intervention or stimulant medication for ADHD appears to be more balanced. In this context the choice of first-line intervention might be influenced by factors other than effectiveness, including possible adverse effects of medication and preferences of the child and/or parent. [34]

Behavioural interventions do not cause insomnia, growth problems, stomach ache, nervousness etc. as well as risks of serious cardiac problems (rare) and, in the case of atomoxetine, the real potential for suicidal thinking, unsuccessful suicide attempts and successful suicide attempts. The Guideline authors suggest that when deciding whether to impose a behavioural treatment or a drug treatment the “possible adverse effects” of “medication” might “influence” the decision. [34]. The “adverse effects” are not “possible”. The MTA study reported that 63% of young people in their programme experienced “side-effects”. They are likely. (Remember too that since it was parents doing the reporting in the MTA study this figure of 63% is likely to be an underestimate). The NICE authors admit in an offhand way: “methylphenidate can cause insomnia.” [35] One of the case studies they include describes how a young person taking methylphenidate had to be prescribed a sleeping medication (clonidine) to counter-act the effects of methylphenidate. [36] This type of practice, of creating a stack of drugs to manage the effects of the original treatment, is common in ADHD drugging. According to Peter Breggin, clonidin is a typical next step in this escalation. [37] The Guideline authors are also aware that one of the MTA follow-up studies showed growth loss associated with drugging. [38] It is dishonest of the NICE Guideline authors to talk about the “possible adverse effects of medication” when they know that adverse effects are routine for young people taking methylphenidate. In some cases the very same drugs used to be marketed specifically and directly for the effect which is now described as a “possible side-effect”. Weight loss, for example, was not described as a “possible” effect when pharmaceutical companies were promoting amphetamines as a weightloss treatment. All this must be known to the NICE authors.

The Guideline authors are suffering, like the MTA study authors, from amnesia as relates to the medically correct way to determine the usefulness of a drug. The UK's Medical and Healthcare Products Regulatory Agency (MHRA) explains the correct approach:

> Do the advantages outweigh the dis-advantages of taking the medicine? [39]

When recommending drugging over behavioural interventions the NICE Guideline authors do not weigh up the manifest and serious harms of “medication” against its claimed “benefits” (symptom scoring system). They just mention the harms as one of various factors which could be considered.

The recommendations support the use of “medication” for those who have “refused” a behavioural intervention. As we have commented above (sub-section i)) this will in effect allow parents to make

the decision about drugging or a behavioural intervention. When the Guideline authors refer to the “preferences of the child and/or parent” we can be quite sure that the preferences of parents will be prioritised. (This effect is likely to be even greater in private practice).

In general it seems parents of young people who are prescribed drugs “for ADHD” are not swayed by concerns about side-effects (until, tragically, sometimes when it is too late). For example; we saw (Section 3) vi)) how Singh 2007 in her study of young people being drugged with methylphenidate found that:

> Children reported that when on medication they had little or no appetite, had trouble sleeping, had headaches or tummy aches. Children reported having no such troubles when not taking medication. [40]

Surveys of “children” taking drugs “for ADHD” routinely turn up the fact that the drugs make them feel unwell, cause sleeping problems etc. It seems to be the case that many parents tolerate these “side-effects”. By implication the ones in Singh 2007 did. It would behove people who wish to be taken seriously as medical practitioners not to pander to parents' willingness to accept these “side-effects” for their children.

Based on the figures provided by the NICE Guideline Authors there is no argument in terms of cost to the Health Service which would justify “medication” over behavioural treatment. The authors cost a years drugging with methylphenidate at £1,038.00. This is substantially more than a typical behavioural intervention utilising group therapy which costs £303.00. [41]. In fact for the cost of a year's drugging with methylphenidate a more extensive one to one behavioural intervention could be provided. Apparently this costs £894.00. [41]  We present this information to clarify that the NICE Guideline does not recommend drugging because it is “cheaper” than behavioural interventions. We are not endorsing an approach which carefully weighs up financial costs but forgets to take the human costs of side-effects into the calculus.

On their own account a behavioural intervention can be nearly as or equally “effective” as a “medication” programme. Behavioural interventions do not cause insomnia, tics, stomach ache, growth loss and possible suicide attempts. The conclusion should be obvious.

*d) Conclusion*

The NICE Guideline authors make very substantial use of the MTA study. It forms the major part of their “clinical evidence review” for comparing drugging against behavioural interventions “for ADHD”. They make no criticisms of the study despite the fact that it is badly flawed even in the

usual terms of this kind of study. Some of the obvious flaws include; lack of neutral observers on most measures, lack of a non-treatment group, entirely selective use of its own results in the conclusions, potential for medication-bias in the selection of subject participants, use of a non-typical “medication” programme and one particular behavioural programme to form conclusions about “medication” and behavioural programmes in general. The construction of the study was mostly in the hands of well-known drugging advocates, and the questionnaire used to generate “data” about “ADHD symptoms” was part created by and is credited to one these well-known drugging advocates. The NICE authors do not offer any criticisms of the methodology of the MTA study. However the NICE Guideline authors devote more than a full page of detailed critique to the findings which have awkwardly emerged from the MTA study that the drug advantage (symptom scoring) does not hold up in the long run. (See the following sub-section). This selectivity in critical stance betrays the fact that the NICE Guideline authors are looking for “evidence” to bolster their favoured position rather than conducting a genuine effort to base clinical recommendations on research. Their final recommendations are hardly “evidence-based”. The thread connecting the “clinical evidence” to the final recommendations is hard to find.

The ADHD Guideline project cost the public purse around £500,000.00 which was paid to National Collaborating Centre for Mental Health, a partnership between The Royal College of Psychiatrists and The British Psychological Society. [42] It would appear that these organisations have been paid £500,000.00 of the public's money to produce recommendations which favour their professional interests in equal measure.

v) How NICE manages the awkward finding from the MTA study of no long-term advantage for “medication”

*a) Introduction*

The original MTA study lasted for 14 months. Some of the researchers from the MTA study continued to work with the same subjects. They continued to monitor “symptom” scores for the original treatment groups. They reported:

> In contrast to the significant advantage of MedMgt+Comb over Beh+CC for ADHD symptoms at 14 and 24 months, treatment groups did not differ significantly on any measure at 36 months. [43]

(“CC” in the above refers to the “Community Care” treatment group in the original MTA study). This study was carried out by ardent ADHD promoters, Peter Jensen [44] and James Swanson [45], and others, and was a specific follow-up to the major NIMH sponsored study which was supposed

to have demonstrated the “superiority” of “medication” once and for all. The result was that the “medication advantage” which was found in the original MTA study was not maintained in the longer term. This was a disaster for the ADHD drugging lobby. We have discussed this result in the context of our review of the MTA study. (See Section 2) vii)).

The findings of the MTA follow-up study were reported in the media. A BBC Panorama programme gave significant coverage to the views of Dr William Pelham. Dr Pelham was one of the original MTA researchers. He was also one of the authors of the main follow-up study. He said:

> I think that we exaggerated the beneficial impact of medication in the first study. We had thought that children medicated longer would have better outcomes. That didn't happen to be the case. There's no indication that medication's better than nothing in the long run. [46]

In an attempt to rescue the drugging position Dr James Swanson and Dr Peter Jensen, and others, produced the inevitable secondary evaluation of the data type study. [47]

Two separate groups of authors working on the NICE Guideline felt a need to respond to the findings of Jensen *at al*. 2007. The first response was by the group who were working on the pharmacological section of the NICE guideline. The second was by the group who were working on the section of the Guideline that compared pharmacological against behavioural “treatments”.

*b) How did the authors of the pharmacological section of the NICE Guideline attempt to deal with the MTA follow-up study?*

Section 10.6 of the NICE Guideline discusses methylphenidate. The authors of this section felt a need to respond to a perceived challenge that the MTA follow-up study had shown that medication had no benefit in the long-run. They say:

> These results have been widely interpreted as showing no long-term impact of medication or behaviour therapy. While this is one possible reading, it is not demonstrated by the study and other explanations need to be considered. [48]

This statement though is confused. It is not a possible reading that the MTA follow-up study showed “no long-term impact of medication”. There was no untreated control group in the MTA study so no inferences can be drawn either way about “medication” as compared to no “treatment”. It is true that the Panorama programme may have over-simplified their presentation of this study

and mistakenly given the impression that it showed “no long-term impact”. (See Section 5) iii)). But the NICE authors appear to be confusing what the study can be used to claim by nature of its construction, and a mistake in reporting. The reason for this is probably that they want to make the same mistake - but in the other direction.

The main finding of Jensen *et al*. 2007 was that they were unable to confirm that at 36 months “medication” was “superior” to a behavioural intervention:

> In contrast to the significant advantage of MedMgt+Comb over Beh+CC for ADHD symptoms at 14 and 24 months, treatment groups did not differ significantly on any measure at 36 months. [43]

The authors of Section 10.6 present several arguments in an attempt to counter the damaging findings of the MTA follow-up study results.

Their first argument is:

> First, the end of randomisation entails that patients and families select which intervention is best for them. [48]

This argument definitely has some merit and is indeed the only serious argument which can be raised to counter the failure to support the “superiority” of “medication” over a behavioural intervention at 36 months. Jensen *et al*. report:

> Indeed, once the delivery of randomly assigned treatments by MTA staff stopped at 14 months, the MTA became an observational study in which subjects and families were free to choose their own treatment but in the context of availability and barriers to care existing in their communities. [43]

However, Jensen *et al*. also report:

> Even though medication use patterns changed significantly from 14 to 36 months, with more cases assigned to the Comb and MedMgt conditions stopping medication and more cases from the Beh starting medication, the initial differences in medication use (especially Beh) and the two MTA medicated groups (Comb and MedMgt) were not completely eliminated. That is, at 36 months, 71% of Comb and MedMgt participants were using medication at high levels compared to 62% and 45% of CC and Beh

> participants, respectively. Groups also continued to differ in average medication doses as well. Yet these medication use variables during the year from 24 to 36 months did not reveal any advantage on 36-month outcomes and instead showed a tendency toward disadvantage. [43]

That is; while there was some convergence of treatment it was by no means complete. At 36 months there were *still* significant differences in “medication” use (71% to 45%). Yet “symptom” scores were equalised. In fact they were not just equalised. For the period 24 months to 36 months there was a *disadvantage* for “medication”. The argument about randomisation ending has some merit but it does not fully explain the convergence of scores.

The second argument is:

> Second, the end of intensive therapy could mean that any effects additional to those of usual good treatment wane when the intensity is reduced: therefore all treatment arms become similar to community treatment. [48]

This appears to be an admission that without the extra-high doses of methylphenidate used in the original MTA study the “medication advantage” wears off. But if this is the case and if this is their argument then it follows that the actual claims of the MTA study are clinically irrelevant, resting as they do on the higher than usual doses of methylphenidate used in the study. The authors of Section 10.6 appear to have shot themselves in the foot. In attempting to limit the damaged caused by the MTA follow-up study they undermine the relevance of the original MTA study.

The third argument put forwards by the authors of the pharmacological treatment section of the NICE Guide to limit the damage caused by the finding that the “medication advantage” is not sustained over time is as follows:

> Third, the absence of an untreated control group makes it impossible to know whether the treatments were better than not intervening. Outcome scores at 36 months remained considerably better than the levels before treatment; the conclusion may be that all treatments work rather than that none do. [48]

The MTA study was set up to *compare* (symptom scoring method) the main “treatments” “for” “ADHD”. The MTA study authors were explicit that there was no control group. The aim of the MTA study was to compare chiefly “medication” versus a behavioural intervention. Continued into the longer term the study has shown that “medication” is not better (symptom scoring system) than

behavioural interventions. An awkward and “unexpected” (Jensen *et al.)* finding. Drug advocates then try to re-use the MTA study as a standard drug study. They attempt to argue that it shows reduced “symptoms” for “medication” over time and therefore justifies “medication”, (as well as behavioural treatments). This way out of the difficulty was first proposed in the MTA follow-up paper itself:

> Thus, an important clinical message to be taken from our findings is that all of the treatment groups showed significant improvement over time. [43]

However, there, at least, there is an admission of the limitation of this:

> Of course, without an untreated control group, no firm conclusions about the possibility of more positive ADHD outcomes can be drawn with confidence. [44]

The NICE authors admit that there is no control group but proceed in the very next sentence to make the impossible claim that “the conclusion may be that all treatments work rather than that none do”. Without a control group such a claim cannot be made or even, from the point of view of the theory behind normal clinical trials, contemplated.

The “fourth” damage-limitation argument in this section again follows the lead offered by the pro-drugging researchers on the MTA study. In fact this apparent fourth argument is just a more detailed presentation of their third argument. Inevitably the attempt was made to recover the position with a secondary evaluation of the data type of study. Swanson *et al*. 2007 [47] was called “Secondary evaluations of MTA 36-month outcomes: propensity score and growth mixture model analyses”. This study did not compare treatment groups. It simply attempted to show that “symptoms” were reduced over time for those on “medication”. It used a statistical method known as growth mixture model analysis which is imposed on the data in order produce classes from which results can be claimed. The NICE authors call this method of statistical manipulation the “best fit”. Since the MTA study did not include a control group all this is a statistical exercise which does not meet the standards required for a randomised clinical trial. It cannot in effect be used to support a claim for “evidence-based” medicine. Swanson *et al*. 2007 divided the groups into those with high medication use and low medication use. High means they were being drugged more than 50% of the time and low means they were being drugged less than 50% of the time. Swanson *et al*. report:

> GMM [growth mixture model] analyses identified heterogeneity of trajectories over time and three classes: class 1 (34% of the MTA sample) with initial small improvement followed by gradual improvement that produced significant medication effects; class 2

> (52%) with initial large improvement maintained for 3 years and overrepresentation of cases treated with the MTA Medication Algorithm; and class 3 (14%) with initial large improvement followed by deterioration. [47]

and

> By the 36-month assessment, the effect of medication status for class 1 was statistically significant ($T = 3.92$, $p < .001$), but the effect of medication status for classes 2 and 3 (initially significant) were no longer statistically significant (class 2: $T = 0.14$, $p < .888$; class 3: $T = 0.48$, $p < .632$). [47]

The authors of Section 10.6 of the NICE Guide report this thus:

> One of the classes (34% of the sample) showed gradual improvement with continuing benefit from medication over the entire 3 years. The second class (52% of the sample) had an initial large response, maintained for 3 years; in another 14% a large initial response was followed by deterioration. In the second group who responded well, there was a significant preponderance of children who had been assigned to the intense MTA medication algorithm in the first 14 months, whether or not they continued medication. [48]

The results reported by NICE are clinically meaningless. There was no comparison with an untreated control group thus no conclusions can be drawn about the long-term effects of drug “treatment” as opposed to no “treatment” - (on the symptom scoring system). But even if we accept these results the conclusion is hardly in favour of “medication”. As the second citation from Swanson *et al*. above makes clear, for classes 2 and 3 at 36 months being on “high” or “low” “medication” made no difference. Classes 2 and 3 constituted 66% of the total sample. For a clear majority of the sample there was no benefit (symptom reduction system) on being on “high” “medication” to “low” (including no) “medication” at 36 months. Furthermore; for Class 3, that is 14% of the sample at the 36 month point the “initial beneficial effect” had “completely dissipated”. [47] For 14% of subjects after three years of “medication” their “symptoms” were the same as they were on day one. If they can be used to claim anything these statistical results show that for the majority of subjects (drugged eight year olds) the “benefits” of “medication” do indeed tend to wear off over time. This should be no surprise at all. It is well-known that people develop a tolerance to drugs of this kind. The NICE authors try to use these statistical results to show that “medication” does have a “beneficial” effect over time but ignore the equally obvious inference; that the effect does indeed wear off over time.

This is not the only aspect of Swanson *et al*. 2007 about which the authors of Section 10.6 of the NICE Guideline have been rather selective. The authors of the original MTA follow-up study attempted to explain away the "unexpected" finding of convergence between the different treatment groups with a hypothesis:

> We hypothesized that this unexpected pattern may be due to a tendency of those who are doing well either to stay off medication or to discontinue it and those doing poorly either to start taking it or to continue it. [43]

The suggestion is that the "medication advantage" was reduced because young people with very bad "symptoms" started use and those with few symptoms stopped. And this would explain the loss of the "medication advantage" rather than the wearing off of the positive "drug-effect". Jensen *et al*. determined to explore this possibility:

> This hypothesis is further tested and discussed in the companion paper in this issue by Swanson *et al*. (2007). [43]

Swanson *et al*. 2007 did indeed investigate the self-selection hypothesis. They divided the subjects into 5 groups with increasing degrees of medication adoption over time. They found that "symptom" scores at 36 months were similar across all groups. If the self-selection hypothesis was correct they would have expected to have found higher symptom scores in those who started on "medication" later. They did not find this. They reported tersely:

> We failed to confirm the self-selection hypothesis. [47]

The authors of the NICE Guideline Section 10.6 fail to mention that this hypothesis had been proposed and not established. Not did they report the conclusion reached by Swanson *et al. 2007:*

> This finding is difficult to explain. In general, it suggests that beyond the 24-month assessment point in the MTA protocol, the overall effect of medication treatment was no longer beneficial for the reduction of ADHD symptoms, although this interpretation must be tempered by the observation of a beneficial effect of medication in one subgroup (i.e., the 34% of children in latent class 1). *This overall finding suggests the possibility of waning benefit for continued medication beyond 2 years for a large number of children with ADHD*. [47] (Emphasis added).

In fact the NICE authors summarise:

> It would therefore not be correct to regard behaviour therapy or stimulant medication as short-term treatments only. [48]

Again; we find a one-sided use of (already loaded) papers. In this case a finding by two ADHD stalwarts about “the possibility of waning benefit for continued medication beyond 2 years for a large number of children with ADHD” simply vanishes and the public is told that “It would therefore not be correct to regard behaviour therapy or stimulant medication as short-term treatments only”.

The irony is that however hard they try to produce a case for drugging out of their material the opposite case keeps emerging awkwardly from the results. This then has to be suppressed.

The NICE authors report that the MTA follow-up studies showed that growth was reduced at the 2 year point with no further reduction at the 3 year point. They refer to “conference reports” that claim that there had been catch-up at 8 years. A conference report is not a peer-reviewed clinical trial study. It is not consistent with a claim for “evidence-based” medicine to rely on “conference reports”. The reported facts are, as Dr Pelham indicated, growth-loss within the 36 month period of the MTA follow-up study. (Even if there is a “growth rebound” the question remains as to whether it is healthy to cause young people to grow in drug conditioned fits and starts).

*c) How did the authors of the treatment comparison section of the NICE Guideline attempt to deal with the MTA follow-up study?*

Section 11.4 of the NICE ADHD Guideline reviews the MTA study from the perspective of comparing “medication” treatment and behavioural interventions. The authors of this section of the NICE report also struggled with the awkward finding of the MTA follow-up study that the “medication advantage” finding of the original MTA study was not sustained in the longer run, that is at 36 months.

The MTA study is the single largest study to compare a “medication” “treatment” versus a behavioural intervention. In practice it is used extensively to promote and justify the “mixed treatment model”. This is the model which promotes both drugging and behavioural interventions in combination. The “unexpected” unravelling of the MTA study from within its own centre was a true disaster for ADHD drugging advocates. This is why we see so much effort in the NICE ADHD Guideline document dedicated to modulating its findings. The evidence we are told is “difficult to interpret”:

> The lack of evidence for the sustained superiority of medication over psychological

> interventions for ADHD is, however, difficult to interpret. [49]

This sudden adoption of a critical stance is surprising. Nothing which can be used to justify drugging is ever described as “difficult to interpret”.
The authors of this section of the NICE Guideline point to the fact that after the end of the original MTA study, at 14 months, subjects were free to choose their own treatment. The argument is that the loss of the “medication advantage” is the result of the original treatment groups diverging. This is the same argument presented by the authors of Section 10.6. Again, however, the lack of sustained evidence for a “medication advantage” cannot be entirely explained away by saying that those in the behavioural group started taking “medication” while those in the original “medication” group stopped. This did happen to some extent but the convergence in “treatments” was not complete; whereas the symptom scoring convergence was.

The NICE authors also reflect the other possible explanations offered by Jensen *et al*. 2007:

> Jensen and colleagues (2007) suggest that factors that may contribute to the convergence of outcomes for the four MTA study intervention groups at longer-term follow-up compared with outcomes at the end of treatment include: a decrease in ADHD symptoms related to age independent of treatment; changes in the intensity of medication use; and different degrees of starting and stopping medication in the different treatment allocation groups that occurred after the end of the MTA interventions. [50]

We have already seen how Jensen *et al*. 2007 hypothesized that one explanation for the falling away of the “medication advantage” was that subjects with especially bad symptoms were more likely to start or continue with “medication” whereas those “doing well” were more likely to stop. This may be what the NICE Guideline authors mean by “changes in the intensity of medication use”. The argument is that this will have skewed the results against medication. This hypothesis was tested in Swason *et al*. 2007. They conceded that they failed to confirm it: “We failed to confirm the self-selection hypothesis”. [47] The authors of the pharmacological versus behavioural treatments section of the NICE Guideline, like the authors of the pharmacological section, do not mention this failure to confirm one of the proposed attempts to explain away the loss of the “medication advantage” over time.

Jensen *et al*. do mention the possibility that one factor which might explain the loss of the medication advantage is the loss of treatment intensity for the medicated group. This is likely. After 14 months families were free to choose their own treatments. Both the assignment to organised treatment groups and the intensive treatments of the original MTA study were ended at 14 months.

This will have included the intensive “medication” regime of the MTA study. But, the implication of this argument is that “medication” is only “superior” to behavioural treatment at the higher than usual doses in the original MTA study. If that is the case the MTA study (even the original MTA study) cannot be used to recommend “medication” over a behavioural treatment in current, ordinary, clinical situations.
Jensen *et al*. 2007 do mention the possibility that the MTA study subjects experienced a reduction in “symptoms” over time due to age. The implication appears to be that this effected all the original treatment groups substantially and caused a levelling out of “symptoms” which obscured the “medication advantage”. This is possible, but does not especially rescue the case for the “superiority” of “medication” over a behavioural intervention.

*d) Conclusion*

As they attempt to stumble out of the dilemmas “unexpectedly” posed by the MTA follow-up study the NICE authors contradict themselves:

> These findings are, however, based on the comparison with baseline data for each group, not on a comparison with an untreated control group, and hence it is not possible to conclude that any of the MTA interventions have long-term beneficial effects over no treatment. (Authors of Section 11.4) [50]

and

> It would therefore not be correct to regard behaviour therapy or stimulant medication as short-term treatments only. (Authors of Section 10.6) [48]

The latter statement depends on citing the follow-up statistical paper to Jensen *et al*. 2007, Swanson *et al*. 2007. One party (more correctly) says that the MTA follow-up study cannot be used to make claims for the long-term beneficial effects of “medication”. The other party references a secondary evaluation of the data study to make just such a claim. The authors of Section 10.6 of the NICE Guide make use of Swanson *et al. 2007* to make a claim about “medication benefit” over time. However they omitt to mention that a) for the majority of the sample being on high rather than low “medication” at 36 months made no difference and b) that study authors admitted that their findings pointed to the “possibility of waning benefit for continued medication beyond 2 years for a large number of children with ADHD”. Nor are they frank about the fact that Swanson *et al. 2007* was a secondary evaluation of the data type study. Like the clinical data on which it was based there was no control group. Thus in terms of standard clinical trial standards it can say nothing about any possible long-term “benefits” of “medication”.

