ADHD

Dividing and Drugging

This paper is one section of a full critique of ADHD drugging in the UK.

For the full paper please visit:

http://thenewobserver.co.uk/features/adhd/

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.

<u>iv) How NICE uses the MTA study</u>

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”.