One unalienable fact from the MTA follow-up study, Jensen *et* al, 2007, is that one group was still more highly medicated than the other but symptom scoring still converged. This at least calls into question claims for the "medication advantage" based on the original MTA study.

We can see two responses to this outcome. On the one hand one of the MTA researchers, Dr William Pelham, broke ranks and told the press:

> I think that we exaggerated the beneficial impact of medication in the first study. We had thought that children medicated longer would have better outcomes. That didn't happen to be the case. [46]

On the other hand some of the ADHD drugging advocates on the study turned to the inevitable secondary evaluation of the data study to try to sure up the claims about "medication" reducing "ADHD symptoms" over time, regardless of any comparisons.

In their treatment of this paper and its findings the authors of the NICE Guideline (it would appear two separate sets of authors) follow the lead given by those involved in the MTA study who attempt to rescue the case for drugging. Their treatment of the material is demonstrably selective.

vi) Ignoring the side-effects

The authors of the NICE ADHD Guideline admit but discount the side-effects of the drugs used to "treat" "ADHD".

*a) Methylphenidate*

The NICE authors reviewed the "clinical trial evidence" for pharmaceutical interventions for ADHD. (In fact they reviewed 49 trials). With reference to methylphenidate they admit:

> The common adverse effects of methylphenidate include decreased appetite, sleep disturbance, headaches, stomach aches, drowsiness, irritability, tearfulness, mildly increased blood pressure and pulse (Wolraich et al., 2007). Rare but more severe adverse events can include psychotic symptoms and sensitivity reactions requiring discontinuation of the medication. [51]

The "common adverse effects" are indeed common, even normal for those on methylphenidate. Since methylphenidate is a stimulant it is no surprise that it keeps people awake at night, makes

them edgy and reduces appetite. These are the effects of stimulants on the human nervous system. It is glib to try to push these away into a box labelled “adverse effects”. They are routine and normal for young people on methylphenidate. “Requiring discontinuation of medication”. This too is glib. The reality is that at least some young people who experience psychotic symptoms will suffer in silence, not wanting to or not feeling able to tell their parents.

The NICE authors also take a glib approach to the problem of growth retardation associated with the long-term use of methylphenidate:

> While there remains some conflicting evidence regarding weight and growth in children receiving methylphenidate (Bereket *et al*., 2005; Poulton, 2006), a significant decrease in appetite can lead to a decrease in expected growth during the active period of drug treatment (MTA Co-operative Group, 2004b; Swanson et al., 2007). Suppression of growth and height may be dose related (Barkley, 1990b). It is unclear whether final adult height is affected (Poulton, 2006). [51]

As we have discussed (Section 3) v)) the known growth-retardation effect of long term use of stimulants may not be simply a result of appetite suppression. There is some work to suggest that methylphenidate disrupts the normal cycle of growth hormone in the body itself. The one paper cited above by the NICE Guideline authors, Bereket *et al*., 2005, found only a small possible link between methylphenidate and hormone disruption. [52] However; Breggin cites 3 separate studies which support this view. [53]. The words “hormone disruption” do not appear in the NICE text. The NICE Guideline authors manage to bury this damaging aspect of the discussion, about whether methylphenidate effects growth through acting on hormones, in a tangential reference to “some conflicting evidence”. This way they cannot be accused of ignoring a contentious medical matter but manage to avoid allowing the dangerous question of hormone disruption to appear directly in their text. It is glib to suggest that “it is unclear whether final adult height is affected”. Even if they do “bounce-back” causing young people to grow in stop-start bursts cannot be healthy. Elsewhere in the NICE ADHD Guide the authors offer this reassurance:

> Growth can be affected, at least in the short term, so height and weight are monitored regularly and plotted on growth charts. [54]

In fact, though, this statement about monitoring and plotting the results on growth charts is phantasy. (Like the glib claim that “medication” is always discontinued when it causes psychotic symptoms [51]). As one might expect it is the case that growth monitoring is not always done. It may even be the norm that it is not done. One educational psychologist interviewed by the Daily Mail said with reference to the monitoring of the growth of young people diagnosed ADHD on

drugs:

> This rule is being breached all over the country. One group of psychiatrists told me point-blank that they do not have the staff to do this. If they haven't the resources to do the thing safely, should they be doing it at all? [55]

Methylphenidate has been implicated in a small number of deaths. In 2004 a US Food and Drug Administration (FDA) report reviewing adverse events indicated 12 sudden paediatric deaths between 1999 and 2003 in the US in which amphetamines were "considered suspect". In 6 of these cases cardiac risk factors were reported. There were 7 cases of paediatric sudden death for methylphenidate. Six of these cases appear to be connected to cardiac events. [56] While the FDA concluded that for methylphenidate the reported numbers of deaths and serious adverse cardiac events did not reach a level high enough to warrant specific regulation they also stated that the reports should lead to a calculation of risk in making prescription decisions. They advised:

> The rare occurrence of sudden death during stimulant therapy of ADHD is an issue that warrants close monitoring and should be considered in the assessment of benefit versus risk during therapeutic decision making for individual patients. [56]

Novartis echoes the FDA advice about avoiding prescribing methylphenidate to young people with cardiac problems:

> Although some serious heart problems alone carry an increased risk of sudden death, stimulant products generally should not be used in children or adolescents with known serious structural cardiac abnormalities, cardiomyopathy, serious heart rhythm abnormalities, or other serious cardiac problems that may place them at increased vulnerability to the sympathomimetic effects of a stimulant drug. [57]

It is clear then there is a risk and that physicians should consider this matter when making prescribing decisions. The NICE Guideline authors however play this down:

> In 2006 the US FDA conducted a review on reports of sudden death in patients treated with ADHD medications using data from the AERS [Adverse Event Reporting System]. The review identified 14 paediatric and four adult sudden death cases reported with methylphenidate between January 1992 and February 2005. The review reported that none of them appears solely or directly related to methylphenidate. Six of the 14 paediatric sudden deaths occurred in children with structural cardiovascular

> abnormalities that likely preceded the use of methylphenidate.
>
> The review concluded that the rate of sudden death with methylphenidate and atomoxetine was below background rates available. However, no definitive conclusions can be drawn from the analyses of AERS cases because of the inherent limitations of the AERS and uncertainty regarding information on drug utilisation and background incidence of sudden death. Further studies were being conducted by the FDA (2008a) at the time this guideline was being prepared (January, 2008). [58]

The figures cited by NICE do not correspond precisely with those in the FDA report we have cited. [56] The NICE data refers to a slightly longer reporting period (1992 - 2005). The findings though are essentially the same. In a small number of cases adverse event reporting data links methylphenidate with cardiac events and sudden death. In some but not all of the cases the young person who died had a prior history of cardiac abnormalities. A small risk of death is acceptable in a life-saving drug; but is it a risk worth taking in order to make young people “squirm” less in class? The NICE authors appear to believe that it is.

The NICE authors summarised the “evidence” for methylphenidate thus:

> In school-age children, there is evidence that methylphenidate when compared with placebo or waitlist control produced a medium to large effect in reducing children’s ADHD symptoms and conduct problems.
>
> Methylphenidate (high dose) is more likely than placebo to cause the following side effects: insomnia, anorexia, increased irritability, moodiness, thirst, itching, diarrhoea, palpitations, stuttering, negativism, reddened eyes, incoherent speech and decrease in bodyweight.
>
> The long-term studies of methylphenidate indicate an increased risk of side effects, increase in systolic blood pressure and heart rate problems. Given the lack of background rates, the association between the use of methylphenidate and sudden death is not clear.
>
> Methylphenidate is effective in reducing ADHD core symptoms and conduct problems in children with ADHD. There is evidence suggesting that methylphenidate may increase side effects. [61]

In fact methylphenidate *routinely* causes insomnia, nervousness and stomach-aches. Insomnia and appetite loss at least are not even “side-effects”. They are the direct results of taking stimulant drugs, which used to be marketed (and to some extent still are) for these purposes. The NICE authors appear to be trying to bury these facts (from their own evidence-base) by linking them to “high-dose” of methylphenidate. However; the “side-effects” they list are typically associated with methylphenidate at normal clinical doses. Dr Peter Breggin reviewed 8 studies and found considerable “side-effects”. He reported that of these studies “most of the doses were in the low to average clinical range”. [60] (See also Section 3) v)). The sentence “There is evidence suggesting that methylphenidate may increase side effects” is a cynical under-reporting of the evidence relating to “side-effects” and stimulant drugs.

*c) Atomoxetine*

Currently the second main drug used to on “ADHD children” in the UK is atomoxetine. Use of atomoxetine (Strattera) is growing rapidly in England. Between 2004 and 2011 it has seen growth of more than 600% in the number of prescriptions issued. Atomoxetine is linked to suicidal thinking, suicide attempts and actual suicides. The NICE authors report:

> In double-blind clinical trials, suicide related behaviours occurred at a frequency of 0.44% in atomoxetine-treated patients (6 out of 1,357 patients treated, one case of attempted suicide and five of suicidal ideation). [61]

Numbers of young people on Strattera are not available. The NHS does not keep records for prescriptions per patient, just overall levels of prescriptions. According to our estimated figure based on the number of prescriptions issued as many as 56,500 young people in England may be on atomoxetine. [62] If our estimate of 56,500 is correct we can extrapolate directly, based on clinical trial “evidence” reported by NICE to a likelihood of 41 attempted suicides related to atomoxetine in 2013 in England and about 250 cases of suicidal ideation. We can note that suicide has the specificity as an adverse event that it is irreversible.

In Section 3) v) we reported that we obtained data from the MHRA relating to adverse events for Strattera. In the period 2004 to 2012 there were 106 cases of suicidal ideation and 12 reported suicide attempts. [63] The vast majority of these were amongst young people. Two of the suicide attempts succeeded. Two may have done; the data is not available. One of the survivors has a brain injury. The status is not known. The Adverse Drug Reaction reporting scheme run by the MHRA is a voluntary scheme. The actual figures are therefore likely to be higher. Not just because some are not reported but because many young people will suffer suicidal thoughts and feelings in silence.

Atomoxetine appears to be directly linked to suicidal behaviour and suicides in young people. This was *predicted* by the clinical trials reported by the NICE authors and *has come to pass* as indicated by the data from the MHRA. It seems to be characteristic of a modern "health" bureaucracy that the clinical trials took place and the figures have been dutifully recorded but no action has been taken.

It is true that young people do commit suicide without drugs. However we should note that the reports to the MHRA scheme are those where "there is a suspicion that it [the medicine] could have been responsible". [63] Anti-depressants, and atomoxetine was originally researched as an anti-depressant [64], are powerful drugs to be giving to 7 to 12 year olds. (The MHRA data relates to all ages with the majority of reports relating to under 18s. The young people on the drug company sponsored clinical trials reported by NICE were aged 7-12 [61]). Strattera has an FDA "black box" warning relating to suicide. This means that the manufacturer, Eli Lilly, must publish a warning prominently on the packaging. This obligatory warning includes the text: "In some children and teens, Strattera increases the risk of suicidal thoughts or actions." This all seems a very high risk to be running so that children can be made to squirm less in class.

The NICE Guideline authors also state in connection with atomoxetine:

> Very rarely, liver toxicity, manifested by elevated hepatic enzymes and bilirubin with jaundice, has been reported. [61]

Indeed, the US FDA has issued a warning about Strattera:

> Postmarketing reports indicate that Strattera can cause severe liver injury. Although no evidence of liver injury was detected in clinical trials of about 6000 patients, there have been rare cases of clinically significant liver injury that were considered probably or possibly related to Strattera use in postmarketing experience… [65]

It also appears to be the case that 7%of Caucasians have a particular genotype with a missing enzyme. NICE tells us that these 7% of people:

> have a several-fold higher exposure to atomoxetine when compared with patients with a functional enzyme. Poor metabolisers may be at higher risk of adverse events. For patients with a known poor metaboliser genotype, a lower starting dose and slower titration of the dose may be considered. Given that 2D6 status is rarely known for an individual patient, a low starting dose and slow titration will reduce the risk of adverse events. [66]

(This matter is also part of an FDA warning about Strattera). [67]

We can note that it is “rarely known” in advance if a young person is in this 7% of the population who will be at “higher risk of adverse events”. Is it always the case that “a low starting dose and slow titration” approach is used?

In their “conclusion from clinical evidence” the NICE Guideline authors also state:

> Common adverse effects associated with atomoxetine include abdominal pain, nausea and vomiting, decreased appetite with associated weight loss, dizziness and slight increases in heart rate and blood pressure (Wolraich et al., 2007). These effects are normally transient and may not require discontinuation of treatment. [61]

The Guideline authors admit that Strattera commonly causes stomach aches, vomiting, weight loss and “slight increases in heart rate and blood pressure”. They admit that it is linked to suicidal thinking. (Though they downplay this). They admit that there is a connection to serious liver disease. They sum up:

> Atomoxetine is effective in reducing ADHD core symptoms and clinical improvement in children with ADHD. There is no effect of atomoxetine on children’s conduct problems as rated by teachers. There is evidence suggesting that atomoxetine may increase side effects when compared with placebo and when compared with methylphenidate. [68]

Again; we have the euphemism. It “may increase side effects”. This is quite a bland way of describing a drug which by their own evidence causes suicidal thinking. The MHRA Adverse Reaction data on atomoxetine which may or may not have been consulted by the NICE Guideline authors appears to suggest that there have been suicide attempts and actual suicides linked to atomoxetine. (The MHRA data we have reported above covers the period 2004 to 2012. However; it seems likely that some adverse events related to suicidal thinking and possibly suicide attempts and actual suicides will have already been recorded when the NICE Guideline was produced in 2009).

A drug which the US FDA asserts is “probably or possibly” linked to some cases of serious liver injury. A drug which commonly makes young people have stomach aches and feel sick (though only “transiently”). A drug which will have all these effects to a much greater extent in 7% of Caucasian subjects; who cannot be determined in advance. All of this so that young people do not “talk excessively” or “blurt out answers before questions have been finished“ etc. (Appendix i)).

“Side-effects” are accepted in medicine because the drug is either saving the patient's life, or likely to produce a significant health effect (longer life, more comfort etc.). ADHD drugs do not provide any benefits of these kinds. An improvement in “conduct problems” for example is not a medical benefit for a young person. In ADHD drugging there is no medical justification for the “side-effects”. This probably explains why they are acknowledged but immediately pushed to one side.

*d) Dexamphetamine*

Dexamphetamine is the third main drug used in the UK to “treat” “ADHD”. It is a stimulant of the amphetamine family. According to the NICE Guideline authors they were not able to find a single (not one) study which could be used to determine the “efficacy” of dexamphetamine for young people:

> For children, we found no trials that met the quality criteria and therefore had no evidence on its efficacy. [69]

According to the NICE Guideline authors they were only able to find one study for the long-term “safety” of dexamphetamine, an 8 week study involving “61 hyperactive boys”. They write:

> There was only one study found that met the criteria set by the GDG: an 8-week RCT of 61 hyperactive boys (Greenberg et al.,1972). [70]

The attentive reader may refer to Appendix 17.5 [71] for a list of studies concerning pharmacological interventions. In this appendix Greenberg 1972 is listed as an excluded study. The reasons given for this study being excluded are:

> Unclear diagnostic assessment; No extractable, relevant outcomes [DEX vs. Chlorpromazine vs. Hydroxyzine vs. PLB] [71] (PLB means placebo).

It appears to be the case that this study was accepted for the purposes of safety review but not for the purposes of assessing “clinical efficacy”. In any event, what does this 1970s study that took 8 weeks tell us about the “safety” of dexamfetamine? It doesn't sound very healthy:

> Children receiving dexamfetamine complained of decreased appetite and had stomach aches more often than the control groups (hydroxyzine and placebo). Of the

> dexamfetamine group, two manifested marked regressive, dependent behaviour, and one became overtly psychotic. The intensity of all side effects subsided with a decrease in dosage. [72]

If we extrapolate from this study of “61 hyperactive boys” to the 3,500 young people we estimate to be taking dexamfetamine in England in 2013 [62] we come to 57 young people who were made “overly psychotic” in England in 2013. The NICE Guideline authors comment that “The intensity of all side effects subsided with a decrease in dosage.” They may argue that in practice the signs of psychosis would be spotted early by monitoring and the dosage reduced accordingly. Just as likely is that the young people who have been given this “medication” by their parents will suffer their night-time hallucinations in silence.

The NICE authors could not find *a single* study relating to the “clinical efficacy” in “children” of the third major drug licensed to “treat ADHD in children” in the UK, a drug for which 42,100 prescriptions were issued by the NHS in England alone in 2013. (They found a single study assessing its “efficacy” in adults). The authors of the NICE ADHD Guideline specially claimed that they undertook “the development of a patient-centred, evidence-based guideline”. They went on to claim:

> The clinical practice recommendations made by the GDG are therefore derived from the most up-to-date and robust evidence base for the clinical and cost effectiveness of the treatments and services used in the treatment and management of ADHD. [73]

By their own admission as concerns one of the three drugs licensed to “treat ADHD in children” in the UK they found no evidence at all for its “clinical efficacy” in “children”. And just one study to assess long-term harm. This study was conducted over 8 weeks. Most ADHD drugging will be for much longer than 8 weeks. For example analysing the 3 case studies presented in the NICE Guideline one young person is “medicated” from the age of 4 to 7 and ongoing, another from the age of 7 to 15 ongoing and a third, in a somewhat chaotic account, appears to be drugged with a range of drugs from the age of 13 to 25, though not necessarily continuously [74]. An eight-week study cannot begin to assess the potential harmful effects of taking a drug for several years. It is not possible to make evidence-based recommendations in the absence of evidence. The above statement cannot be true.

*e) Rehashing the fiction of the 'paradoxical effect'*

In Section 3) ii) above we discussed the myth of the paradoxical effect. This is a long-standing part

of the ADHD narrative. The claim is that there is some magical factor whereby hyperactive young people are somehow “calmed” by stimulant drugs. The “paradoxical effect” theory serves to explain why drugs which are generally deemed to be dangerous are “beneficial” for young people “with ADHD”. However, psychiatry, as we have seen, is willing to admit officially that “ADHD does not imply a medical or neurological cause.” [75] Psychiatry cannot therefore provide a theory of this kind of supposed reversed biology. If there is no theory of biology there can't be a special theory of reversed biology. Indeed even to discuss the possibility is to move into the realm of phantasy. People do not have special biological responses to drugs just because they have been placed into a “diagnostic category” of psychiatry.

The paradoxical effect myth was formed in the culture of 1930s psychiatry. As we saw, (Section 3) ii)), it has been discredited, with the final admission by psychiatry in the 1970s that in fact stimulants have the same effect on all young people. Nonetheless the fiction lingers on and while it is not deployed openly it is still sometimes implicitly used. The NICE Guideline authors adopt this strategy:

> The question of a paradoxical effect of stimulants on people with ADHD has been raised but is not well studied. For example, do stimulants have an impact on the same processes and in the same way in all people, whether they have ADHD or not? Or is there a different pattern of effects in people with high levels of ADHD symptoms compared with people with low levels? The GDG concluded that the critical question for these guidelines is whether stimulants and other non-pharmacological interventions effectively treat the impairments associated with high levels of ADHD symptoms. [76]

The “has been raised but is not well studied” is a characteristic polemical device of the NICE Guideline authors. When they have evidence which is contentious but which supports their position they tend to float it out without however specially staking a position on it. (We saw this for example in their use of some of the secondary data from the MTA study. See sub-section iv) b) above). Nor is it true. We have discussed the study by Volkow *et al*. 2007 (Section 3) ii)) which compared how the brains of ADHD labelled subjects and non ADHD labelled subjects processed methylphenidate. Volkow *et al*. showed that people with an ADHD label have (on average) a greater resistance to methylphenidate than people without a label. Greater resistance (on average across a group) is not the same as a reversed effect. Even more telling is Rapport J.L *et al.* 1980 which concluded:

> While there were some quantitative differences in drug effects on motor activity and vigilance between these different groups, stimulants appear to act similarly on normal and hyperactive children and adults. [77]

Singh 2008 referenced the above study and reported:

> In the 1970s, researchers showed that a positive response to stimulants is not limited to children with ADHD: 'normal' children show improvements in attention and focus as well. Therefore, to some degree, the medications enhance performance rather than treating the specific psychopathology. [14]

Dr Singh is not on the fringe of the ADHD narrative. She is funded by The Wellcome Trust and contributed as a special advisor to the NICE ADHD Guideline.

It is therefore not true that the "paradoxical effect of stimulants on people with ADHD has been raised but is not well studied".

At any event the "positive response" of stimulants on "normal children" is something which is known by the 2% of young people (16-24) who tried amphetamines in 2011/12 [78] as well as by the government which keeps telling them to stop. [79]. Psychiatric studies are not needed to discover that amphetamines produce a "positive response" in "normal children".

The myth of the "paradoxical effect" is theoretically untenable and has been empirically shown to be invalid. However it is kept in circulation in the ADHD narrative because it is needed to explain why drugs which are said to be harmful and dangerous for young people are suddenly "beneficial" when prescribed by a psychiatrist "for ADHD".

*f) Medicine as punishment*

The authors of the NICE Guideline admit that the drugs used to "treat" "ADHD" have potentially serious health risks. They seek to play these down. They are careful to acknowledge the main problems, while trying to cast them aside with phrases like "may increase side effects". In fact "side-effects" are normal for anyone taking stimulant drugs. 63% of subjects in the MTA study were recorded as suffering mild to serious side-effects.

ADHD drugging does not cure anyone of anything. The NICE Guideline authors admit this:

> There is little evidence that stimulant medication alters the relatively poor long-term outcome for many of those with ADHD (Weiss & Hechtman, 1993). [80]

The NICE authors produce page upon page of "evidence" about "clinical trials" which are

presented as showing “benefits” and “clinical efficacy”. But *all* these studies do is show that the drugs “reduce symptoms”. That is when you give a young person these drugs he may: “talk excessively” less, “squirm in his seat” less, be less forgetful, do his chores more, “argue with adults” less, be less “negative, defiant or disobedient to authority figures”, act “smart” less, “disturb other children” less etc. The reader is invited to review DSM-IV, Appendix i) and the SNAP-IV rating system [11] to see exactly what “clinical efficacy” actually means. None of these are medical problems.

We have discussed, for example Section 3) vi), how the ADHD narrative is permeated with a kind of rather old-fashioned moral tone. ADHD promoters are strong believers in the concept of “children” and in a strong duality of role between “children” and “adults”. The former should obey the latter and not “argue”, “act smart” etc. [11]. In Dr Singh's paper [40] it appears to be assumed that if an adult “reprimands” a young person the adult is necessarily “in the right”. The mere fact that they are reprimanding the young person assures this. We are in the world where “doing wrong” is defined purely in terms of parental expectations and demands. Being “good” is doing what your parents tell you. The possibility that sometimes some adults may make unreasonable demands on their children (and/or fail to adequately meet their needs) is not countenanced. The “benefits” of ADHD drugs are a reduction in unwanted behaviours. The drugs cause suffering. ADHD drugs thus act in the same way that old-fashioned punishment does. They hurt the young people and induce more “subdued” and “compliant” [81] behaviour. And they produce young people who are “easier to handle”. [82] These terms all come from within the ADHD narrative. In the NICE Guideline a parent describes (with a sigh of relief) how the drugs made their son “compliant”. [81] “Subdued” comes from the report on the wonderful effects on disruptive young people by the original discoverer. (See Section 3) ii)). The term “easier to handle” comes from a 1970s ADHD study designed to assess the long-term efficacy of methylphenidate. [82]  We review some aspects of this paper, Weiss *et al*. 1975, in the next section.

But surely this is all backed-up by scientific studies?

vii) The “scientific” studies which the NICE Guideline authors use to justify drugging

*a) The studies are all short-term but ADHD drugging is in the long-term*

The studies NICE use to justify drugging are short-term studies; but ADHD drugs are generally applied in the long-term.

The NICE Guideline authors reviewed 49 studies in their review of studies to assess the benefits of “pharmacological treatment”. 18 of these compared methylphenidate to placebo. [83] The average duration of the 49 studies appears to be about 60 days. Only 5 appear to have lasted longer than

100 days. Some were for as few as 7 days. It is these studies which showed a “reduction in ADHD symptoms” and “conduct problems”. However, it is the case that a young person who is prescribed “medication” “for ADHD” will typically be on “medication” for a much longer period than this. The case studies in the Guideline document itself bear this out. As we noted above (sub-section vi) d)) these three case case-studies all showed examples of young people being drugged over several years. In general it appears that once a young person is started on drugs (at whatever age) they will be likely to stay on them until late adolescence. (See Section 4.4.3 Case Studies D to F).

The claims about “benefits” (symptom scoring system) are typically made on the basis of studies which last about two months. When longer terms studies are conducted the evidence is that even these supposed “benefits” tend to decrease over time. In terms of a comparison between “medication” and a behavioural intervention, the evidence from the MTA follow-up study was that the slight advantage on the symptom scoring system which drugging had over a behavioural intervention at 14 months was not maintained at 36 months. (Section 2) vii) and sub-section v) above). In their review of the possible long-term harms of methylphenidate the NICE authors cite Weiss *et al.* 1975. [84] This paper is used to show that long-term use of methylphenidate does not cause emotional problems. However, Weiss *et al*. also reported on whether there are any “benefits” to long-term drugging with methylphenidate:

> Our failure to demonstrate a better 5-year outcome in adolescence in the children who had received methylphenidate for 3 to 5 years than in children treated with chlorpromazine or not treated at all is difficult to explain, because methylphenidate has proved itself efficacious in several short-term drug studies and in clinical practice. [82]

The detail is even more telling:

> Hyperactivity scores decreased significantly over the 5 years in all three groups ($P < 0.01$) (Table I). Analysis of covariance indicated that there was no difference in the degree of improvement on this measure between the three groups (Table II). [82]

The 3 groups were those treated with methylphenidate (for 3 to 5 years), those treated with chlorpromazine (for 18 months to 5 years) and those not treated at all. The finding was clear. After 5 years, in terms of “hyperactivity” those who had been on methylphenidate had not improved more than those who were not been drugged at all. Characteristically, for ADHD drug enthusiasts the authors found their results “surprising” and “difficult to explain”. [82] This shouldn't be the case. Stimulants have no enduring effect. Once a young person stops taking them they will immediately revert to their previous position. In Weiss *et al.* 1975 drug treatment was discontinued two weeks prior to assessment. Thus when the measurements were taken the group who had been drugged

for between 3 and 5 years were in effect in the same position as the ones who had not been drugged at all. Their 3-5 years on methylphenidate had had no enduring effect. The most likely explanation for why there was a convergence of scores in the MTA follow-up study is because the effects of engaging in a behavioural programme do endure beyond the period of participation. Behaviour programmes can produce a long-term benefit. A behaviour programme thus has the potential to liberate an individual. Once they have completed the programme they may continue to benefit from it. No one has to pay for this continuing "benefit". It is the result of learning. A drugging regime creates dependency. They must keep taking the drugs to get the "benefit".

The NICE authors must have read Weiss *et al.* 1975. They use it to make a claim about how long-term use of methylphenidate does not lead to negative emotional outcomes. It would appear however that they found the study did not meet their criteria for included studies used to assess the benefits of methylphenidate. Appendix 17.5 lists Weiss 1974 as an an excluded study:

> WEISS1974 Abstract only; inappropriate comparator [MPH vs. Chlorpromazine vs.no meds.] [71]

The study referred to in Appendix 17.5 as WEISS1974 must be the same study the NICE authors refer to in the text as Weiss *et al*. 1975. It uses the same comparators, has the same four authors and has a nearly identical title. It is not clear why a study which compared methylphenidate and another drug to an untreated group could not be used for purposes of comparing methylphenidate to the untreated group. All you have to do is discount the results for the other drug. In any event, whether specifically excluded or simply not used, Weiss *et al*. 1975 was not used to assess the benefits of "medication". Was Weiss *et al*. 1975 not used to assess the benefits of "medication" because it showed that there were no enduring benefits beyond treatment end, at all? The NICE authors were prepared to use the same paper to make a claim for the lack of harm for long-term use of methylphenidate. This appears to be a particularly egregious example of the way that the "evidence" is mined selectively to build the case for drugging.

The above considerations show that when studies run by believers in ADHD drugging are run into the longer term (over a few years) they consistently produce evidence that shows no long-term enduring benefit beyond the end of the treatment. No wonder the vast majority of the studies are for at most 14 weeks. The manufacturers know perfectly well that all the "benefits" that there are can be demonstrated in this time period.

Short-term drug studies do not provide a possibility of noticing harms which occur over long-term use. The US FDA approved label for Ritalin says:

> Sufficient data on safety and efficacy of long-term use of Ritalin are not yet available.

[85]

The NICE Guideline authors are aware of this problem (after a fashion) and they set out to find some long-term “clinical” and “observational” studies to address the question. The NICE Guideline author's definition of a long-term study for evaluating harm is two months. [86]. Since use is typically for several years it is hard to see how a two month study could in fact provide a valid clinical picture of the possible long-term harms associated with stimulant drugging.

For methylphenidate the NICE authors found 9 studies or reports [86] which provided information about its potential for causing “long-term” harm. One of these was the FDA report concerning serious adverse events, which we have previously mentioned. [56] Another was the MTA follow-up study (which was intended to further promote ADHD drugging, but went wrong). Another was a 2 year study of young people with tics or Tourette's syndrome. Based on their assembled hodgepodge of 9 studies we can see that no great efforts have gone into researching the long-term harms of methylphenidate. These 9 studies do not represent a systematic attempt to investigate the possible long-term harms of methylphenidate. This isn't really surprising; most drug studies, as we shall see in the next section, are funded by pharmaceutical companies as part of their promotional efforts for the drugs. Naturally they do not fund research into the harms which their products do.

Included in these 9 studies is a 5 year study which the NICE authors claim found “no significant differences between children taking methylphenidate and those taking placebo with respect to emotional adjustment, delinquency or the mother-child relationship”. This is Weiss *et al*. 1975 which we have already mentioned in the above. Weiss *et al*. 1975 did indeed report that compared to an untreated control group methylphenidate did not cause any significant harms in terms of emotional adjustment. It is worth pointing out that Weiss *et al.* 1975 was not intended to look for harms. They set out to try to find a long-term benefit for stimulant “medication”. NICE use the finding of no benefit on emotional adjustment to make a claim about no harm. The study also reported that a group treated with methylphenidate for 3-5 years failed to show a greater improvement on a score for hyperactivity than an untreated group. As we have noted above, NICE did not use Weiss *et al*. 1975 for the purposes of assessing drug benefit and so did not report this result.

This was not the only way in which the NICE authors made selective use of Weiss *at al.* 1975. This is how the NICE authors report on what Weiss *et al*. 1975 found about growth:

> the growth curve increased after methylphenidate was discontinued (Weiss et al., 1975). [84]

That sounds good. There is a slowing down of growth but growth picks up again after the “medication” was discontinued. (The fabled growth rebound). But is this what Weiss *at al.* 1975 actually reported? It certainly wasn't the spirit of their treatment of this subject. They said:

> Data for growth curves were obtained in a clinical manner without stringent research methodology, and an untreated hyperactive control group was unfortunately lacking. Nevertheless, inspection of the growth curves of those children who took methylphenidate for 3 to 5 years gives some cause for caution and concern. Findings suggest that children who take methylphenidate even in moderate doses for several years may in some cases fail to grow at expected rates. [82]

It is true that they reported that for 8 of the 12 children who stopped receiving methylphenidate after 3 years did show a growth rebound (page 163 in the study). However; their general report was one of concern. And, by their own admission, for this measure there was no control group. Thus the data does not meet the standards for a randomised clinical trial on this measure. The way that NICE reports Weiss *et al.* 1975 on the subject of growth is in effect to misrepresent the paper.

Based on the somewhat random collection of studies which they used to asses the possible long-term harm of methylphenidate the NICE authors find enough evidence to make a brief (4 paragraph) summary of “key findings”. They record that growth “may be affected”. That “there is evidence of tics”. That one study reported a problem on one measure of blood pressure. And that the data on possible adverse cardiac events was inconclusive. Absent from this review is any concern for the subjective experience of the drugged young person. 10 years of sleeplessness may not cause a health problem for a young person requiring a health intervention. But it can hardly be much fun.

The NICE Guideline authors tactility admit that ADHD drugging does not lead to any better long-term outcomes:

> Longitudinal studies indicate that ADHD symptoms are predictive of both current and future impairments [87]

and (as we have already seen):

> There is little evidence that stimulant medication alters the relatively poor long-term outcome for many of those with ADHD (Weiss & Hechtman, 1993). [12]

Long-term use of methylphenidate does not lead to long-term improvements or better "outcomes" for a young person. So; what does it achieve? The Weiss *et al*. 1975 study we have discussed above contains the telling phrase:

> Although the hyperactive child on stimulants generally becomes easier to handle, his ultimate outcome may be only slightly or not at all affected. [85]

In an appendix to the NICE Guideline which includes counter-points of view (which don't form part of the recommendations) the noted critic of ADHD-drugging Dr Sami Timimi quotes Dr William Pelham as saying:

> No drug company in its literature mentions the fact that 40 years of research says there is no long-term benefit of medications. That is something parents need to know. [88]

Typically using the symptom reduction scoring system short-term studies (almost all less than 14 weeks) are used to generate claims about the "benefits" of stimulant "medication". The rare longer-term studies produce evidence that there is no enduring benefit beyond treatment time and, relative to behavioural interventions, the "benefits" tend to wear off. Yet ADHD drugging is typically in the long-term.

The situation with atomoxetine is similar to that with methylphenidate. The NICE Guideline authors found only two studies [89] which they could use to assess the potential long-term harm of atomoxetine compared to the 14 studies [90] they found they could use to assess its "clinical efficacy". With both methylphenidate and atomoxetine then there are systematic efforts to produce evidence for symptom reductions. There are no systematic efforts to investigate possible long-term harms. The NICE Guideline authors omit to discuss the possible reasons for this fact. Could it just be because the "dominant scientific-medical" paradigm which provides their "evidence base" is influenced by the commercial interests of pharmaceutical companies?

*b) Most of the studies in the "dominant medical scientific paradigm" turn out to be commercial endeavours*

In Appendices 17.5.1 and 17.5.2 [71] the Guideline authors list 56 studies which they referenced for evidence about pharmacological "treatment" in young people and adults. Of these; 35 were funded in part or in whole by pharmaceutical companies, 5 were funded by the US NIMH (well-known for its support of ADHD drugging) [91], in once case funding is not given but it is noted that the study authors are funded by pharmaceutical companies, and for 9 funding was not recorded or is not given. Just 6 appear to have been funded by other types of organisations

(including a government and a health insurance provider). Thus, of those where funding was recorded 64% were funded directly or indirectly by pharmaceutical corporations. 8% were funded by the NIMH which has a strong pro-drugging position. In 16% of cases no data is given. Some of these may also have been drug company funded. The majority of the studies, possibly a large majority, are directly funded by pharmaceutical companies.

The situation is especially striking for Strattera. Of the 14 studies used be the NICE Guideline authors as evidence for the benefits of Strattera 13 were funded by Lilly USA who makes the drug. In the other case the funding is not recorded. Thus in all cases where funding is recorded the “trial” was funded by the manufacturer of the drug. Strattera is a relatively new ADHD drug. It was first licensed for use in the UK in 2004. [92] These studies then were concerned to facilitate the entry of a new drug to the market and were paid for by the manufacturer. It is entirely misleading of the NICE Guideline authors to cite these studies as medical scientific evidence of a benefit to young people. They are commercial efforts to promote a commercial product.

The studies typically use “ADHD symptom” score-cards. The Connors rating system features heavily. This is a commercially available copyrighted check-list of “ADHD symptoms”. One standard Connors question for teachers is whether the student has been “in trouble with the police”. [93] This makes it clear that young people are being drugged for being a social nuisance.

There is no research into the physiological basis for ADHD and thus its treatment. There can't be because ADHD isn't a biological condition. The drugs are not the fruit of medical-scientific research which links a drug to a specific biological process in the body as for example, the drugs given to HIV positive patients are. Clinical trials for “ADHD medication” are all, or almost all, designed to show “symptom reduction” scores, using standard ratings scales, for the drug in the short-term. The majority of such studies are funded by the manufacturer of the drug being tested. The ratings scales are derivatives of the diagnostic check-lists invented by psychiatry which define “ADHD”. The drugs have been shown (using parents and teachers as raters in the main) to control the “disruptive” behaviours defined by psychiatry as constituting the “diagnostic category” of ADHD. This is a circular process owned by psychiatry and the pharmaceutical companies which enlists parents and teachers as (willing) adjuncts. This is not about a medical treatment for a biological condition.

Multiple research projects have identified that studies funded by manufacturers are more likely than those funded by government funded bodies to find positive results for the drugs they are testing. One such paper, published in BMJ in 2003, concluded:

> Systematic bias favours products which are made by the company funding the research. Explanations include the selection of an inappropriate comparator to the product being investigated and publication bias. [94]

This result has been replicated in other similarly constructed studies. It is not a surprising result. A pharmaceutical company setting up a trial is doing so in order to prove their product so they can take it to market and compete with other products. They are not paying to carry out an impartial investigation. They will be careful to avoid setting up a study which might expose deficiencies in their product, for example a lack of long-term efficacy.

A recent book “Bad Pharma” by Ben Goldacre investigated the pharmaceutical industry. It is reviewed by the Guardian newspaper. The Guardian summarises:

> New drugs are tested by the companies that make them, often in trials designed to make the drug look good, which are then written up and published in medical journals. [95]

No one disputes the “facts” of the 49 clinical trials used by the NICE Guideline authors to justify ADHD drugging in the UK. (The Appendix lists 56 studies. The text refers to 49 studies. We haven't been able to resolve this minor anomaly. Perhaps some of the studies listed in the Appendix were not used).

These trials though are more like the “trials” which a washing powder manufacturer conducts in order to be able to make “truthful” claims about their product which they can use in an advertising campaign, than serious clinical investigations. It is somewhat surprising then that the NICE Guideline authors can say:

> It is accepted that the research literature reflects the dominant medical scientific paradigm and hence the nature of the evidence base. [15]

Psychiatry depends on the biological model of mental illness and childhood behavioural disorders. This is the point on which psychiatry differs from clinical psychology and is its cornerstone. This means that psychiatry has a dependency on the pharmaceutical industry. It needs the pharmaceutical products in order to continue to impose the biological model, which justifies its existence as a profession. In turn the pharmaceutical companies depend on psychiatry to produce the ratings scales which show that their products do something (even if it isn't cure an illness). In effect there is a merry-go-round. The relationship of mutual dependency is existential. Each depends on the other. The flows of funding from the pharmaceutical industry to psychiatry can be understood as the lubricant of this deep and intertwined relationship. The flow of funding creates publishing opportunities, speaking opportunities and opportunities for professional development. The American Psychiatric Association for example receives funds from pharmaceutical companies. [96] The lead author of the NICE Guideline and the research department to which he belongs have

both “received fees for lecturing at educational meetings and scientific conferences that had sponsorship from pharmaceutical companies – including Eli Lilly and Janssen-Cilag, who manufacture drugs used in ADHD”. [97] And many of the ADHD studies are, as we have seen, funded by the drug companies.

It is in this context perhaps that the surprising willingness of the NICE Guideline authors to simply “accept” the skewed nature of the “evidence base” on ADHD can be understood.

viii) Making them fit into school

> Once again we had a son who seemed more compliant... [98]

Parent of an “ADHD child” interviewed by NICE after the son was prescribed Concerta (methylphenidate).

> Although the hyperactive child on stimulants generally becomes easier to handle, his ultimate outcome may be only slightly or not at all affected [by drugging]
>
> Our impression was that methylphenidate was helpful in making hyperactive children more manageable at home and at school, but did not significantly affect their outcome after 5 years of treatment.
>
> [82]

ADHD researchers in the 1970s.

This is the goal of ADHD drugging. Young people (boys chiefly) become “more compliant”, “more manageable” and “easier to handle”. The fact that the young people would rather not be on the pills is discounted altogether:

> As soon as medication was discontinued we received complaints from nearly all of the teachers of these children (many of whom had not known that the children were previously on medication). Most parents also found those 2 weeks very difficult, but the children on the whole preferred being without "the pills". [82]

The “easier to handle” benefit of stimulant “medication” is hardly startling. A key part of the definition of “ADHD” in DSM-IV is that the behaviour is “disruptive”. Since the whole aim of ADHD drugging is to reduce the “signs” “of” “ADHD” it follows that the aim is to make the drugged young people less “disruptive”.

It comes as no surprise then that when the NICE Guideline authors turn their attention to educational interventions for “ADHD children” the focus is, as it was with drugging, on “reducing ADHD symptoms”. The “signs” of “ADHD” are defined as disruptive behaviour which is “inappropriate for developmental level”. (See Appendix i)). At stake is a set of young people whose ability (for whatever reason, biological or otherwise) to pay attention in class is sufficiently below that of the class average for it to become a source of disruption. The most obvious solution might perhaps be to take them out of the large class where this is a problem. If that were to happen though there would be no ADHD. In other words, ADHD is predicated on maintaining the existing schooling system. The educational interventions assessed by the NICE authors are focussed on managing the “ADHD child” in the classroom. Nothing more fundamental than that.

In the section headed “Interventions For Children With ADHD In Educational Settings” the Guideline authors review 6 studies. It is significant that the Guideline authors could only find 6 studies on educational interventions compared to the dozens they found for drug interventions. Of the six studies three involved giving advice to teachers; for example sending a booklet to schools “containing information about ADHD” or engaging teachers in some in-service training sessions. Two appeared to assess the impact of a teacher training with or without a parent training programme. One study investigated a method of managing “children’s” behaviour with a system of “commands” and “warnings” and “threats” to “improve student compliance”.

The study which assessed the system of commands and threats was Kapalka *et al*. 2005 [99]. The behaviour management method investigated is known as “reduced repetitions”. In this approach the “child” is given a “command” and if they do not carry it out the “child” is warned and if they still don't comply in the words of the NICE authors “the threat is carried out”. [100] The “reduced repetitions” approach was evolved by someone called R. A. Barkley. His approaches were used in the behavioural intervention programme on the MTA study. Kapalka *et al.* 2005 taught some “teachers of ADHD children” the “technique” and some not. The teachers who used the technique got more “compliance” than those who didn't. This showed that quickly following up a command with a threat and then acting on that threat was more effective in securing compliance with the command than in repeating the command multiple times. (Leaving aside the “threat” aspect this is a truism of parenting). We can note that the emphasis is on obedience and compliance. Another study by Kapalka, Kapalka 2004, (not used by the NICE authors) also established that if parents use a “stare technique” and maintain eye contact for 20 to 30 seconds “following the command” that gets more compliance. [101] Obviously this is an invitation to objectify the young person.

Kapalka *et al.* 2005 simply shows that “compliance” can be secured by this method. The study does not appear to even base its claims directly on ADHD. The subjects were, according to the NICE authors, diagnosed ADHD with an “unknown tool”. The only assessment used was the School Situations Questionnaire (SSQ). The School Situations Questionnaire is a ratings scale developed by an individual ADHD practitioner “to gather information from teachers about behaviours and symptoms directly associated with Attention Deficit Hyperactivity Disorder that may displayed in a classroom setting”. [102] [103] In fact the tool was developed by R. A. Barkely who was also the author of the behavioural control method of “reduced repetitions” which was being “assessed”.

Kapalka 2005 also shows a nice example of the kind of circularity in the ADHD narrative which we discussed in the Introduction (sub-section iii)):

> Students with attention-deficit hyperactivity disorder (ADHD) often exhibit non-compliance that presents a significant management problem for classroom teachers. [99]

“Non-compliance” is presented as a feature of “ADHD”. In fact it could not be otherwise. Non-compliance is one of the main points at stake in getting “diagnosed” “with” “ADHD”. (See Appendix i) DSM-IV). The emphasis on non-compliance and compliance in Kapalka 2005 reminds us that the “symptoms” of ADHD are inconvenient behaviours which “present a significant management problem for classroom teachers”. Not all illness from which anyone suffers.

3 of the six studies used by the NICE Guideline authors for educational interventions appear to use the Conners rating systems. One uses another behaviour check-list system called the “Child behaviour check-list”. As we have mentioned above Kapalka 2005 uses the “Schools Situations Questionnaire”. What is being assessed with these ratings systems are “ADHD symptoms” and derivative behaviours. (The Schools Situations Questionnaire also appears to cover other “disruptive behaviours”). This use of pseudo-clinical ratings scales objectives the problem *in* the “child”. The demands of the teacher and school situation are absolutely reified. The NICE authors make a token gesture in the direction of considering how the school might to change to meet the needs of the young person. They cite one study (not one they referenced for their review of the “clinical evidence” for educational interventions) which proposes amongst other approaches more “stimulating activities”. But there is no serious discussion of an educational provision built around the needs of young people. (Even the paper which proposes more “stimulating activities” includes the inevitable punishment system, including isolation and taking away “tokens or points if the child misbehaves”). [104]

Of the 6 studies which NICE used to investigate educational interventions 3 were concerned with

evaluating the effect of giving advice to teachers. Of these only one compared giving advice to teachers directly with not giving advice. This was Tymms *et al*. 2006. [105] The advice was given in the form of a booklet. The NICE Guideline authors report:

> The evidence suggests that there is little to no effect in providing advice to teachers in relation to children's ADHD symptoms or academic achievement. [106]

and

> There is limited evidence from one study (TYMMS2006) of the combined effect of advice given to teachers and screening. The results indicate little to no effect in children's ADHD symptoms or academic achievement. [106]

This is surprising. Tymms *et al*. sum up their results much more positively:

> For school-level interventions, advice had a significant positive effect on the attitudes and behaviour of pupils with ADHD characteristics but not on their attainment levels. [105]

and

> It was calculated that providing schools with research-based advice on how to work with inattentive, hyperactive and impulsive pupils in the first two years of schooling is cost-effective and could be beneficially used on a wide scale. [105]

This practice, of minimising the positive value of non-drugging interventions is commented on by ADHD critic Dr Peter Breggin. He quotes a psychiatrist, Lester E. Shapiro, who, in 1991, wrote an opinion column in *Psychiatric News* (the newsletter of the America Psychiatric Association):

> It is far better that we engage in a serious examination and dialogue of the issues I have raised than to act in collusion with an industry whose goal is to increase drug usage by broadening indications for their drugs, advocating long-term administration, minimizing adverse side-effects, overstating effectiveness, de-emphasising adjunctive treatments or denigrating generic drugs. [107]

The two summaries of the results of the Tymms 2006 study may not be altogether incompatible. Nonetheless the NICE authors have chosen to emphasise the areas in which Tymms did not report

a result, namely academic achievement and, apparently, “ADHD symptoms”. The result of a positive effect on behaviour and the remark that this was achieved at a low cost was not reported by NICE. Once again; they can be seen to be selective in their treatment of the material.

The NICE authors found two studies which compared the effects of teacher-training programmes with control (no intervention). One of these was Bloomquist, M.L 1991. [108] Bloomquist 1991 compared a basic teacher training intervention with a more complex one with no intervention. (3 treatment groups). The NICE authors report that there were some positive effects when the multi-component teacher training intervention was compared to control (no intervention) but they were not statistically significant and “there was little to no effect of this intervention on reducing children’s ADHD core symptoms”. [109] This may be the case. Nonetheless the authors of this paper reported:

> The multicomponent CBT condition was significantly better than the other conditions at improving observed off-task/disruptive behaviour at post-test [108]

That is the CBT intervention which involved parents, teachers and the young people was more effective than nothing and better than an intervention which just trained teachers at achieving better scores on a behaviour rating scale. Again; the summary of the study by the NICE authors is not wrong. But they have chosen to emphasise the negative results and not the positive results. And their summary is less positive about the results than that of the study authors.

The second study which the NICE authors used to assess the effectiveness of teacher training interventions was Barkley R. A. 2000 *et al*. [110] Barkley R. A. 2000 compared three interventions with each other and with no intervention. The three interventions were a teacher training programme linked to teaching being delivered in special classes, a parent intervention and a combination of these two.

Again the NICE authors report some positive effects but say that for both the teacher training intervention and the combined parent and teacher training intervention they were not statistically significant. Nonetheless Barkley *et al.* 2000 reported:

> The classroom treatment produced improvement in multiple domains: parent ratings of adaptive behavior, teacher ratings of attention, aggression, self-control, and social skills, as well as direct observations of externalizing behavior in the classroom. Neither treatment improved academic achievement skills or parent ratings of home behavior problems, nor were effects evident on any lab measures of attention, impulse control, or mother-child interactions. [110]

Once again; the study authors present a more positive account of their results than the NICE authors who seem determined to only focus on the negative aspects of these interventions.

The NICE authors summarise the findings concerning teacher training (multicomponent or basic) thus:

> To summarise, there is some evidence that teacher-training and multicomponent teacher-training involving parent training and child interventions have a small effect in improving the behaviour of children with ADHD. Because of the lack of statistical significance of all these results, the findings are inconclusive. [109]

Again; NICE can be seen to minimise the results of these studies. Bloomquist, M.L for example reported that the finding for a combined CBT intervention was “significant” in terms of improving on-task/disruptive behaviour. The positive results in terms of an advice booklet and teacher training on some aspects of disruptive behaviour should surely be investigated further. The NICE authors call for more research into teacher training but focus their call on research into “improvements in ADHD symptoms” and “academic achievement”. That is on the areas which reason, as well as the studies they have reviewed, suggest are less likely to respond to these interventions. For example; if more teacher training could “raise the academic achievement” of young people with impulsivity problems who are (by definition) already below the ability for the class it would be a miracle. While focussing on these areas, the NICE authors ignore the findings that some areas of behaviour can be improved by these kinds of interventions.

Some young people are significantly below the developmental level in terms of attentiveness and impulse control, (and in the main IQ), which is typical for their age. Developing strategies to manage this better within the existing classroom set-up will achieve only somewhat limited results. The elephant in the room in NICE's discussion here is; if these young people are disruptive and below the level which is “appropriate” for their age and if this is indeed something “in” them, that is a property they have, all of which is enshrined in the definition of “ADHD” then - why not grip the bull by the horns and take them out of the classroom where all this is a problem? If, for example, a young person aged 10 is really struggling to consume the academic diet which an educational committee somewhere has determined is suitable for a 10 year old at what point does it make sense to stop trying and try something else, that is give them something which is suitable for them, which is commensurate with their actual abilities at this time? Be definition if they have an ADHD label they are not at the expected standard of behaviour for their chronological age or “developmental level”. Trying to force them to fit into school having just “diagnosed” them as not being able to fit in at the present time seems fundamentally negative. The drugging option doesn't lead to any better results or “outcomes” for the young people in the long-term. They just suffer nausea and head-aches for 10 years so they don't disrupt the rest of the class. In short; the ADHD

programme is about trying to make square pegs fit into round holes. It can't really do this. The square pegs can be got to be more “subdued” and “easier to handle”. But they don't become round pegs. If these comments are interpreted as calling for separate schooling for special needs students that would be an error. The problem is mass schooling. Any regimented system where cohorts of year groups are supposed to move forwards in unison is bound to produce a few stragglers. Yes; taking the stragglers into separate classes is better than drugging them. But, better still, would be to re-think mass schooling. “ADHD” exposes the nature of this anti-educational system. This system forces in rather than “draws out”. “ADHD young people” are (some of those) who won't or can't be force-fed. If no one at all was being force-fed there wouldn't be a problem. That is; if education was designed around the needs of young people, rather than young people being manipulated to fit the needs of the education system, there would be no need to drug some young people to make them fit in. (Even though it doesn't really work anyway).

There is no sign that the NICE authors have given any serious consideration as to what kind of educational provision might benefit or be suitable for young people who can be categorised as having unusually low attentiveness in classroom situations for their age group. The focus is resolutely on getting compliance with the demands of the school system as it is. At the same time the definition of ADHD defines ADHD as being a “condition” characterised by “significant impairment”. [112] This is a striking anomaly. It just seems odd that no one is talking about what kind of provision would be suitable for these young people who, by their own system, suffer from “significant impairment”. It is as if on the one hand psychiatry defines a disability but then, on the other, sees this as a fault to be corrected. This response to defined disability is not the normal social response to disability. The normal social response is to seek to fit the person's environment around their disability. Not to try to force them to fit into the environment.

The recommendations for educational interventions do not even countenance alternative and more suitable forms of provision. Indeed the NICE Guideline authors take the opportunity to promote the “diagnosis” (and therefore, incidentally, their profession). The recommendations are chiefly around increasing communication. For example if a “child” is “diagnosed” the “healthcare professionals” should contact the teacher and explain the “diagnosis” and care plan. Equally; if a SENCO (special needs co-ordinator in a school) “suspects” ADHD they should inform the parents and advise them about any local parent training programmes. There is also a recommendation that The Department for Children, Schools and Families should consider developing training programmes for trainee teachers to help them “support children with ADHD”. None of these measures, even if adopted, would alter one iota the concrete situation of the young person. At best some of the behaviour techniques that the “research” has found to be effective at “managing children with ADHD” would be introduced. On the basis of their own review there is no evidence that this would do anything to improve the core “symptoms of ADHD”, that is inattention and hyperactivity. There could potentially be an improvement in “conduct problems”. However; an improvement in “conduct problems” does

not have any obvious benefit for the young person. An improvement in “conduct problems” chiefly improves the ordered running of the school. The main effect of the recommendations seems to be that the concept of “ADHD” would be further promoted and would become more deeply embedded in the daily life of schools, while nothing at all would be done to meet the needs of young people who are said to have “significant impairment”.

A research recommendation is:

> While universal screening of the school population is not recommended, teachers may benefit from receiving some training to help them spot children who are suspected of having ADHD in order to initiate referrals and to implement support packages at the earliest possible stage. [113]

The language of “spotting” those with “suspected ADHD” indicates that teachers are being recruited to generate more “diagnoses”. As we have seen; if the young person who has been “spotted” as “having suspected ADHD” fails to confirm this in the psychiatrist’s office it isn’t because he doesn't “have ADHD”. It's because he is “regulating his behaviour”:

> Direct observation of an individual with ADHD, particularly older adolescents and adults, for short periods of time during assessment sessions may not demonstrate any obvious features of the condition. This should not exclude the diagnosis where there is a clear account of inattentive, impulsive or hyperactive behaviours in usual situations. [18]

In these circumstances:

> The GDG advises that diagnosis should only be made on the basis of a full assessment. [18]

The “full assessment” and “clear account” will include reports from the teachers who have have referred the young person with their “suspected” ADHD. This system clearly provides a mechanism where psychiatrists and paediatricians may just “rubber-stamp” applications from teachers to have a young person “diagnosed”. This mechanism in effect extends the franchise on ADHD “diagnosis” and hence ADHD drugging to teachers.

The recommendations concerning educational provision are mostly focussed on propagating information about the “diagnosis” to teachers and parents. The result of these recommendations if

implemented would be to sure up the position as regarding the “validity of the ADHD diagnosis” in schools. Completely absent from the recommendations is any kind of consideration concerning alternative educational provision of any kind. This despite the fact that the definition of “ADHD” is that the young people are “significantly impaired” and are “hyperactive” and inattentive to a degree which is “inappropriate for developmental level”.

ix) The recommendations aren't “evidence-based” at all

To recap the recommendations, they are:

> Drug treatment is not indicated as the first-line treatment for all school-age children and young people with ADHD. It should be reserved for those with severe symptoms and impairment or for those with moderate levels of impairment who have refused non-drug interventions, or whose symptoms have not responded sufficiently to parent-training/education programmes or group psychological treatment. [114]

and

> In school-age children and young people with severe ADHD, drug treatment should be offered as the first-line treatment. Parents should also be offered a group-based parent-training/education programme. [115]
>
> If drug treatment is not accepted by the child or young person with severe ADHD, or their parents or carers, healthcare professionals should advise parents or carers and the child or young person about the benefits and superiority of drug treatment in this group. If drug treatment is still not accepted, a group parent-training/education programme should be offered. [116]

These recommendations only have the status of recommendations. No individual psychiatrist is obliged to follow them. Even if they are followed they still allow the individual practitioner to prescribe at will. There is no specific diagnostic category of “severe symptoms” so this is open to individual judgement. The recommendations also allow parents to decline to accept a parent-training programme and opt instead to have their child drugged.

In private practice psychiatrists may be more inclined to provide a diagnosis and prescription on demand than in state practice. The clients will be the parents. Like any business they will need to satisfy their clients. One of the case studies included in the NICE Guide concerns a parent failing to get their son diagnosed by the NHS (Case Study E). They were referred to a parent training

programme by their doctor. The parent claims that there were no spaces available. They then got funded by the NHS to get a diagnosis from a private psychiatric clinic. The clinic also put their son on methylphenidate. [36]  There is no evidence that in this case the parents were telling anything other than the truth. However; it seems evident that parental attendance at parent-training programmes can be patchy. In Barkley *et al.* 2000, which we discussed above, it was reported that attendance by parents at the parental intervention part of the programme was scarce:

> Results showed that parent training produced no significant treatment effects, probably owing largely to poor attendance [110]

In the MTA study parental attendance at the parental part of the programme was 77.8%. [24] No doubt there are some, possibly many, parents who would prefer not to attend a parental training programme. Given the choice between this and drugging their son they will choose drugging. The recommendations by the NICE authors will facilitate these decisions. Arguably it will be just the parents who make this kind of decision who *might* have poor parenting skills. A second case study (Case Study F) included in the NICE Guide tells the story of parents who accidentally received a letter sent from a psychiatrist to their GP about their son. The letter, apparently, said that their son's behaviour was due to “poor parenting”. The parents kicked up a fuss and demanded a second opinion from another psychiatrist who duly diagnosed ADHD. Shortly afterwards this boy was also on methylphenidate. [98]  Again; it is possible that this was a perfect set of parents. But it is clear that, perhaps even within the NHS, if parents demand loudly enough that their son be diagnosed and drugged that will happen.

The recommendations appear to be balanced. No doubt they were intended to appear so. The recommendation that “medication” should only be used for those with “severe symptoms” sounds “responsible”.  This recommendation appears to be derived from a single secondary evaluation of the data type study. This does not provide a basis for making a clinical recommendation, which is probably why, as we discussed above, (sub-section iv) b)), it is presented outside of the main flow of the document which reviews evidence and then makes recommendations based on that evidence. It appears, separately, in a section entitled “Further considerations with respect to the treatment of ADHD – additional evidence from the MTA study”. So; this cannot then be the basis for the major national recommendation about the appropriateness of “medication” for those with “severe symptoms”. But what then is?

Even if Santosh *et al*. 2005 were to be accepted as “clinical evidence”, even then it could not form the basis for the recommendation about the “the benefits and superiority of drug treatment in this group”, that is for those with “severe symptoms”. All Santosh *et al*. 2005 showed was that in the MTA study for a retrospectively extracted “severe” ICD-10 sub-group there was a somewhat greater “advantage” (symptom scoring system) for the special MTA “medication” regime over the

MTA behavioural programme, than was apparent in the overall results. The absence of a control group from the MTA study means that absolute claims about the efficacy of any of its treatments cannot be made. Therefore no absolute claim can be made about the "superiority of drug treatment in this group". Santosh *et al*. 2005 showed that for those in an ICD-10 group the difference between the "medication" regime and the behavioural programme was somewhat greater than for those in the wider ADHD group. This alone does not mean that "medication" should be recommended or is a "superior" treatment in preference to a behavioural intervention. The other factors would still need to be considered. These include the accepted harms that "medication" causes all users. (63% of subjects in the MTA study suffered side-effects). The MTA follow-up study indicated that the advantage for methylphenidate versus a behavioural intervention wore off in the longer term. It is likely that this would also apply to the statistical results for Santosh's ICD-10 group. Santosh *et al*. 2005 based their results on a (retrospectively identified) ICD-10 group. ICD-10 typically produces far less "diagnoses" than DSM-IV. The NICE authors say half as many. [13]. "Severe symptoms" is a much looser criteria. This term is deliberately flexible to allow as wide as possible scope for interpretation presumably. The MTA study compared methylphenidate versus a behavioural intervention; not atomoxetine. No study, not even a statistical one, appears to provide any basis for claiming the "superiority of drug treatment in this group" in connection with atomoxetine. So there is no basis at all not even on the basis of Santosh *et al*. 2005 to make claims for "drug treatment" in general being a "superior treatment" for any group.

NICE *appea*r to use Santosh *et al*. 2005 to promote their claim concerning the "superiority" of drug "treatment" for those with "severe ADHD". The NICE authors used Swanson *et al*. 2007 to attempt to mitigate the damage done to the drugging cause by the failure of the MTA follow-up study to confirm the "medication advantage". (See sub-section v) b) above). However one of the findings of Swanson *et al*. 2007 was that for 14% of young people in the MTA study despite 3 years of drugging their "symptoms" were the same as they were on day one. The "initial beneficial effect" had "completely dissipated". Tellingly, as Swanson et al. 2007 report: "This subgroup of 14% of the MTA sample was characterized by high initial symptom scores and baseline aggression, lower IQs, lower social skills, and other risk factors." [47] This would appear to argue very strongly against the idea proposed of a "superior benefit" in those with "severe ADHD". The evidence from Swanson et al. 2007 is that in the sub-group with "high initial symptom scores" after three years the "benefits" of medication had "completely dissipated". If the NICE authors had been seriously trying to engage with their "evidence-base" they could not have ignored this and they could not have offered a claim about the "superiority of drug treatment" for those with "severe ADHD". They treat their "evidence-base" entirely selectively.

The stated approach of the NICE Guideline authors was to conduct a meta-analysis. This means that the results from multiple studies are assessed in order to produce, so goes the theory, a more robust and reliable result than might be obtained in a single study. It is not clear why in the NICE Guideline which was intended to be based on meta-analyses where applicable [117] a single

statistical study *appears* to be informing the final recommendations. For all these reasons Santosh *et al*. 2005 provides only the most flimsy “evidence” for anything. It certainly cannot explain a national recommendation about “the benefits and superiority of drug treatment” for those with more “severe symptoms”. Aware of this perhaps, the NICE authors avoid making a direct connection between this study and their recommendations. But then, and again, what is the basis for this major claim shaping national policy about drugging being “superior” for those with more “severe symptoms”?

We noted that in the “research literature” reviewed by the NICE authors there appears to be no systematic attempt to investigate the long-term possible harms caused by “medication” and this contrasts with the abundance of short-term symptom reduction studies. For example 14 studies were found to assess the “efficacy” of atomoxetine but only 2 to assess the possible long-term harms. 18 studies were found to evaluate the “efficacy” of methylphenidate. But only 9 studies were found to assess the long-term harms. [86] These nine studies do not in any way though represent a systematic attempt to assess the potential harms of methylphenidate. Two were not studies intended to investigate harm. They were general studies which included measures of both “benefits” and “side-effects”; (Weiss et al., 1975 and Schachar et al., 1997). Two investigated the impact of methylphenidate on growth; (Spencer et al., 1996 and Swanson et al., 2007). One investigated the impact of methylphenidate on growth and possible adverse cardiac events; (Gittelman-Klein et al., 1988). Two investigated tics; (Gadow et al., 1999 and Palumbo et al., 2004). Gadow *et al*. 1999 did not include a control group. [118] Palumbo *et al*. 2004 was a small meta study of 5 other studies. It found only slight evidence of tics being exacerbated by methylphenidate and concluded that methylphenidate does not significantly cause or increase tics. [119] The ninth study was the US Food and Drug Administration safety review of methylphenidate which focussed on adverse cardiac events (FDA 2004). Growth problems, tics and possible adverse cardiac events relate to well-known concerns about methylphenidate. These are headline issues which cannot be avoided. This explains why these studies have been conducted. Young people taking methylphenidate routinely experience insomnia, stomach-aches, nervousness and sometimes psychosis. That side-effects such as these are common was confirmed in the MTA study. It appears to be acknowledged by the manufacturers of Ritalin. Insomnia and nervousness or irritability appear to be the most common side-effects. The NICE authors do admit that they occur. [120] But they do not appear to consider the impact of experiencing these effects day in day out for years on end. However, that is the reality for thousands of young people condemned to take stimulant “medication” to help them be less disruptive in class.

Also striking is the paucity of studies used to assess educational interventions (6). The studies which have been conducted are the ones linked to commercially exploitable products. Not ones linked to changes in educational provision. The NICE authors “accept” that “the research literature reflects the dominant medical scientific paradigm and hence the nature of the evidence base”. [15] But this appears to be a casual acceptance that “truth” can be determined by financial power.

The disparity between the conditions of the “clinical evidence” (short-term, focussing on “benefits”) and the conditions in which drugs “for ADHD” are actually taken (long-term, potential harms) should be enough to raise serious concerns about whether it is even possible to derive clinical recommendations from the available research literature.

The NICE authors reviewed psychological interventions. These are behavioural programmes and/or parent training programmes. They found 10 studies to include in a meta-analysis. They reported:

> Overall, the evidence shows that compared with control conditions psychological interventions for children with ADHD have moderate beneficial effects on parent ratings of ADHD symptoms and conduct problems at the end of treatment. These beneficial effects are sustained at follow-up 3 to 6 months after the end of treatment. If the small study by Pfiffner and McBurnett (PFIFFNER1997) is excluded from the analysis the effect of psychological interventions on conduct problems at the end of treatment remains positive, but beneficial effects do not reach statistical significance at the later follow-up. The meta-analysis therefore cannot be regarded as establishing that psychological interventions have sustained effects on conduct problems in children with ADHD. [121]

The NICE authors sound a cautionary note. These results reply on reports by parents:

> In the absence of evidence that psychological interventions have a positive effect on teacher ratings of ADHD symptoms and conduct behaviour, the evidence of beneficial effects based on ratings by parents should be interpreted with some caution. Parent ratings may be potentially subject to bias because in trials of psychological interventions for children with ADHD that do not use a control intervention, parents will know whether they and/or their child has received the intervention. [121]

This appears to be a case of “de-emphasising adjunctive treatments”. [107] (See above sub-section viii)). The NICE authors have not complained about the possibility of parent bias in, for example, the MTA study. In the MTA study for the ADHD “symptom” of hyperactivity only parents reported a “benefit” to “medication” and not teachers. The finding wasn't even confirmed by the neutral classroom observers. And the parents in that study also knew what “treatment” their child was on. And indeed 31% of the subjects were already being “medicated” by their parents before the study started. (See Section 2) v)). Nor is it clear why the NICE authors felt they had to exclude Pfiffner *et al*. 1997 from the meta-analysis. Pfiffner *et al*. 1997 found:

> Significant improvement in children's skill knowledge and in parent reports of social skills and disruptive behavior occurred for both treatment groups relative to the wait-list control group and maintained at a 4-month follow-up [122]

True; it only had 27 subjects, but then two of the other 10 studies used also had less than 27 subjects. Fehlings, D. L *et al*. 1991 had 25 subjects. [123] Hoath, F. E. *et al*. 2002 had 20 families. [124] Among the 56 studies accepted to show symptom reduction claims for pharmaceutical interventions there are 8 which had 30 or less subjects. [71] The NICE authors conclude that “The meta-analysis therefore cannot be regarded as establishing that psychological interventions have sustained effects on conduct problems in children with ADHD”. Nonetheless, even if we allow the curious exclusion of Pfiffner *et al*. 1997 it would appear that the meta-analysis has reported a benefit enduring beyond treatment time for “ADHD symptoms” (as opposed to “conduct problems”) for psychological interventions. This is in contrast to treatment with methylphenidate. As we saw (sub-section vii) a) above) Weiss *et al*. 1975 showed that there was no benefit (symptom scoring system) extending beyond “treatment” time for methylphenidate.

Even if taken on its own terms. ADHD is a valid “diagnostic category”, reducing “symptoms” is a valid goal of something called “treatment” etc. there remains a critical deficiency in the NICE Guideline. The two rival “treatments”, “medication” and behavioural interventions are assessed (according to the symptom scoring system). In terms of “symptom reduction” there doesn’t appear to be much to choose between them:

> While there is no evidence that psychological interventions are favoured over stimulant medication for any outcome, or at any time point, it is also the case that medication does not appear to be strongly favoured over psychological interventions. [27]

On the evidence reviewed by the NICE authors “medication” is associated with side-effects ranging from discomfort and “embarrassment”, through insomnia and psychosis, to death and suicidal despair. No such side-effects are reported for behavioural interventions. Not one. The conclusion should be entirely obvious. A further consideration concerns the ethical difference between a biological intervention and a behavioural one. A behavioural intervention engages with a subject's capacity to learn. This is why the effects can last beyond the period of the intervention. It is at least potentially humanistic. A biological intervention does something to someone at a biological level. The effects last as long as the drug is ingested. There may even be a falling off of effect as tolerance develops. (For example Weiss *et al.* 1975 commented: “Possibly when methylphenidate is given for 3 years or longer it becomes increasingly less effective and "tolerance" slowly develops”). [82] And even Swanson *et al*. 2007 countenanced: “the possibility of waning benefit for

continued medication beyond 2 years for a large number of children with ADHD". [47] Altering brain chemistry in order to effect a behavioural change is not a humanistic intervention.

We can witness in the NICE Guideline a single-minded determination to extract evidence in favour of drugging. The "evidence-base" is mined for extracts to favour "medication". The selective citations in connection with the MTA follow-up study is a case in point. Jensen *et al*. 2007 showed that the "medication advantage" tended to wear off. One attempt to recover the position was Swanson *et al*. 2007. This was only partially successful. Only the material from this paper which seemed favourable to drugging was used. For example Jensen *et al*. 2007 raised the possibility that the convergence of scores between the "medicated" and behavioural groups in the MTA follow-up was due to something they called the "self-selection" hypothesis. The idea was that young people with especially bad "symptoms" would start "medication" while those who were on "medication" and were doing well would stop. This would avoid the unpalatable conclusion that the "medication advantage" over a behavioural intervention wears off over time. One of the tasks of Swanson *et al*. 2007 was to test this hypothesis. They did so and reported that they failed to confirm it. This was not reported by the NICE authors in their discussion of Jensen *et al*. 2007. [125] The evidence from within their own "evidence-base" that the "positive effects" of medication may wear off in the longer term either in comparison with a behavioural intervention or compared with no treatment, are studiously ignored. The evidence that psychological interventions may have an enduring effect beyond "treatment" time seems to be minimised by the unexplained exclusion of a certain paper from the meta-analysis. (Though even when this is done the evidence still shows an enduring benefit for "ADHD symptoms").

As reported by NICE the MTA study showed that the results for the MTA behavioural intervention were similar to those for routine Community Care, which included "medication", for the majority of subjects:

> A further tentative inference from the data gathered at the end of treatment is that the intensive MTA behavioural intervention may have had similar effects to routine medication because the majority (66%) of the community care group received medication for ADHD and the behavioural intervention group did not differ significantly from the community care group for end of treatment outcomes. [26]

This is a much more applicable finding than the finding that the clinically atypical and heavily optimised "medication" regime of the MTA study scored better for attentiveness than the behavioural programme. It suggests that when the MTA behavioural programme is compared (symptom scoring system) against what young people actually receive it performs as well as a programme which includes drugging. This finding is further evidence from within the

“evidence-base” reviewed by NICE which should lead towards a recommendation for behavioural interventions.

The evidence from within the “evidence base” reviewed by the NICE authors points towards behavioural interventions being comparable with “medication” in terms of the symptom reduction system. Behavioural interventions may also have an effect which endures beyond treatment time. No such evidence exists for “medication”. Behavioural interventions cause not one of the serious and in some cases very serious side-effects associated with “medication”. If cost is a factor it seems that group based behavioural interventions are competitive with even the less expensive drug regimes. All this points irrevocably in the direction of behavioural interventions. (If the starting point is that “ADHD” is a “valid disorder”, it has to be “treated” and so on). A single secondary evaluation of the data study which shows that for a group of ICD-10 young people there is a greater gap between symptom reduction scores for a specific “medication” regime and a specific behavioural intervention than there is for a wider group of ADHD young people does not provide a basis for a recommendation about drug “treatment” being especially suitable for any category of ADHD.

The NICE Guideline is not a work where clinical evidence is used to form recommendations based on that evidence. It is a work of polemics. The clinical evidence, already the product of a commercially skewed research environment, is selectively minded to build a case for drugging. It has to be, because even this evidence, viewed dispassionately, makes an overwhelming case for behavioural interventions over drugging.

x) “Consulting” young people

The NICE authors commissioned some work into how young people experience “ADHD” and being on “medication”. This study is attached to the guideline as Appendix 15. [126] One of the authors of this paper was Dr Ilina Singh of The London School of Economics and Political Science. The others were Sinead Keenan, also of The London School of Economics and Political Science and Dr Alex Mears of the Healthcare Commission, a body set up by the Department of Health. Dr Singh is a Wellcome Trust funded ADHD researcher. We have already reviewed one of Dr Singh's studies in Section 3) vi) above. In that paper, published in 2007, Dr Singh declared her opinion that methylphenidate has a “tolerable side-effect profile”. [40] Therefore it must be open to question whether Dr Singh was the best-placed person to “capture the voice of the service user” in relation to their “experience of psycho-stimulant medication” [126]

In her paper for the NICE Guideline Dr Singh investigated how young people “experience” “psycho-stimulant medication”. She did this by holding a focus group of 16 “children” aged 9 to 15 all of who “had all been diagnosed with ADHD and all were taking stimulant medication”. Singh

embarks on a discussion of the existing research literature into what it means to “live with the disorder”. ADHD is a “diagnostic category” into which people may be placed. There is no “disorder”. Statistical correlations based on averages over groups for a widely divergent range of biological factors mostly with very small degrees of probability do not establish a clinical disorder. Since there is no clinical disorder (identifiable biological condition which people actually have) we are already launched into what is, effectively, nonsense. It would be more accurate to say that they are studying the experience of being placed into the “diagnostic category” of ADHD by a psychiatrist, and then drugged. That is the real subject of this study.

The interviews with the focus group of young people are imbued with the core ADHD reification. For example:

> Children were asked to think up and discuss an invention that could help children with ADHD.
>
> The doctor thinks the child has ADHD. Your sports hero/heroine wants to know what kinds of things he/she can do to help the child’s behaviour get better. [126]

Dr Singh is in touch with celebrity culture but still thinks that ADHD is something which “children” “have”.

A big part of the experience of “living with the disorder” for a young person is precisely the experience of “having” a “psychiatric condition”. That is, a condition which does not relate to some biological fact. It concerns their mind and its supposed abnormality. Dr Singh and her colleagues explain that she and her fellow researchers thought that the “issues of stigma, labelling and difference” would be similar for young people with “other conditions”. She was surprised to find that there were quite significant differences on this measure between those with epilepsy and those “with ADHD”. Unlike Dr Singh, a young person who is “diagnosed” as “having ADHD” and is prescribed “medication” for it will be well aware that this is altogether something different than being diagnosed as having epilepsy or asthma and being given medication for those conditions. Their peers will understand this as well. It is strange that the people who create this situation appear not to understand this.

Out of the 16 “child participants” in this study 14 were boys and 2 were girls. This shows the usual and unexplained massive preponderance of boys in ADHD diagnoses. (Dr Singh's previous 2007 study had 20 boys and 3 girls). [40] Once again the massive gender disparity does not appear to cause the researchers any serious problems. It should stop them in their tracks. You cannot seriously claim that ADHD is an objective “disorder” of some kind when faced with the evidence that who gets the “diagnosis” is a matter of gender. The young people were told at the start that

“everyone here wants to hear from you”. Of course this isn't true. Dr Singh (at least) has already decided that methylphenidate has a “tolerable side-effect profile”. [40] Unless someone has an adverse cardiac event in front of her it seems unlikely that this is going to change. The “child participants” were also told that “everyone here is friendly”. One can ask why they needed to be told that? The subjects are asked leading questions. Having been told they “have ADHD” they are then asked questions such as:

> Why do you think you need to be taking tablets for ADHD? [126]

and

> In what ways do you think the tablets have helped you? [126]

The way these questions are framed would make it very difficult for a young person to say “I don't need to be taking them. They have not helped me”. The questions are designed to elicit positive statements about the drugs. The “children” were also asked “So, what is ADHD?”. This is breathtakingly cynical. Dr Singh, at some level, presumably knows what ADHD really is. It is a “diagnostic category” of psychiatry. There is no mystery about that and no lack of clarity in fact about exactly what ADHD is. So why ask them? (In her 2007 study Singh asked young people “Can you point to where the problem is that the tablets are helping?”) [40]

One of the aims of Dr Singh's research was to:

> Elicit ideas from children about resources that could help them have more positive experiences of ADHD diagnosis and medication [126]

Neither the “diagnosis” nor the “medication” were up for grabs. Despite this pre-loading of the “research” the 16 members of the focus group offered some really quite harrowing accounts of what it is like to be on “medication” “for ADHD”:

> A number of participants also talked about not wanting to take medication because they did not like the change it made in them. According to one participant: ‘I don’t like it. I just want to be myself. My Mom makes me take it so I can focus. . . but I just want to be myself’. Other comments included: ‘It just like changes me. . . it makes me awful, like this way. . . It’s like, I don’t like to play that much anymore’ and ‘I don’t take [Ritalin] anymore. I didn’t like how I felt on it. I felt real depressed on it.’ [126]

Nonetheless Dr Singh summarises:

> Children who participated in this study had a generally positive experience of tablets. This does not mean that they liked being on medication; rather that they were willing to put up with the 'annoying' dimensions of taking medication in return for the perceived benefits. [126]

It is not entirely clear what this "generally positive experience" is based on. In the study, Figure 5 "Areas in which tablets help" lists a number of possible benefits. These include; concentration, physical aggression, homework, school-marks, reading, writing, relationship with parents, relationship with teachers, relationship with peers etc. However; it is not entirely clear whether this table reports areas in which the young people actually reported positively or simply those that they were asked about. What is clear is that Singh and her colleagues report:

> The most noticeable impact of tablets in the classroom context was their perceived effect on disruptive behaviour. Many children reported that tablets helped them to be less disruptive in the classroom. [126]

and

> Individually and collectively children associated their tablets primarily with helping to improve their social behaviours, and, consequently, their relationships with peers. [126]

"Social behaviours" appears to be a gloss for aggressive and disruptive behaviours. And, beyond this benefit (which was only reported by some subjects) the study authors admit that they had to prompt the young people to try to get them to make positive statements about how the drugs "help them" at school:

> Disruptiveness was discussed both in terms of verbal disruptiveness ('I'm always talking when I shouldn't be') and physical disruptiveness ('I can't sit still'). Most groups had to be encouraged to identify other ways in which tablets might have an impact on school work and school-related functioning. [126]

It is hardly a consultation if the young people have to be prompted to offer product endorsements. Based on the actual text of the study (Section II Perception of Impacts), the claim about "generally positive experience" appears to be a case of making the most of quite limited endorsements. The list of possible wondrous results (Figure 5) do not appear to have been supported by the study. This is not surprising. Those familiar with the ADHD narrative will recognise that this list of (possible) wondrous results relate to *claims* ADHD drug enthusiasts make about the "tablets"

rather than to actual “benefits” of the tablets. Weiss *et al*. 1975 found that after 5 years the group who had been medicated for 3 to 5 years did not score better on emotional adjustment, delinquency, mother-child relationship and mother's impression of change than the group who had not been medicated at all. [82] The evidence for methylphenidate improving academic scores is tenuous. The NICE authors admit as much:

> Equally, studies have not demonstrated clear effects of stimulants on academic performance or learning (Swanson et al., 1993). [127]

Based on the text of the study in Section II “Perception of Impacts” the positive impacts appear to relate to a reduction in disruptive behaviours and an increased ability to concentrate on class-work. There are some reports which were subject to “debate” about improved scores in school-work. But the young people had to be “encouraged” to make them.

Singh and her colleagues report that:

> A few children had experienced ‘acting like a zombie’ on certain medications and/or at certain dosage levels. [126]

Given that the study was based on just 16 young people “a few” would appear to be quite statistically significant. If a significant percentage of the study reported being turned into a zombie by “medication” is is difficult to see how the data can be reported as describing a “generally positive experience”.

This consultation with young people appears to establish little more than what is already known about stimulant drugs. They can help young people to concentrate more in class and therefore be less “disruptive”. But they come at quite a heavy price in terms of side-effects. Not unusually for an ADHD study the conclusion put on the results is much more positive about the effects of drugging than is actually merited by the material in the study.

Singh and her colleagues report:

> The positive effects of the tablets on behaviour were reported most clearly and consistently by children with aggression problems (see Text box 1). They reported that tablets helped them not to feel ‘angry’, helped to calm them down and to ‘think first’ before acting out. Children felt that these positive effects had an associated positive impact on their ability to make and retain friendships.
> [126]

The MTA study did not show any benefit to “medication” over a behavioural intervention as measured by either parents or teachers for “aggression”. Peter Breggin's analysis of the MTA study showed that peers did not rate the subjects more improved by “medication” than by a behavioural intervention. [128] The strongest area for claims about drug benefit in this consultation exercise then are in an area which could equally well be addressed by a behavioural intervention, according to the “clinical evidence” reviewed and accepted by NICE. This “consultation exercise” with young people appears to be used to produce an endorsement of drugging. But, if all the “evidence” is considered together (as it should be in a Guideline which sets out to conduct a review of all the “evidence”) the conclusion, once again, should be that behavioural interventions are better than drugging.

Not one of the 16 subjects in the NICE consultation exercise with young people had (apparently) experienced a behavioural intervention. There is no mention of parents having attended a parenting programme. However, Dr Singh and her colleagues glibly write:

> All children in the study believed medication to be the most effective available treatment for their ADHD symptoms [126]

This exact same sentence appears in the summary of Dr Singh's paper by the NICE Guide authors. They also comment:

> Interviewees were less likely to identify spontaneously effective formal non-drug interventions for their ADHD behaviours (such as CBT or parent training) [129]

The other “treatments” mentioned by the subjects included dietary interventions such as “IQ vitamins” and sports. Since none of the young people had experienced a formal behavioural intervention let alone one which had included their parents they were not able to form a view about these interventions. To report this “finding” without this clarification is to falsely use the young people to endorse drugs. The admission that the young people in the study did not “spontaneously” “identify” CBT or parent-training appears to be an admission that the young people had not encountered behavioural interventions. But it is not very clear.

Dr Singh and her colleagues explore the question of “stigma”. In Section VI of their paper they describe the considerable problems caused to young people by being “diagnosed” ADHD. For example:

> A majority of children reported being called names and bullied about their ADHD behaviours and/or ADHD diagnosis and need for tablets. [126]

and

> Children reported that the negative assumptions of others about them were especially burdensome. They felt they received negative differential treatment because of their diagnosis. [126]

and

> Children felt exposed by the need to take medication, especially if they needed to take tablets during the school day. [126]

and

> Both girls in the study (in separate groups) reported feeling that teachers ignored them completely because of their ADHD diagnosis. [126]

and

> They felt peers and teachers were ‘unkind’; and they reported experiences of feeling ‘different’ and ‘isolated’. [126]

Dr Singh's attempts to relocate responsibility for the “stigma” away from the “ADHD diagnosis” and onto the “ADHD behaviours” and thus away from psychiatry and onto the young people is unconvincing. If the reader has been in a modern school she will be aware that it is the “diagnosis” that young people try to hide. (As an example; this writer has worked as learning support assistant in a modern comprehensive. His job was to offer extra support in mainstream classes to young people who have been identified as having special needs, for example those “with ADHD”. Many of the young people openly asked him not help them for the simple reason that if he did it meant that their cover was blown. They were “exposed” to their friends as having some kind of official “condition”. Few young people want to be “special” in this sense). Dr Singh writes:

> In general children felt there was a lack of empathy and a lack of understanding about children with ADHD. [126]

and

> One of the most strongly stated, and most resonant, desires communicated by this group of children was for better public understanding of ADHD. [126]

In their summary of this study the NICE authors repeat the claim that the study found that young people are calling for a “better public understanding of ADHD”. [128] It seems likely that this call for a “better public understanding of ADHD” is something which has been injected into the “consultation” exercise by Dr Singh and her colleagues. At least there is no evidence that this was in fact called for by young people whose voices appear to be represented in the reports of feeling “exposed, “isolated”, “different” and “ignored”.

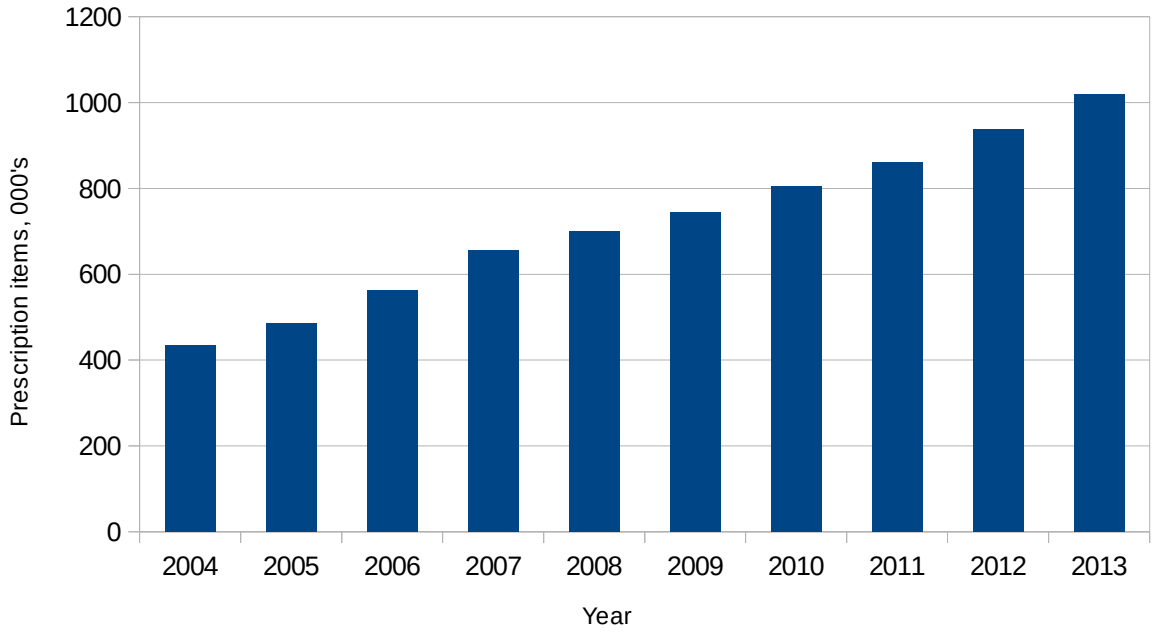
A psychiatric label is a sign not only of difference but of problematic difference. No wonder young people feel isolated by it and try to hide that they have been given this label. It would appear that rather than face up to the very considerable “stigma” caused to young people by ADHD labelling, and drugging, which the study unveils the study authors and the NICE Guide authors have attempted to turn the situation to their advantage. They will use it as a further occasion to promote their product. The ADHD label. In any event; what is this “better public understanding of ADHD” that the NICE authors propose will help address this problem? It can't be an explanation of a biological problem because none such exists. In reality all it could be would be an amplification of the criteria for “getting” the “condition”. Inappropriate behaviour for developmental age. How will that lessen the “stigma”? If anything it will make it worse. In general, attempts to promote the “public understanding of ADHD” mean more diagnoses and more drugging. We are in the realm of product endorsements and sales; not science.

There is no evidence that the “voices” of the 16 “service-users” interviewed by Dr Singh and her colleagues about their experiences of “psycho stimulant medication” including their reports of side-effects and suffering “stigma” have had any effect on the clinical recommendations produced by NICE. Rather it appears to have been an exercise in wringing endorsement of drugging from a group of young people in school. The young people had to be “encouraged” to produce endorsements for drugging beyond the one sure effect of stimulants, that they can improve concentration and help people to sit still and talk less. That “a few”, out of 16, reported being turned into a “zombie” by the product probably won't find its way onto the packaging.

xi) The show must go on

Even within their own terms (ADHD exists, “symptom reduction” is a good in itself etc.). the NICE authors have failed to prove that there is any reason to recommend drugging over behavioural interventions “for ADHD”. Behavioural interventions do not carry appalling side-effects. Unlike “medication” they may have an effect which lasts beyond treatment time. That is they may really help people rather than just suppressing the awkward behaviours. From a “clinical” point of view behavioural interventions are clearly “better”. Nor do they cost the tax-payer any more. However,

the main purpose of the NICE Guideline recommendations on ADHD appears to have been to allow individual psychiatrists and parents to choose at will between the two “treatments”. The drugs have the attraction that they make “disruptive” young people “easier to handle” and “more compliant” just by popping a pill into their mouths. The financial cost for this convenience is largely borne by the tax-payer. In England alone this was around £45 million in 2013. As an estimate perhaps around 131,508 young people were drugged in 2013 “for ADHD” in England alone. [62] The NICE ADHD Guideline was published in 2009 and appears to have done nothing to slow the steady year on year growth in the market for “ADHD drugs” in England as this chart shows:




(This chart uses the data we have already presented in Section 3) iv) from the NHS Information Centre)).

## 5) Discussion

i) Manipulative solutions

The genetic study which we reviewed in Section 1) was funded by Action Medical Research (a charity), Baily Thomas Charitable Trust, the Wellcome Trust, the UK Medical Research Council and the European Union. The authors of the study state, formally: "We declare that we have no conflicts of interest". We know what they mean perhaps; none of them is a Trustee of one of the funding charities for example. While routine it is a meaningless claim which obscures the real webs of interest.

A charity is simply an organisation that does not make and distribute profits when it conducts its business. It must also meet the criteria for charitable purposes. In England and Wales charitable purposes include “the advancement of health or the saving of lives”. [1] A charity may be completely linked to the corporate and government world, sharing its free-market aims and values. The legal status of charity does not in fact ensure charity. The Charity Commission is the body which regulates charities in England and Wales. Research into “ADHD” is carried out by prestigious medical schools and funded by prestigious charities such as The Wellcome Trust. It would require more bravery than perhaps the Charity Commission is known for were it to question whether proving that 14% of an ADHD group possess a certain genetic variant as compared to 7% in the general population is to do with “the advancement of health”.

The fact that a group of active medical research charities have combined to pay for a research project may have the effect of sanctioning the medical research status of that project to a greater degree than the content of the research. The fact that NICE has sponsored a report on ADHD is perhaps more determinant of how that report is received than the quality of the research and reasoning in that report. For example, when the Panorama programme critical of some aspects of ADHD drugging was broadcast, (see sub-section iii) following), the Department of Health was able to issue a reassuring statement explaining that:

> The Department of Health has supported a number of initiatives taken by professional bodies and voluntary groups to raise awareness of ADHD and ensure accurate diagnosis and appropriate treatment.

and

> We have also asked NICE to develop a clinical guideline on both the pharmacological and psychological interventions to treat ADHD. The guideline will cover the care provided by primary, community and secondary healthcare professionals who have direct contact with, and make decisions concerning, the care of children, young people

> and adults with ADHD. [2]

NICE in turn commissioned The Royal College of Psychiatrists and The British Psychological Society to develop the Guideline. It doesn't matter that the piece of work produced is not “evidence-based” as it claims to be. The key point is that a document has been produced and government can point to that. The boxes have been ticked. No one is expected to look inside the boxes.

In *Deschooling Society* [3] the writer Ivan Illich proposed a way of looking at institutions which considers the way they organise their relations with their members/consumers/subscribers as their defining characteristic. He proposed a spectrum from “left” to “right”. Illich uses the traditional terms “left” and “right” though he uses them in a new way. On the “right” of this spectrum are institutions which are manipulative and hierarchical. Behaviour of members is controlled by rules. If a right-wing institution is a service institution it will aim to develop addiction in its clients and make them dependent. Right-wing institutions tend to escalate participation. They try to make people believe that they cannot live without the service or product provided. Illich writes:

> Right-wing institutions tend to be highly complex and costly production processes in which much of this elaboration and expense is concerned with convincing consumers that they cannot live without the product or treatment offered by the institution. [3]

The considerable research efforts which are undertaken to promote the “benefits” of ADHD drugging are an example of this kind of process.

On the “left” are institutions which are convivial. Left-wing institutions are governed by rules too, but these are a minimal set of rules whose purpose is to ensure the continued functioning of the institution, to the benefit of all. If a left-wing institution is a service institution its clients remain free agents as they consume its service. Once the service or product has been used that is it. There is no in-built tendency to get the client to use more. Illich gives the FBI, the modern prison system and the military as examples of right-wing institutions. As examples of left-wing institutions he gives public parks or public markets.

Schools are on the far right of the institutional spectrum. Consumption of educational packages leads to the consumption of more educational packages until we reach the absurd point that education becomes a purpose in itself. Schools take over the natural desire to learn and grow. They teach that learning and growth is only possible through curricular teaching. They inculcate dependency. As Illich says:

> By making men abdicate the responsibility for their own growth, school leads many to a kind of spiritual suicide. [3]

The education system treats its subject population as consumers. Consumers of educational packages created elsewhere and passed down to the actual consumers through several tiers of a hierarchical system. At the point of delivery consumption is entirely non-negotiable. If some small percentage of the school population choke at the point of consumption the *only* solution that this kind of hierarchical and manipulative thinking can consider is one which attempts to solve the problem by coercion. Basically, if they won't eat their dinner they will be force-fed their dinner.

It is inevitable that the government aligns itself with the efforts of the education system. Mass public education is one of the key “services” delivered by government. Compulsory one size fits all mass-schooling was an innovation of industrial revolution. [4] Mass-schooling is linked to the way the economy is structured. You cannot reform or change the one without questioning the other. Modern governments have no interest in bringing into question the mass consumption and mass production industrial-technological nature of the economies they manage. Equally, government is concerned with the maintenance of the existing social order. Thus it is also likely that government will align itself with institutions which act to preserve social order. Psychiatry is one such institution. Government is likely therefore to be a promoter and a defender of both compulsory mass schooling and psychiatry. These are the two social institutions without which not there would be no ADHD.

Pharmaceutical companies are at the right of Illich's institutional spectrum. It often seems that pharmaceutical companies are on a mission to make the entire population dependent on drugs. If this is the case, they are doing well. According to a recent survey 50% of women and 43% of men in England regularly take prescription drugs. [5] According to the same survey 20% of women in economically deprived areas are using anti-depressants. It is difficult to argue that this is a natural state of affairs; unless one proposes that 20% of the population in poorer areas are born with a need to take powerful anti-depressants to cope with life. The NHS predictably tried to manage the media reporting of these ghastly figures. Their chief line was that the elderly population taking non-addictive self-sustaining drugs skewed the figures. [5] However, a review of the actual data shows that this is a cover-up. [6] There is a high-level of use of prescription drugs for all ages. For example; of women aged 16-34 it appears that 28% are taking one prescription medicine a week. For men in the 16-24 age range the figure is 12% and in the age range 25-34 it appears to be 16%. Again; these figures are too high to be explained by the treatment of illnesses unless we assume that the human race is a very sickly creation indeed. “Right-wing” pharmaceutical companies are doing well at creating addiction and escalating participation.

In terms of Illich's institutional spectrum the family in which a behaviour problem is resolved by popping a pill into the mouth of the “naughty” child could be seen as a right-wing family. This is an

attempt to alter the behaviour of the child by altering his brain chemistry. It is a hierarchical and manipulative solution. In contrast, if the problem is addressed by both the young person undertaking a behaviour programme and the parents attending a programme, this shows at least a potential commitment on the part of all members of the family to work together to address the problem. (A truly convivial solution would probably dispense with the behaviour programme and just work to meet the young person's needs in a normal way). In the MTA study the group of subjects where a behaviour programme was followed achieved comparable results (symptom scoring system) to those receiving typical outpatient care which in 67.4% of cases included "medication". [7] That is; if we were to accept the MTA study as a guide then it tells us that a change from the current treatment model which is heavily weighted towards "medication" to one which mandated only behavioural programmes would be no less effective at "managing ADHD symptoms". The support given by the NICE Guideline authors (see Section 4)) for drugging can be explained as a built-in "right-wing" preference for drugging and for manipulative solutions. This was also evidenced in their review of educational interventions. Again; the emphasis was on modifying the young person to fit into the existing class-room and curriculum set-up. In the papers NICE selected to review educational interventions there was very little sign of any thought being given to the possibility of modifying the educational system to fit the needs of the young person. Evidence is present in material reviewed by NICE that one way of helping young people with attention problems is to help them become more motivated. (See Section 3) iii)). But this way of helping people, which emerges as a study result, (and which still exists within the field in which "ADHD" is seen as a "disorder" of some kind) is not followed up. The only solutions which can be contemplated are ones of the style which Illich would characterise as "right-wing". Dominant, hierarchical, manipulative.

The ADHD drugging programme essentially involves a collusion between multiple right-wing social institutions. Pharmaceutical companies, the modern education system, psychiatry and some "right-wing" style families. Their practices are superintended by a government which essentially acts not in a liberating way but in an oppressive way towards its own population. Beneath all this manipulative weight the young person "with ADHD" stands no chance.

ii) The Welcome Trust

The Wellcome Trust was one of the sponsors of the genome-wide association study which we reviewed in Section 1).

The Wellcome Trust was founded in 1936 by a legacy in the will of a US pharmaceutical magnate, Henry Wellcome. It is thus hardly surprising that its world-view is one which sees pharmaceuticals in medicine as desirable, as indeed, some at least, incontestably are. The Wellcome Trust is a major sponsor of medical research in the United Kingdom. One of the departments involved in the

genetic study we reviewed in Section 1) is The Department for Psychological Medicine and Neurology, Cardiff University. This department is located in a building which is called after the pharmaceutical magnate who founded the Wellcome Trust. This is The Henry Wellcome building. This is obviously co-incidental but it does help to illustrate that there is a related web of interests between pharmaceutical business, funding charities and academia. The Wellcome Trust with its origins in and most likely therefore its allegiance to US pharmaceutical corporations is at the heart of academic medical research in the UK.

Statistical studies and especially genome-wide association studies around ADHD often appear to be no more than a game of proving the "odd man out". While they do not provide a scientific basis for a medical treatment they may nonetheless serve to make drugging more acceptable. They do this by fuelling the biological narrative about ADHD. The biological narrative in turn implicitly supports "medication". This is because if ADHD is conceived as a "biological deficiency" it becomes easy to propose that "medication" is the fix. In Section 1) ii) we discussed the false claims made by one of the Cardiff genome study authors about the Cardiff genome study. As we saw, Professor Thapar appears to claim that all "children with ADHD" have a biological brain disorder:

> Now we can say with confidence that ADHD is a genetic disease and that the brains of children with this condition develop differently to those of other children [8]

And from this it follows that:

> Most importantly, the results can help us understand the causes and biology of ADHD, which can suggest how it might be treated. At the moment, we only have a limited range of treatments available; but if we can understand what is happening in the brain during the development of ADHD, we might be able to develop more precisely tailored, more effective treatments. [9]

It seems likely that these "more effective treatments" will be pharmacological. But the Cardiff genome study did not establish that "ADHD is a genetic disease". Nor did it establish that "the brains of children with this condition develop differently to those of other children". It established a statistical correlation between possession of an ADHD label and a particular genetic factor; 14% of the ADHD group had the identified genetic factor compared to 7% of the general population. The study could not establish causality. (See Section 1) ii)). Nonetheless and the actual results of her study notwithstanding Professor Thapar used the study to elaborate a biological narrative for ADHD. She promoted this narrative to the press. The Wellcome Trust part funded this study and is today amplifying the false claims made by one of its authors, to the world. Claims which at least implicitly support notions of pharmaceutical "treatments" "for ADHD".

In one of the Wellcome Trust articles cited above we can see both stories being told. In the introduction to the story the reader is told:

> It has recently been discovered that children with ADHD are more likely to have pieces of their DNA duplicated or missing than other children. [9]

This is correct. However, some lines later the fact that this was a statistical finding relating to only a relatively small percentage of the group is forgotten. The reader is told:

> Findings in a paper published in the 'Lancet' in September 2010 confirmed that there are differences in certain parts of the genome between children with and without ADHD. [9]

This statement is not true. This elision, from statistical correlations, which do exist, to a supposed clinical condition which effects all young people “with ADHD” is at the heart of how the ADHD myth is built. (It cannot be argued that this is simply a badly explained report of a statistical correlation. Any member of the public reading the above would understand that this is saying that *all* young people “with ADHD” have the genetic difference).

Someone is so keen that these false claims and the biological narrative they support should be transmitted to the public that they have been advertising them on the Google internet search engine. In April 2014 an advert was running which stated “ADHD: a genetic disorder. New research finds direct evidence that ADHD is a genetic disorder”. [10] The advert linked to the Wellcome Trust press pages about the Cardiff genome study. (Since this was three and a half years after the publication of the study it can hardly honestly be described as new; but truthfulness is not in high evidence here). The claim in the advert uses the language which the Wellcome Trust initially put on their press release about this study; “Study finds first direct evidence that ADHD is a genetic disorder". The title of the press release was subsequently changed to the more defendable: “Study finds first direct genetic link to ADHD". (See Section 1) ii) for a discussion of this alteration to the title of the press release). Who was running this advert with its false claims about the Cardiff genome study?

One of the major research areas in which The Wellcome Trust awards grants is “Society and Ethics”. The Wellcome Trust has funded work on the “ethics” of “ADHD”. Dr Ilina Singh was awarded funding in this area in 2006. [11] We have already met Dr Singh twice (Section 3) vi) and Section 4) x)). In her 2007 study into Ethical concepts and young people taking methylphenidate

for ADHD Singh believed that she had shown “empirically” that “children with ADHD” have “moral conceptions” of themselves in which they see themselves as being persistently “bad”. These apparent self-perceptions of “badness” constitute something called their “authentic selves”. Because their “authentic selves” are “bad” there is no need to give them a break from the drug at week-ends. (As is apparently common in the US). Dr Singh also contributed a paper for the NICE ADHD Guide about “Young Peoples' Experience of Psycho-stimulant Medication”. This was based on a focus-group in which “Children discussed a range of ways in which their tablets helped them”. As usual the “children” accepted that they “have” ADHD (that is what they've been told). And they are allowed to voice an approved objection to the “side-effects” of the drugs: “In the context of this generally positive attitude, more negative reactions to medication were also frequently expressed.” [12] (See Section 4) x)). But, overall, despite the “negative reactions” to “medication” being “frequently expressed” in the end the “experience of tablets” was “generally positive”:

> Children who participated in this study had a generally positive experience of tablets. This does not mean that they liked being on medication; rather that they were willing to put up with the ‘annoying’ dimensions of taking medication in return for the perceived benefits. [12]

No one can say that these academics are failing to deliver the results which their sponsor, The Wellcome Trust, with its links to the US pharmaceutical industry, is likely to be pleased by. Another paper from Dr Singh is: “Beyond polemics: science and ethics of ADHD”. This paper was published in December 2008. [13]  In this paper, we are told, Dr Singh will “explore the current state of scientific research into ADHD and the key social and ethical concerns that are emerging from the sharp rise in the number of diagnoses and the use of stimulant drug treatments in children.” This, unfortunately, is an unpromising start. Since “ADHD” is a “diagnostic category” of psychiatry which does not “imply a medical or neurological cause” there can't really be any “scientific research” into ADHD. There is nothing to research. What there are are endless studies which manage to show statistically significant differences on a given variable between an ADHD group and a “normal” group. These studies are essentially promotional efforts whose purpose is to promote the “validity” of the “disorder”. They are not investigations of a disease. We could add that since Dr Singh has already decided that stimulants have a “tolerable side-effect profile” [14] it is arguable whether she is the best placed person to discuss the “ethical concerns” arising from the use of “stimulant drug treatments” “in children”.

In this paper, Dr Singh outlines what she sees as the three main “positions” in the “debate” about ADHD. These are a view that “ADHD is primarily caused by a combination of biological factors”, a view that “ADHD is caused by a combination of biological and social factors” and a view that “ADHD is a valid disorder but its primary causes are environmental”. We are informed that there is also a fourth position “which is sceptical that ADHD is a real disorder” and that “This position is

sometimes identified with scientologists, but it is also represented by a separate, and more thoughtful, sociological critique." [15] Dr Singh gives a single reference to a representation of this more "thoughtful, sociological critique", a paper in the Journal of Medical Humanities. [16] At least it wasn't from the scientologists. All of this discussion about possible "causes" is nonsense. There is no identified biological disease about which one could even begin to discuss the "causes". The single "cause" of ADHD in any young person is the act of "diagnosis".

Dr Singh informs us that:

> This divisive debate [about the "validity" of the "ADHD diagnosis"] no longer accurately reflects the state of scientific understanding of ADHD, which highlights the complexity and heterogeneity of the disorder. [15]

The "highlighting of the complexity and heterogeneity of the disorder" is about re-engineering the narrative. It does not arise from developments in "scientific understanding". The NICE Guideline authors adopt this new version of the narrative:

> The aetiology of ADHD involves the interplay of multiple genetic and environmental factors. ADHD is viewed as a heterogeneous disorder with different sub-types resulting from different combinations of risk factors acting together. [17]

Since "the interplay of multiple genetic and environmental factors" provides an explaining theory of all human behaviour it follows that this position cannot be disproved. ADHD promoters have simply adopted an explaining theory which is so all encompassing that it cannot be disputed. This secures the "validity" of the diagnosis for ever. Dr Singh is in line with recent developments in the narrative. Again; this has no connection with advances in "scientific understanding".

The "divisive debate" referred to by Dr Singh is an artificial "debate". The public has no say in whether "ADHD is valid". It is. It is the property of psychiatry and they can determine it is valid if they so wish. The more serious questions concern the role of psychiatry in society. How is it allowed to produce these systems of diagnosis? What role do they play in managing deviance? What is the relationship between psychiatry and its "diagnostic categories" and the pharmaceutical industry? To what extent is deviance in contemporary society being managed with drugs? The "is ADHD real" debate is something of a smokescreen which protects psychiatry from these kinds of more challenging questions.

In the passage quoted above the NICE authors develop a theory concerning the "aetiology" of ADHD. However; it cannot have an "aetiology". There is, by their own admission "no medical or

neurological cause” nor “biological marker”. Without a biological fact there can be no discussion off aetiology. Leaving that problematic aside, the theory is quite generous as to the state of the genetic correlations to the label which have so far been established by genome-wide association studies. To her credit Dr Singh accepts this:

> Genome-wide association studies have been largely inconclusive, although one study has found weak associations between variants of the dopamine transporter (DAT) and the dopamine receptor DRD4 and ADHD. [18]

The same applies to the MRI scan research:

> At present, however, both structural and functional neuroimaging data on ADHD are inconclusive, owing in part to the use of different imaging technologies across studies and to a lack of adolescent and adult data. [19]

Dr Singh's paper pre-dated the genetic study we reviewed in Section 1).

But despite these observations about the paucity of the neurological and genetic “evidence” “for ADHD” Dr Singh does not question the matter deeply enough. If the genetic case (for a correlation between possession of the label and possession of identifiable genetic factors) and the MRI scan material is “inconclusive” why is the biological strand within the ADHD narrative so strong? This problematic of the excessive prominence of the “genetic neurodevelopmental disorder” narrative strand within ADHD was addressed by the noted critic of ADHD excesses, Dr Sami Timimi in a contribution to the NICE Guideline “consensus conference”. This conference was held by the NICE authors “to debate the key issues of the use of ADHD as a diagnostic category.” [20] Dr Timimi explains:

> The main problem with current theory and practice in ADHD is the prevalence of the underlying assumption that ADHD is a genetic neurodevelopmental disorder and that clinicians have valid and reliable ways of identifying what behaviours are the result of such neurodevelopmental disabilities in any individual child. [21]

and

> Current evidence does not support a simplistic view of ADHD type behaviours. Genetic studies have relied on poor standards of evidence (such as the disputed ‘equal environment’ assumption), and have failed to replicate genetic associations consistently, thus the null hypothesis stands – no genes exist for ADHD. Similarly, neuroimaging studies suffer from serious methodological failings and interpretive

> inadequacies; thus there are currently no neurological markers for ADHD (nor are there likely to be). [21]

(Again, Dr Timimi seems to have been writing before the genome study we reviewed in Section 1) identified a small correlation between possession of an ADHD label and a specific genetic factor). Dr Timimi associates the dominance of the genetic neurodevelopmental disorder narrative strand with “biological remedies”:

> The most important implication of the dominance of biological theory in ADHD is that it has led to a rapid rise in the use of biological remedies as the first-line and often only treatment for those diagnosed with ADHD. [21]

For reasons of space or otherwise Dr Timimi does not explore how the biological narrative comes to the fore in the wider ADHD narrative in a way which is out of all proportion to the actual evidence for it. What forces or collations of forces are behind this? At least one beneficiary of the “rapid rise in the use of biological remedies” attendant on this (largely) un-evidenced theory though is obvious. The market for ADHD drugs in England alone has gone from £14 million in 2004 to £45 million in 2013. (Section 3) iv)).

We could add that even if the statistical correlations found in genome association studies and MRI scan studies were stronger this would still not establish ADHD as a clinical condition.

We have seen how the NICE authors attempt to slither off the fact that there is no biological test “for ADHD” with a series of linguistic manipulations which are characteristic of the ADHD discourse. They explain that there is no “gold standard”, [22] “no specific biological test”, [23] and “no definitive biological test”, [24]. In fact it is not that there are no “specific tests”. There are no tests. Not one young person “diagnosed” “with ADHD” has had anything even vaguely resembling a biological test for their “ADHD”. No blood test. No test-tubes. No swabs. Nothing. “Diagnosis” is by a psychiatrist (or paediatrician) based on a behaviour tick-box system which is based in turn on rules determined by psychiatry. Reports from parents and teachers can also help secure the “diagnosis”. Dr Singh is aware of the problematics of this:

> There are no laboratory tests to determine unequivocally whether a subject has the disorder. [19]

But Dr Singh also tries to fudge the absence of a test. In reality; no laboratory tests. Not even equivocal ones.

Dr Singh notes that there has been a steady rise in the rates of prescription for methylphenidate in recent years. She includes a table which appears to show that there has been a nearly 800% rise in methylphenidate consumption in the UK from 1999 to 2003. [18] She comments on how the increases in diagnoses “of ADHD” are linked to this rise in the sales of drugs:

> ....in the past decade rates of diagnosis have increased sharply in most countries around the world. These increases are linked to parallel growth in the consumption of stimulant medications. [15]

Dr Singh suggests that the rise in rates of “diagnosis” may be due to either “over-diagnosis” or “an actual increase in ADHD prevalence”. She suggests that “a better scientific understanding of the aetiology of ADHD” “might” clarify this question. This is implausible, almost preposterous. If we accept that the growth in drugging and “diagnosis” is “parallel” as Dr Singh reports then we have to explain a nearly eightfold increase in rates of “diagnosis” between 1999 and 2003 (assuming that dosage levels remained the same). An increase of this order in “rates of prevalence” cannot plausibly be explained by any of the three main “positions on ADHD” which Dr Singh enumerates. The genetics of a population do not change over this kind of time period. “Social” and environmental factors do change more rapidly but it is not possible to realistically propose that such factors could have changed enough in a single four year period to cause an eightfold increase in the “prevalence” of “ADHD”. We do not need to wait for “scientific understanding” to clarify this. Dr Singh does not discuss the most obvious reading. This is a typical case of market penetration for a new product. This is a sales curve. It needs an economist to interpret it; not a “scientist”. Dr Singh moots the possibility of “over-diagnosis” but she avoids making the connection between “over-diagnosis” and the marketing aims of pharmaceutical companies. But; what other explanation is possible?

Dr Singh quotes the claims-making of the unscientific MTA study as a matter of established fact:

> These drugs have been shown to be more effective at treating ADHD symptoms than behavioural therapy alone, and also more effective than behavioural therapy combined with drug treatment. [19]

Dr Singh's reference is to a paper by one of the MTA study authors [25], a well-known proponent of ADHD-drugging [26], in a separate paper in which he cites the MTA study. Apart from any other consideration the MTA study compared (“symptom reduction” scoring system) a highly unusual and heavily optimised “medication” regime with one specific behavioural intervention (at which attendance in one component was just 77%). It is not possible to extrapolate from these unique

conditions to make general statements about “these drugs being more effective than behaviour therapy”. In normal out-patient contexts drug doses will be significantly lower than in the MTA study “medication” group. (In the MTA “medication” group methylphenidate dosage was 37.7 mg per day. In the community care group, which is likely to reflect typical out-patient care, it was 22.6 mg for those being drugged). While it is widely reported that the MTA study showed that “medication” (its heavily optimised programme) was “superior” to its behavioural intervention less well-reported are the facts that this result was only confirmed by both parent and teacher raters for inattention. Only one of these groups (probably parents) confirmed the “superiority” of “medication” for hyperactivity. The neutral classroom observers did not report “medication” outperformed the behavioural intervention at all. These are very slender results on the basis of which to be proclaiming that “These drugs have been shown to be more effective at treating ADHD symptoms than behavioural therapy alone” as Dr Singh does. (See Section 2) for a full criticism of the MTA study).

Dr Singh concedes that stimulant drugs do improve performance in “'normal' children”:

> In the 1970s, researchers showed that a positive response to stimulants is not limited to children with ADHD: ‘normal’ children show improvements in attention and focus as well. [19]

Dr Singh even appears to realise the implications of this:

> Therefore, to some degree, the medications enhance performance rather than treating the specific psychopathology. [19]

This fact poses a serious problem for the ADHD narrative. If ADHD drugging is about “enhancing performance” the whole “treatment for a disorder” narrative unravels. Dr Singh, possibly to her credit, sails into the uncharted territory that this admission effectively opens up:

> However, cognitive enhancement in children must be acknowledged as a growing social practice that currently lacks regulation. This increases the potential for physical and ethical harms to children. One possible solution is that cognitive enhancement in children be introduced as part of clinical services, with appropriate boundaries and safeguards in place. [27]

The proposal that young people should be drugged in clinics in order to enhance performance does not seem to sit well with the government's general attitude towards young people and drugs.

Methylphenidate is, according to the US Drug Enforcement Agency, “structurally and pharmacologically similar to amphetamines and cocaine and has the same dependency profile of cocaine and other stimulants”. [28] So; the harms associated with taking these kinds of drugs will apply to young people taking methylphenidate to enhance performance in one of Dr Singh's clinics.

This really is a Brave New World of state prescribed drugs for no medical reason. There are various rational and evidential flaws here too. If drugging young people for “cognitive enhancement” may cause them “physical harm” how, exactly, will this be resolved with “appropriate boundaries and safeguards”? How much “physical harm” would be ethically acceptable? Dr Singh makes a claim about how stimulants “improve academic performance”. This claim depends on a reference to a single paper which in Dr Singh's words showed that stimulants can have a “short-term positive effect on academic performance”. [27] The referenced study [29] is not credible even in the usual terms of ADHD drug studies. There was no properly constructed control group. A total sample size of 31 was measured for IQ. After a year 24 subjects were found to be being drugged and 7 not. The IQ tests were repeated. Those in the drugged group showed improvements in the IQ test but not the 7 who were not being drugged. The measure thus appears to have been an IQ test not “academic performance”. The result may reflect no more than stimulants can improve concentration and thus short-term test performance. There is no wondrous improvement in academic performance, let alone ability. The claim for improved academic performance is a familiar one in the ADHD narrative. However; the evidence in tenuous. The MTA study found that their heavily optimised “medication” programme if combined with the behavioural programme produced a better score on the reading test than the behavioural programme alone. But this result was not produced for spelling and maths. (Breggin refers to a scholarly assessment which claims that the reading result was the result of an error in handing the statistical methods [30]). The authors of the NICE Guideline seem to be generally unconvinced about the claims that stimulants improve academic performance:

> Equally, studies have not demonstrated clear effects of stimulants on academic performance or learning (Swanson et al., 1993). [31]

Even if the affect of improving concentration can produce slightly higher scores in tests this is not a long-term benefit. It remains the case, for ADHD labelled young people, that drugging does not improve long-term outcomes:

> There is little evidence that stimulant medication alters the relatively poor long-term outcome for many of those with ADHD (Weiss & Hechtman, 1993). [32]

The improvement in “academic performance” is over-hyped.

In considering drugging young people for performance benefits Dr Singh has really gone out on a limb. The reason some students take stimulants to help them in exams is because the improved concentration may help them get a better score on the day. It doesn't improve their long-term academic ability. It just helps them get a higher score on the test. They are cheating. Is this what Dr Singh is recommending? Another consequence of such an open programme of drugging to improve school performance would be to make it almost inevitable that nearly all young people would drugged. Those on drugs would have a perceived advantage and others would be drugged so they were not at a disadvantage. The world of organised sport has already ruled against the use of drugs to enhance performance. One can see why. The idea of drugging young people in special clinics for purposes of “cognitive enhancement” is not just bizarre it is irrational.

But Dr Singh's irrationality is a result of her frank admission that the point about stimulant drugs is that they are “improving performance” - not treating a disorder. Dr Singh in her rather naive way has simply brought to the fore an intrinsic implication of the ADHD drugging programme. The problem is that drugging young people with stimulants to increase attentiveness just looks very bizarre when presented outside of the “clinical disorder” garb it is usually cloaked in. No wonder the NICE authors caution that stimulants should not be prescribed just to improve academic performance. [33] They are aware of where this leads.

In the section of her paper entitled “Ethical aspects of medicating children” Dr Singh focuses on topics such “side-effects”, and possible “moral harms”. As concerns these possible “moral harms” Dr Singh references her own 2007 work on “Moral self-understandings of children taking methylphenidate for ADHD”. She says that this work has shown that:

> ...stimulant drug use has been shown to affect children’s concepts of identity and personal authenticity, but the available evidence suggests that these effects are largely positive for most children. [27]

Strangely, we can note that this was not what her own 2007 study said. Here she said:

> Second, children’s moral conceptions of their authentic selves are characterized by persistent badness, despite medication. [14]

In the 2007 paper the argument was that methylphenidate does not make them morally bad. They are already. This “well; it doesn't do any moral harm” argument appears to have enjoyed a post-hoc elaboration into “largely positive effects” on “concepts of personal authenticity”. To be fair to Dr Singh, in the 2007 study the story about how “medication” helps “children be good” was never far below the surface. Some of the interviews with young people are used to show how

methylphenidate can help them have “moral” reflections on their “bad behaviour”. It was just that at that time she appears to have chosen the “bad in their core despite medication” position for her conclusion. At any event this is not looking quite like the “empirical” research promised in the 2007 paper. And this is no surprise; there cannot be an “empirical” approach to this kind of social science material. (See Section 3) vi) for a discussion of Singh 2007 and the problems with her “empirical” approach). At any event even if being on methylphenidate can improve a young person's concentration to the extent that they listen to and accept the “moral” messages about their behaviour given to them by their parents this does not mean that methylphenidate has had a positive effect on their sense of “personal authenticity”. The following indicates what some of the young people consulted by Singh have said about methylphenidate and what might be called their “authentic selves”:

> Mark: Well of course I’m not real with the tablets! [14]

and

> A few children had experienced ‘acting like a zombie’ on certain medications and/or at certain dosage levels. [12]

Dr Singh also reports that her previous work has shown that “children with ADHD”:

> successfully negotiate the stigma around drug treatment. [27]

One of the references she gives for this claim is to her paper (along with two other authors) on “Young people's experience of psycho-stimulant medication” which was included in the NICE ADHD Guide as Appendix 15. [12] But in this paper she said:

> Children felt exposed by the need to take medication, especially if they needed to take tablets during the school day. The need to take tablets made them ‘feel different’ in a negative way. [12]

This does not sound like the “children” were “successfully negotiating” “the stigma around drug treatment”. It sounds like the opposite.

In her 2008 paper on “Beyond polemics: science and ethics of ADHD” Dr Singh states that her earlier work shows that:

> children with ADHD express desire for psychotropic drugs [27]

In her 2007 study the “children” were told that:

> All the children in these pictures have ADHD, like you, and they take Ritalin tablets to help them. [14]

It would be difficult in these circumstances for a young person aged 8-12 to not “express desire for psychotropic drugs”. But the “desire” for psychotropic drugs may be genuine. Stimulants are pleasurable. They are also addictive. Of course the young people “desire” them. It is just this “desire” for stimulant drugs that the government has to battle against when it tries to advise young people not to take stimulants. Is the fact that young people who are given stimulants “express a desire” for them again really an endorsement of drugging?

Dr Singh suggests a number of ways that social scientists could contribute to standardising the “diagnosis” of ADHD. For example such work might illuminate “the problem of inconsistency in ADHD diagnoses within particular populations”. [34] This is, within the frame in which it is conducted, (acceptance of the reification that the ADHD label refers to something which exists), worthwhile work. However, the value of this challenge is immediately thrown away when she suggests that the kind of collaboration between social scientists and scientists required to define “diagnostic standards” in a more reliable way may be carried out by “authoritative groups” such as NICE. The authors of the NICE ADHD Guideline called a Consensus Conference “in order to debate the key issues of the use of ADHD as a diagnostic category”. [20] Dr Singh contributed to this conference and high-lighted the “socio-cultural” factors in ADHD diagnosis and treatment. However in their preamble to discussing the fruit of this conference the NICE authors write:

> While it is important to acknowledge the validity of the social scientific paradigm and its body of literature, in the context of the development of practical clinical guidelines, it is not possible to offer alternative processes for clinical assessment or treatment. [20]

This precise sentence exists with exactly the same wording in the draft of the NICE Guidelines to which Dr Singh refers when she suggests that authoritative bodies such as NICE collaborate more with social scientists such as herself. [35] NICE has clearly set its face unambiguously against just such an endeavour as she proposes. This appears to leave Dr Singh's project to include more “social science” work in the ADHD programme as being in need of a new direction.

Dr Singh's article is naive with regards to fiscal and power interests. The “sceptical” position has been left safely represented by scientologists and the single cited article in the Journal of Medical Humanities. Dr Singh acknowledges some of the sore points in the narrative but consistently pulls her punches.

Dr Singh work appears to have a specific function within the ADHD narrative. She engages with or at least claims to engage with the “ethical aspects of medicating children”. She “empirically” addresses the argument that “medication” damages the “natural self” of “children”. (See Section 3) vi)). She holds a focus group which allows the authors of the NICE ADHD Guideline to claim that young people have been consulted. (Section 4) x)). She seems to argue for more involvement of “social science” in the “diagnosis”. All this allows what Dr Singh might call the “science-side” of the “debate” to claim that these issues have been addressed. However; the project lacks a critical dimension. All the usual reifying and mystifying language is used. “Children” have “symptoms” of ADHD which, based on the MTA study, are more “effectively” treated by “psycho-stimulant medication” than by behavioural interventions (which she doesn't seem to be very interested in). But there is no critical discussion of what is meant by “ADHD symptoms”. The consultations with young people which seem to be central to the project are pre-loaded by the “children” being told that they “have ADHD” and the tablets “help them”. Dr Singh has decided, and this can hardly be called “empirical”, that methylphenidate has a “tolerable side-effect profile”. The end result is that the ADHD narrative is strengthened.

The Wellcome Trust funds Dr Singh. They funded the genome-wide association study which we reviewed in Section 1). The Wellcome Trust funds research at both ends of the spectrum. But whether they fund research into genetics or research into “ethics” the research always seems to help develop a narrative which promotes the use of drugs.

iii) Silencing Panorama

In 2007 the BBC's Panorama programme covered the follow-up study to the MTA study which we have discussed above, (Section 2) vii)). The follow-up study failed to confirm the “superiority” of “medication” over a behavioural intervention which had been partly established by the original MTA study, using the symptom scoring system. Dr William Pelham, one of the original MTA study researchers, was also involved in the follow-up study. Dr Pelham was a key source for the program. He had said:

> I think that we exaggerated the beneficial impact of medication in the first study. We had thought that children medicated longer would have better outcomes. That didn't happen to be the case.

> The children had a substantial decrease in their rate of growth so they weren't growing as much as other kids both in terms of their height and in terms of their weight. And the second was that there were no beneficial effects - none. In the short run medication will help the child behave better, in the long run it won't. And that information should be made very clear to parents. [36]

These comments were on the BBC's Panorama website but were removed as part of the rulings which we discuss below. [39] At the time of writing they appear in full on another page on the BBC web site. [40]  This report additionally reports Dr Pelham as saying:

> There's no indication that medication's better than nothing in the long run [40]

A cached copy of the original Panorama page is still available on the Wayback Machine. [41]

The Panorama programme gave wide publicity to mainstream research which failed to demonstrate (using the “symptom reduction” scheme) a long-term “benefit” to drugging over behavioural training. This was potentially very damaging for the ADHD drugging story. Panorama averages more than 3 million viewers per episode. [42]

An unnamed individual or organisation made an enduring and persistent complaint to the BBC about the Panorama programme. The complaint was handled by two separate BBC committees.

Initially, the BBC Editorial Complaints Unit (ECU) made a ruling against the programme. This ruling stated:

> However, the research was not conducted in such a way as to be able to determine that long-term medication conferred no benefit at all, and to that extent the impression given by the programme was misleading. [43]

The complaint to the Editorial Complaints Unit was that Panorama had reported that it had been found that “medication” showed no benefits in the long-run when in fact the follow-up research had showed that “medication” had no benefits *as compared to behavioural treatments* in the long-run. This was probably a valid comment on the Panorama programme. However; this rendering was probably a journalistic over-simplification. Simplifying stories is something which the BBC like other news outlets does frequently in order to communicate a complex story to a mass audience in limited time-frames. According to the BBC documentation the ruling of this committee was passed to the production team for consideration.

(It is possible that Dr Pelham had over-simplified the matter. His reported comments to the press do not always appear to have made it completely clear that “medication” was being compared to a behavioural programme and not to no “treatment”. However one press report does include the comment from Dr Pelham that “There's no indication that medication's better than nothing in the long run”. [40] This is correct. The MTA study did not have an untreated control group and therefore cannot indicate that “medication” is better than no treatment. For this reason it is not possible to be clear whether Dr Pelham failed to explain the exact limits of the MTA study to Panorama or whether he was misunderstood and badly reported).

However; the unnamed complainant was not satisfied and requested that his or her (or their) complaint be escalated. The complaint was duly handled by another BBC complaints body, the Editorial Standards Committee. The full report of the Editorial Standards Committee is available online. [44] This body found a number of new faults chiefly relating to “accuracy” with the Panorama programme and required Panorama to broadcast a “correction” and apology on air. This was a substantial victory for the unnamed complainant. The story put into the public domain that “medication” is not effective in the long-run had been squashed.

The BBC Editorial Standards Committee unfortunately doesn't understand basic science. The ruling states that

> The programme did not make it clear that all the treatment groups had improved at the 36 month stage and that medication did offer a significant improvement over time, albeit not over the other treatment groups, at 36 months. [44]

The MTA study had not been designed to evaluate the effects of “medication” over time. The Editorial Complaints Unit had correctly understood this when they made it the basis for their criticism of Panorama: “the research was not conducted in such a way as to be able to determine that long-term medication conferred no benefit at all”. The MTA study was designed to compare “treatments”, chiefly “medication” versus a behavioural intervention. There was no untreated control group. In the MTA 36 month follow-up study (Jensen *et al*. 2007) [45] it is explicitly conceded that the MTA study *cannot* assess whether or not “medication” leads to an “improvement” over time because the study did not have a control group:

> Of course, without an untreated control group, no firm conclusions about the possibility of more positive ADHD outcomes can be drawn with confidence. [46]

It is a colossal irony that the second BBC committee found fault with Panorama for not doing something which the first committee had pointed out was impossible. The first Committee found

fault with Panorama for saying that “medication” was not effective in the long run. The second Committee found fault with Panorama for not saying that “medication” is effective in the long-run. The MTA follow-up study did not have a control group and cannot be used to make any claims about the absolute “efficacy” of “medication” in the long-run, either way. The Editorial Complaints Unit was correct. The Editorial Standards Committee misunderstands the material they are reviewing.

The BBC Editorial Standards Committee appear to lack the ability for the task they have assigned to themselves. They do not appear to be able to critically assess scientific research papers. For example; the “symptom reduction” scoring game on which the MTA study claims are based is blandly reported by this committee as “offering a significant improvement.” We have discussed how a study based on quantification of interpretations of behaviour made by interested parties is more akin to a marketing survey than “scientific research”. (Section 2)). For a full scientific appraisal of the MTA study see Breggin 2000 [30]). The BBC committee shows no sign of being interested in or capable of critically engaging with the methodological problems posed by the MTA study. They blandly discuss “those affected by ADHD”. [44] In doing so they accept without qualification the reification that when a young person is said to “have ADHD” they actually have some objective condition. This reification essentially accepts the biological narrative about ADHD, the phantasy that behind every “diagnosis” there is a real physical condition. However; not only is this impossible. There is “no specific biological test”. It is also highly unlikely even on the basis of statistical correlations. As we have seen above (sub-section ii)) both Dr Illina Singh and Dr Sami Timimi summarise the evidence from genome studies and MRI scan studies as being at best “inconclusive”. No one is “affected” by ADHD. The “affected by ADHD” text shows us that we are in the realm of the second-tier folksy narrative about “ADHD” which assumes “it” is “biological”. As we discussed (Introduction ii)) this more folksy rendering of the narrative is typical of local authorities.

The BBC Editorial Standards Committee does know how to defer to the establishment. It appears that in response to the original complaint the BBC Editorial Complaints Unit conducted its own mini-research project into “ADHD”.  They consulted Professor Jensen, author of the MTA follow-up study who stated that he believed that “medication” still had some role to play. This is not surprising; Peter Jensen is a die-hard ADHD drugging promoter. [47] Even without knowing anything about the history of Peter Jensen and the NIMH a reading of the MTA follow-up study should disclose that the study authors were disappointed and surprised not to be able to confirm the original MTA study claims that “medication” was “superior” to behavioural treatment and that it is evident that the paper was seeking to explain this in ways favourable to the drugging position. The Editorial Standards Committee appears to have reviewed Professor Jensen's contribution to the ECU. It seems that either this or Jensen *et al*. 2007 itself are likely to have been the basis for their finding that in the original Panorama programme:

> It was not clear that all the treatment groups had improved at the 36 month stage and that medication did offer a significant improvement over time, albeit that at 36 months it had no superiority over other treatment groups. [44]

This echoes Jensen *et al*. 2007:

> Because there was no untreated control group and because all of the treatment groups were improved in terms of relevant symptomatology at 36 months compared to baseline, it is possible that all of the treatments worked, but at different rates or during different time periods. Thus, an important clinical message to be taken from our findings is that all of the treatment groups showed significant improvement over time. [45]

But, without an untreated control group no absolute claims can be made about “medication” being “better” than no “treatment”. The BBC Editorial Standards Committee fails to see the nature of these somewhat desperate manoeuvrings to produce a pro-drugging conclusion out of a study which has failed to do so and holds Panorama to account for not accepting them as fact.

The Editorial Complaints Unit also consulted Professor Eric Taylor. Professor Taylor was the lead on The National Collaborating Centre for Mental Health committee which produced the NICE ADHD Guideline. Professor Taylor is a leading promoter of ADHD in the UK. He is on the board of the pro-drugging ADHD parents group ADDISS (which has accepted funding from at least three separate pharmaceutical companies [48]). He himself has spoken at conferences funded by drug companies. [49]

The BBC Editorial Standards Committee reports that the programme makers had consulted Professor during the course of researching the programme. They found that Panorama had not taken account of Professor Swanson's views:

> The programme did not take proper account of the views of Professor Swanson – set out in an email to the production team - that in some cases the beneficial effect of medication would not be lost after 3 years. [44]

Professor Swanson was a co-author with Professor Jensen of the MTA follow-up study. Like Dr Peter Jensen Professor Swanson is another well-known ADHD-drugging stalwart. [50] Professor Swanson was the author of the secondary evaluation study which attempted to rescue the

drugging position after the “unexpected” results of the MTA follow-up study. We discussed Swanson *et al.* 2007 [51] in Section 4) v) b). The BBC Editorial Standards Committee describes Professor Swanson as being “lead author of one of the MTA papers” without mentioning that this is a secondary evaluation of the data study. This study was partly intended to follow-up and expand the arguments in Jensen *et al*. 2007 which claimed that “all of the treatment groups showed significant improvement over time”. [45] But, Swanson *et al. 2007* is a statistical study based on the MTA follow-up study. It has no untreated control group data either. And thus still it is the case that no claims can be made about “medication” being more “effective” than no “treatment” at all. The BBC Editorial Standards Committee does not appear to understand that the “beneficial effects” reported by Professor Swanson are statistically derived from a study which had no control group. No “benefit” for “medication” has been shown at all according to the terms of a normal randomised clinical trial.

Swanson *et al.* 2007 also showed that after three years for the majority of the sample (66%) it made no difference in terms of ADHD “symptom” count if they were on “high” or “low” (including no) “medication” use at 36 months. (See Section 4) v) b)). One wonders if Professor Swanson told the BBC this? One wonders whether anyone on the BBC Editorial Standards Committee had read his paper and discovered this for themselves? It is in a graph on page 1011 and reported in the text on page 1010. [51] It is true to say, in terms of the study, that “in some cases the beneficial effect of medication would not be lost after 3 years”. But it is also true that at 36 months being on “high” “medication” rather than “low” (or no “medication”) only made a difference (on the symptom reduction scoring system) for 34% of the sample. And, again, this claim about a “beneficial effect” refers to a comparison with baseline. “Symptoms” have dropped but there is no evidence that they have dropped more than they would have done in an untreated control group. Clinically, it is not clear what this finding is supposed to prove. The BBC Editorial Standards Committee holds Panorama to account for not reporting these statistical attempts to recover the drugging position. But these “views” the BBC Editorial Standards Committee says should have been reported by Panorama are the clinically irrelevant findings from a secondary evaluation of the data study carried out by a well-known supporter of ADHD drugging. And even then the statistical finding is only partly supportive of the “benefits” of drugging. For most of the subjects it made no difference whether they were on high or low “medication” at 36 months. The makers of the original Panorama programme quite rightly did not include Professor Swanson's tenuous manoeuvrings in their programme. They are not obliged to report attempts by ADHD promoters to recover from an awkward study finding by statistical manoeuvrings.

One of the stated aims of Professor Swanson's secondary evaluation of the data paper was to try to explain the convergence of scores in the MTA follow-up study between “medication” and a behavioural programme. This convergence of scores suggested that there is no medication advantage in the long-term. This was what the Panorama report was discussing and what the controversy was about. The MTA follow-up study authors (Jensen *et al. 2007)* had proposed the

“self-selection” hypothesis to account for this convergence. The suggestion is that those who were “doing well” on “medication” stopped using “medication” while those whose “symptoms” were high were starting. This would have explained the convergence of scores by some other interpretation than the “medication advantage” wearing off. This would, if proved, therefore have preserved the “medication advantage” theory. Testing this hypothesis was one of the stated aims of Professor Swanson's paper. Swanson *et al*. stated that they failed to confirm this hypothesis:

> We failed to confirm the self-selection hypothesis [51]

This evidence from Swanson *et al*. 2007 supports the interpretation of the MTA follow-up study that the “medication” advantage wears off over time. Possibly in his email to the programme makers Professor Swanson did not mention the details which we have just reviewed. If he did the BBC Editorial Standards Committee did not allow them to interfere with their judgement. If he didn't the Committee appears to have failed to discover these facts for themselves.

A chink of light in the ADHD (drugging) narrative was accidentally opened up by the MTA follow-up study. Accidentally, the study had shown that, even in terms of the “symptom reduction” game, in the longer run medication is no “better” than a behavioural treatment. (Or at least there was a substantial convergence of scores. (See Section 2) vii))). The ADHD-drugging machine went to work quickly to get the narrative back on track. When Panorama gave publicity to the fault-line that had opened up that the “medication advantage” was not sustained in the long term a public sector committee in the BBC management chain established what the “views” were of a number of key proponents of the ADHD drugging story and simply rendered their version. The problem with this is that if the BBC management's response to a complaint about a controversial piece of journalism will be to simply refer to and accept the “views” of those most closely linked to the centre of power then there is no point BBC journalists ever trying to report on controversial matters. The BBC will simply exist to echo mainstream establishment views without reference to the scientific credibility of those views.

The BBC refused to divulge to this writer who the complainant was in this case, citing legal provision in the UK's Freedom of Information legislation designed to protect journalistic sources. The public is thus not able to know what forces were able to stifle this relatively rare critical reporting of the ADHD narrative in a mass-audience programme. However it is clear from reading the Editorial Standards Committee report that whoever they were they were persistent, almost driven, in their pursuit of justice. They had also received “letters from third-parties” in support of their campaign. [44]

iv) Symptoms and treatment: behaviour modification disguised as medical science

In the above discussion about the MTA study (Section 2 iii)) we have seen how an appropriation of medical terminology is used to disguise what is really happening with ADHD drugging. We have seen how the NICE Guideline authors by accepting the MTA study methodology uncritically have adopted this language of “symptom reduction” (Section 4) iv)) in their assessment of the MTA study. The MTA study talks about “symptoms” no less than 34 times in a paper whose overall length is about 6,000 words. The phrase “ADHD symptoms” occurs 293 times in the NICE Guideline. It is used to by the NICE Guideline authors to make claims which are intended to seem “medical” and “scientific”. The essential way the “symptom reduction” game is works is this. Parents and teachers (usually) complete rating questionnaires about their child or student's behaviour. What is being rated are behaviours related to “inattention”, “hyperactivity” and “oppositional” or difficult behaviours; ”getting up from seat when remaining in seat is expected”, “acting smart” etc. These subjective interpretations of behaviour assessments are then aggregated and a way found to quantify what is now called “data”. Thus it can be claimed that drug X “reduced symptoms” compared to no drug (or “better” than a behavioural programme as in the MTA study). This is then taken as a self-evident good and a flag goes up “in a clinical trial of 50 young people with ADHD drug X reduced ADHD symptoms”. The first part of the hoax is the “with ADHD”. This implies that the 50 young people had something. They don't; other than a label related to their behaviour. As NICE admits, the ADHD label implies: “no medical or neurological cause”. The second part of the hoax hinges on misuse of the word “symptoms”. Everyone who is ill wants to have their symptoms reduced. So claims that “symptoms have been reduced” sounds like a clinical benefit. But; these are behaviours, not symptoms. There is no medical condition and thus there are no “symptoms” which could have been reduced. No suffering has been reduced. Often, perhaps usually, suffering will have been created. Both “disorder” and “symptoms” are fake. This talk of the “symptoms” of a “disorder” being reduced” is intended to make the programme of mass drugging of young people to curtail disruptive behaviours acceptable to the public. In fact, as it turns out this does not lead to any long-term improvement for the young person (Section 4) vii) a)). It does lead though to an “easier to handle” (Section 4) viii)) and more “compliant” (Section 4) viii)) young person.

Other aspects of the hoax include a certain discretion with regard to the short-term nature of most of the “symptom reduction” claim studies, a certain discretion concerning the fact that the majority of the studies are funded by the same companies which sell the drugs, and a tendency to play down the horrendous side-effects.

v) “ADHD” is a product of psychiatry and modern schooling

In *Madness and Civilization* [52], the French social critic Michel Foucault showed how modern psychiatry has its roots in an authoritarian and moral tradition. The modern asylum, which came into being around 1800, is a police institution, not a medical one. Foucault shows how after 1800 madness was conceived and treated differently than it had been in the Classical Age. In the Classical Age madness had been confronted (even with chains). It was unreason and error, but not a moral error. With the birth of the asylum at the start of the 19th century madness is now understood as a moral disease. This is Foucault:

> Surveillance and Judgement: already the outline appears of a new personage who will be essential in the nineteenth century asylum.
>
> For this new reason which reigns in the asylum, madness does not represent the absolute form of contradiction, but instead a minority status, an aspect of itself that does not have the right to autonomy, and can live only grafted onto the world of reason. Madness is childhood. Everything at the Retreat is organised so that the insane are transformed into minors. They are regarded as “children who have an abundance of strength and make dangerous use of it. They must be given immediate punishments and rewards; whatever is remote has no effect on them. A new system of education must be applied, a new direction given to their ideas, they must first be subjugated, then encouraged, then applied to work, and this work made agreeable by attractive means”.
>
> [52]

The Retreat mentioned in the text was the new asylum set up near York by the Quaker William Tuke in 1796. The citation in Foucault's text is of De la Rive.

We are reminded of the “reduced repetitions” method of handling “ADHD children” promoted by the psychiatrist and leading ADHD advocate Russell Barkley, which we met with in Section 2) iv) and again in Section 4) viii). This approach emphasises giving commands and warnings and following up on the warnings rather than just repeating the command. This is how NICE reports this approach:

> ... consists of the teacher giving the child a command once and, if necessary, proceeding to a warning where the child is informed of the consequences of not carrying out the command; in cases where the child does not comply, the threat is carried out [53]

This is rather similar to dog training. The idea that it is acceptable to “threaten” “children” reflects a sense of “children” as beings with *less rights* than adults. This idea of the minority status of the “child with ADHD” is something we have seen repeatedly in reviewing the “science” of ADHD. ADHD promoters seem to have an especially strong sense of the division between adults and “children” and how the latter should behave in terms of the expectations and requirements of the former. This is the emphasis on the minority status of the “mad” which Foucault found in his study of the birth of the asylum.

Foucault saw in the birth of the modern asylum and in the origins of modern psychiatry a bourgeois and patriarchal defence of the family against disorder. The first asylum was run like a family and madness was that which confronted the order of this institution. Victory, of reason, was secured, in advance, by the designation of the mad as minors, not just in a legal sense but in a real concrete situation. They were treated as minors. One feature of The Retreat was tea-parties. The patients attended. All those attending acted out a ritual of a formal and correct English tea-party. But this was not, as Foucault points out, “one of intimacy, of dialogue, of mutual acquaintance”. The patients are only accepted by reason when they have been objectified in the eyes of reason as “the perfect stranger, that is, as the man whose strangeness does not reveal itself”. Everything “strange” about them must be hidden, suppressed. The “ADHD child” is similarly objectified. The “diagnosis” turns the individual with unique needs and feelings into the ADHD child. Thus objectified no one needs to forge a mutual acquaintance with them. They are a “child with ADHD”. The drugs repress the behaviour which they would otherwise display. They become the “perfect stranger”.

Dr Singh is pleased that methylphenidate suppresses the glee in being naughty that the “ADHD children” displayed. (See Section 3) vi)). Reason, in the form of a methylphenidate prescription, has conquered the unreason of glee in naughtiness. Modern psychiatry was first and foremost a moral endeavour. Its aim was to correct the moral deficiency which was madness. It did this through authoritarian methods. It subsequently found scientific rationales to justify its operations and explain its successes. ADHD drugging is a technology of psychiatry but is no less “moral” for being a drug.

ADHD is a product of psychiatry. It is a “condition” only because psychiatry has defined it as such. As we have seen psychiatry (Section iv) ii)) is in fact quite careful to acknowledge this, at least in its official pronouncements. The definition may change over time. Thus ADHD is the property of psychiatry. Something which is maintained by psychiatry.

To understand ADHD therefore we must understand the history of psychiatry. We would need to discuss how psychiatry has come to be able, historically, to create social categories and designate some people as “having” the conditions they have defined. We would need to discuss the role such dividing practices play in the management (or “policing” in Foucault's sense of the word) of the

population. Certainly statistical correlations can be found between the ADHD label and genetic or other factors. This “scientific fact” (if you like) does not obviate the need for such a historical and political enquiry.

In the case of ADHD labelling psychiatry has become overtly involved in a behaviour management problem in schools. Given the mass nature of the modern schooling system it is inevitable that the standard (requirement to pay attention, “sit still”, etc.) will be set around the average. Given that human beings vary around an average it is inevitable that some young people in school will be below average for the group they are in. This will become especially apparent when schools are rigorously age-banded as modern schools are, when classes are large and, when the curriculum demands that the whole group move together at a certain pace and when the curriculum is quite academic and intensive. In these circumstances young people who are below the average in ability to pay attention for their age-group will “stick out”. This may become problematic in terms of classroom management and for the orderliness of the school. At this point it would be entirely possible to re-think modern schooling, especially its mass approach. And why not? But; the dominant thinking of the day is to look for manipulative solutions (as we have discussed above, sub-section i)). From this viewpoint a service or product has to be *delivered* which will fix the problem. An ADHD “diagnosis” firmly locates the problem “in” the “child”. Pharmaceutical companies provide the solution in the form of drugs. The school system does not need to consider its practices. This is a “right-wing” solution, in Illich's sense of “right-wing”. That is, hierarchical, dominating, manipulative and self-interested.

Aware of the criticism that drugging is for behaviour modification not for “treatment” of a putative disease psychiatry offers a last ditch defence. ADHD drug treatment is an act of compassion to help “children” who are “impaired” (i.e. disabled). This is very much in line with how power presents itself in the UK today. Every scheme to manipulate the recalcitrant and non-compliant into line is presented as “supporting” them. This is how the NICE Guideline expresses this concern with “impairment”:

> All the speakers acknowledged the importance of functional impairments in relation to diagnosis. In other words, the diagnostic threshold should be based on pragmatic grounds such as impairment and the need for treatment. [20]

This is Professor Eric Taylor expressing the same view:

> This interaction between the child and the expectations of the adult world is important clinically. It is a reason to take more pains in making a diagnosis than just accepting a rating from a parent or teacher. Impairment and risk are as important as symptomatology. [49]

This is an attempt to put the ADHD diagnosis onto an objective and “clinical” footing. (Interestingly this also appears to be an admission from Professor Taylor that in the UK it sometimes happens that psychiatrists “diagnose” “ADHD” by “just accepting a rating from a parent or teacher”). What is “impairment”? This is how the NICE ADHD Guideline defines “impairment”:

> Moderate ADHD in children and young people is taken to be present when the symptoms of hyperactivity/impulsivity and/or inattention, or all three, occur together, and are associated with at least moderate impairment, which should be present in multiple settings (for example, home and school or a healthcare setting) and in multiple domains where the level appropriate to the child’s chronological and mental age has not been reached: self-care (in eating, hygiene, and so on); travelling independently; making and keeping friends; achieving in school; forming positive relationships with other family members; developing a positive self-image; avoiding criminal activity; avoiding substance misuse; maintaining emotional states free of excessive anxiety and unhappiness; and understanding and avoiding common hazards. [54]

“Impairment” is when the “child” is, on certain measures, below the level which would be appropriate for their “chronological and mental age”. (In DSM-IV the “signs” are said to be relevant if they are “disruptive and inappropriate for developmental level”). How is “inappropriate” for chronological and mental age to be defined? As far as inappropriate for chronological age, the only way that appropriate can be determined is by comparison with what is typical. Because of the way that statistics works some people are *bound* to fall outside the average (calculated either as the mean or the median). The only way this could not be true would be if everyone was *exactly* the same. A *Brave New World c*lone. To consider a specific example. A young person may be young for their age and not able to travel on a bus alone at an age when it is normally expected that they should be able to travel on a bus alone. But is this a reason to try to batter them into shape with drugs? Perhaps (in some cases) this really is the reality. In which case we may just have to accept that for this young person being able to travel independently may come a little bit later. The second concept here is more nebulous. Being unable to do something which is normal for “mental age” or for “developmental level” (DSM-IV) suggests a real “impairment”. (Though, again, “impairment” is still being defined according to an external standard, a process which inevitably focusses on and creates a sense of a deficiency). The question might be in these cases: is our intervention supportive of the person with this impairment, will it help them achieve the best they can despite the impairment? This is how the modern discourse on disability usually works.

The definition of impairment in the NICE Guide lists a number of examples. These include: “self-care (in eating, hygiene, and so on); travelling independently; making and keeping friends; achieving in school; forming positive relationships with other family members; developing a positive self-image; avoiding criminal activity”. But there is an anomaly here. The evidence from within the

ADHD narrative itself is that the drugs don't improve these measures (or, in some cases if they do, no better than a behavioural intervention). In the MTA study the young people did not report that anxiety/depression was more improved by methylphenidate than by a behaviour programme. The NICE authors accept that drugging does little to improve academic performance:

> Equally, studies have not demonstrated clear effects of stimulants on academic performance or learning (Swanson et al., 1993). [31]

Even Dr Singh seems to accept methylphenidate does not offer improvements in the area of self-image:

> Second, children's moral conceptions of their authentic selves are characterized by persistent badness, despite medication. [14]

The NICE authors appear to concede no long-term improvement:

> There is little evidence that stimulant medication alters the relatively poor long-term outcome for many of those with ADHD. [32]

Is putting a young people on amphetamines really likely to improve their peer friendships? On the contrary it seems more likely that this will "make them 'feel different' in a negative way". [12] The drugs don't appear to fix the "impairments".

In the side-effects reporting of the MTA study it was reported that 14% of the young people on methylphenidate became "impaired" as a result. The drugs cause impairment.

Behavioural interventions often come from the same conceptual basis as drugging interventions. Johnny needs to be fixed so that he is not "disruptive". However; at least these interventions do not cause insomnia, nervousness, stomach aches, suicidal thinking and so on. Furthermore; behavioural interventions can potentially offer a meaningful way to engage with "impairment". If a ten year old has problems catching the bus on his own because he is forgetful and easily distracted, by improving concentration stimulants may temporarily help with that in the short-term and in a very marginal way. But they won't help him *learn* how to catch a bus. On the contrary by masking the problem they are likely to work against him learning how to catch a bus. They may make him dependent on the drugs to catch the bus in the future. No wonder "There is little evidence that stimulant medication alters the relatively poor long-term outcome for many of those with ADHD". [32]

Some young people may be “impaired”. But if the proposed solution does not help we can question the nature of the concern with “impairment”. And, in reality, if young people really are “impaired” and this really is the result of a genetic coding error, as may indeed be the case in a small number of young people who attract an ADHD label, is it likely that giving them drugs which are like cocaine and amphetamines is going to help them?

A humane discourse about “impairment” and disability seems to have been invaded and taken over by a more punitive discourse where a deficiency justifies an intervention, though that intervention is not one which is about meeting the needs of the person with the “impairment”. In this discourse the impairment is a deficiency to be corrected. Not a reality which may require adaptations to the young person's environment so they can flourish despite the impairment.

ADHD is similar to EBD (Educational and Behavioural Difficulties). EBD is a management category used in schools. A “pupil” may be labelled “EBD” (Educational and Behavioural Difficulties) if they misbehave a lot at school and offer, intentionally or unintentionally, resistance to the processes the schooled form of education is trying to put them through. The labelling is a convenience for the school. The designation serves to create a managed distance between the teacher and the difficult student. Into that distance is now inserted “support”, which is a euphemism for more intensive surveillance and management. The designation of EBD may make it easier to dispatch the young person to a special school of some kind. Given the size of a typical secondary school class in UK schools it is entirely understandable that many teachers welcome a system which corrals and manages especially disruptive students.

As with ADHD, boys significantly outnumber girls in the EBD category. One figure suggests that in England boys are ten to twelve times more likely to end up in a special school for “children with EBD” than girls. [55] As with ADHD the literature on EBD frequently talks about young people “with EBD” [55] as if they *have* something. Some of the EBD literature acknowledges that the “context”, which is taken to include factors such as the ethos of the school, is a relevant factor. [56] This represents a more sympathetic discourse than is characteristic of the ADHD discourse. “EBD” shares several similarities with “ADHD”. These include:

- The EBD label is attached to a young person by someone in authority, in this case the process is managed by the local authority and may involve an educational psychologist.
- There is no biological test for EBD.
- It is a case of diagnosis by check-list.
- Like ADHD this does not stop people discussing the “possible biological causes”.

- EBD is designed to solve management problems in schools.

As with ADHD the designation of EBD moves the young person into a special category where they may get special interventions. People in power will talk about the young person as “having” something objective. This reification (treating as objective and part of the natural world something which is man-made and arbitrary) masks the act of power which imposes the label. An ADHD “diagnosis” is reached in the same way as a designation of “EBD”. There is nothing more “scientific” about ADHD than “EBD”. However “ADHD” has more status than “EBD”. This greater status derives not from any actual scientific difference between the two labels but simply from the greater status of psychiatry as compared to teachers, and the pretensions of psychiatry to be related to medical science.

## Conclusion

In order to explain the programme of drugging a narrative is constructed about a “disease” being “treated” and “symptoms reduced”. In genome studies and MRI scan studies small statistical differences between an average value from an ADHD group and a “normal” group are used to promote the idea of the “validity” of the “disorder”. But correlations are not evidence of causality. No clinical condition is established by these studies, which simply show a range of statistical correlations to the label. Often MRI studies cannot properly distinguish between effects which may be caused by ADHD drugs themselves and intrinsic “abnormalities”. Nor do MRI studies typically consider the effects on behaviour of being labelled. Many studies compare the ADHD group not with the population average, but with a group from whom the ADHD set has been subtracted. This attenuates findings of difference. Often findings from studies are further manipulated and/or selectively reported in order to build the case for drugging.

Psychiatry responds to the challenge of the tenuousness and varied nature of the statistical correlations of ADHD studies by defining “ADHD” in such a broad way that the “disorder” can be permanently upheld. The current definition is: “The aetiology of ADHD involves the interplay of multiple genetic and environmental factors”. This is a definition which can never be disproved. Thus psychiatry ensures the safety of its “diagnostic category” and the inevitable continuance of drugging.

There is a “public debate” about “whether ADHD is a real disease”. But this “debate” misses the point. ADHD is what it is: a “diagnostic category” of psychiatry which “does not imply a medical or neurological cause”. “Diagnosis” is via a behaviour check-list. The more serious question concerns the role of psychiatry in society. How is it allowed to produce these systems of diagnosis? What role do they play in managing deviance? What is the relationship between psychiatry and its “diagnostic categories” and the pharmaceutical industry?

Drugs to “treat” ADHD are licensed in the UK by the MHRA. (Some drugs are licensed at a European level by the EMA). The MHRA specifies that in considering the merits of a drug it is necessary to consider: “Do the advantages outweigh the disadvantages of taking the medicine?” On the evidence reviewed in this paper it is quite simply hard to see how drugs such as methylphenidate and atomoxetine can have been licensed to “treat ADHD” in the UK. Once we probe behind the “symptom reduction” claim the alleged “benefits” of the drugs are difficult to ascertain. Claims tend to be somewhat folksy such as “improving the quality of life”. The only certain positive effect of stimulant drugs is a short-term increase in ability to concentrate; an effect which is the same for everyone whether or not they have an ADHD label. But the ADHD narrative concedes that this does not translate into an improvement in long-term outcomes. The actual “beneficiaries” of ADHD drugging may be those parents and schools who are glad to see a reduction in the disruptive behaviours which constitute an ADHD diagnosis. But this is not an

advantage to the young person. On the other hand the harms are real and tangible and accrue to the young person. For example, methylphenidate routinely causes insomnia and stomach aches. Imagine the effect of suffering from drug induced insomnia throughout your childhood. Atomoxetine is linked to suicidal thinking and suicidal attempts. Under a heading which includes self-injurious thinking, self-injurious acts, suicidal thinking, suicidal attempts and actual suicides the MHRA's adverse event reporting scheme recorded 122 cases in under 18s between 2003 and 2013 where atomoxetine was suspected as being responsible. According to the manufacturer, the most common side-effects of atomoxetine in young people are upset stomach, decreased appetite, nausea or vomiting, dizziness, tiredness, and mood swings. The US FDA has issued a warning that post-launch adverse event reporting has associated atomoxetine with possible serious liver damage. These considerations make it hard to see how the MHRA's test for whether or not to license a drug can have been seriously applied.

NICE was asked by The Department of Health to produce a Guideline on the “diagnosis and management” of ADHD. NICE commissioned The Royal College of Psychiatrists and The British Psychological Society to produce the Guideline. This is like commissioning Procrustes to manage your patient bed problem. Further problems exist. The MHRA licenses each drug on a case by case basis. The MHRA is not required to consider whether a non-drug behavioural intervention might be equally as “effective” as a drug intervention. This explains the astonishing state of affairs that permits drugging at all when, even on the most biased evidence, it appears that behavioural interventions can be nearly as “effective” as drugging and yet have none of the serious life-threatening or debilitating “side-effects”. There appears on the surface to be a system of “checks and balances” but, in reality, there appears to be nothing in the way of the flow of toxic drugs from US pharmaceutical companies straight into the mouths of British young people.

## Appendices

**i) DSM-IV.**

IA. Six or more of the following signs of inattention have been present for at least 6 months to a point that is disruptive and inappropriate for developmental level:

- Often does not give close attention to details or makes careless mistakes in school-work, work, or other activities.
- Often has trouble keeping attention on tasks or play activities.
- Often does not seem to listen when spoken to directly.
- Often does not follow instructions and fails to finish school-work, chores, or duties in the workplace (not due to oppositional behaviour or failure to understand instructions).
- Often has trouble organizing activities.
- Often avoids, dislikes, or doesn't want to do things that take a lot of mental effort for a long period of time (such as school-work or homework).
- Often loses things needed for tasks and activities (such as toys, school assignments, pencils, books, or tools).
- Is often easily distracted.
- Often forgetful in daily activities.

IB. Six or more of the following signs of hyperactivity-impulsivity have been present for at least 6 months to an extent that is disruptive and inappropriate for developmental level:

- Often fidgets with hands or feet or squirms in seat.
- Often gets up from seat when remaining in seat is expected.
- Often runs about or climbs when and where it is not appropriate (adolescents or adults may feel very restless).
- Often has trouble playing or enjoying leisure activities quietly.
- Is often "on the go" or often acts as if "driven by a motor".
- Often talks excessively.
- Often blurts out answers before questions have been finished.
- Often has trouble waiting one's turn.
- Often interrupts or intrudes on others (example: butts into conversations or games).

II. Some signs that cause impairment were present before age 7 years.

III. Some impairment from the signs is present in two or more settings (such as at school/work and at home).

IV. There must be clear evidence of significant impairment in social, school, or work functioning.

V. The signs do not happen only during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder. The signs are not better accounted for by another mental disorder (such as Mood Disorder, Anxiety Disorder, Dissociative Identity Disorder, or a Personality Disorder)

NB. Depending on whether 1A or 1B applies or both, different 'types' of ADHD can be 'diagnosed'.

Source: From Wikipedia.

**ii) ICD-10**

Inattention

At least 6 of the following symptoms of attention have persisted for at least 6 months, to a degree that is maladaptive and inconsistent with the developmental level of the child:

- Often fails to give close attention to details or makes careless errors in school work, work, or other activities
- Often fails to sustain attention in tasks or play activities
- Often appears not to listen to what is being said to him or her
- Often fails to follow through on instructions or to finish school work, chores, or duties in the workplace (not because of oppositional behaviour or failure to understand instructions)
- Is often impaired in organising tasks and activities
- Often avoids or strongly dislikes tasks, such as homework, that require sustained mental effort
- Often loses things necessary for certain tasks and activities, such as school assignments, pencils, books, toys, or tools
- Is often easily distracted by external stimuli
- Is often forgetful in the course of daily activities.

Hyperactivity

At least 3 of the following symptoms of hyperactivity have persisted for at least 6 months, to a degree that is maladaptive and inconsistent with the developmental level of the child:

- Often fidgets with hands or feet or squirms on seat

- Leaves seat in classroom or in other situations in which remaining seated is expected
- Often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, only feelings of restlessness may be present)
- Is often unduly noisy in playing or has difficulty in engaging quietly in leisure activities
- Exhibits a persistent pattern of excessive motor activity that is not substantially modified by social context or demands.

Impulsivity

- At least one of the following symptoms of impulsivity has persisted for at least 6 months, to a degree that is maladaptive and inconsistent with the developmental level of the child:
- Often blurts out answers before questions have been completed
- Often fails to wait in lines or await turns in games or group situations
- Often interrupts or intrudes on others (e.g., butts into others' conversations or games)
- Often talks excessively without appropriate response to social constraints
- Onset of the disorder is no later than the age of 12 years.

Pervasiveness

The criteria should be met for more than a single situation: for example, the combination of inattention and hyperactivity should be present both at home and at school, or at both school and another setting where children are observed, such as a clinic. Evidence for cross-situationality will ordinarily require information from more than one source; parental reports about classroom behaviour, for instance, are unlikely to be sufficient.

The symptoms cause clinically significant distress or impairment in social, academic, or occupational functioning.

The disorder does not meet the criteria for pervasive developmental disorders, manic episode, depressive episode, or anxiety disorders.

Source: http://bestpractice.bmj.com/best-practice/monograph/142/diagnosis/criteria.html

## Notes & references

Some references are to web links. Internet resources can be moved. For all references the author has a copy of either the web page or the document at the time of writing. If the reference is missing and you would like to review it please contact the author of this essay.

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Figures for number of ADHD labelled young people on drugs

The NHS does not record how many young people are being “medicated” “for ADHD”.

The Daily Mail gives a figure of 100,000 in Britain for Ritalin. This is on a par with other figures circulating in the press. Britain includes England, Wales and Scotland:

http://www.dailymail.co.uk/health/article-180565/Is-end-Ritalin.html

It is possible to attempt to calculate the numbers based on the number of prescriptions issued and the fact that a prescription for a controlled drug has to be renewed every 28 days. We can use the Prescription Cost Analysis data for England provided by the NHS to obtain an estimate for England

(excluding Scotland and Wales). On this basis, if the 859,000 prescriptions for methylphenidate preparations in 2013 in England were for a fixed number of young people repeated once each month that gives a figure of 71,500 on methylphenidate in England. Given the rate of growth in prescriptions this figure corresponds reasonably well with a figure given by the BBC in 2007 for methylphenidate of 55,000. (See below). On the same basis the 42,100 prescriptions for dexamfetamine sulphate in England in 2013 would produce a further 3,508 individuals. Since atomoxetine hydrochloride is not a controlled drug prescriptions for this substance only need to be renewed every 6 months. On this basis the 113,000 prescriptions in 2013 in England produces 56,500 individuals. These calculations would make an approximate overall total on ADHD drugs in England alone in 2013 of 131,508. These figures depend of course on a number of assumptions. (One assumption is that all “ADHD” drugs are given to young people. It is the case that the vast majority of ADHD “medication” is aimed at young people. Dexamfetamine and methylphenidate are only licensed to treat “ADHD” in young people. Atomoxetine is licensed to “treat” ADHD in children and is also licensed for adults who were “diagnosed” as young people).

Very approximately, assuming the same rates of drugging Scotland would add a further 12,900 and Wales a further 7,650 and Northern Ireland a further 4,400 making a total for the UK of somewhere around 156,500.

The 55,000 figure for methylphenidate is given by the BBC quoting the Centre for Paediatric Pharmacy Research, University of London. It appears to cover the UK as a whole.

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http://thenewobserver.co.uk/wp-content/uploads/2015/06/nice-guide.pdf

We haven't linked to the current revised version because it is not the purpose of this paper to track all the edits to the document. (NICE has removed the revised version from their web site which now returns an error message).

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* This paper used a copy of the NICE Guideline which has been subsequently revised. The copy which this paper refers to is available here:

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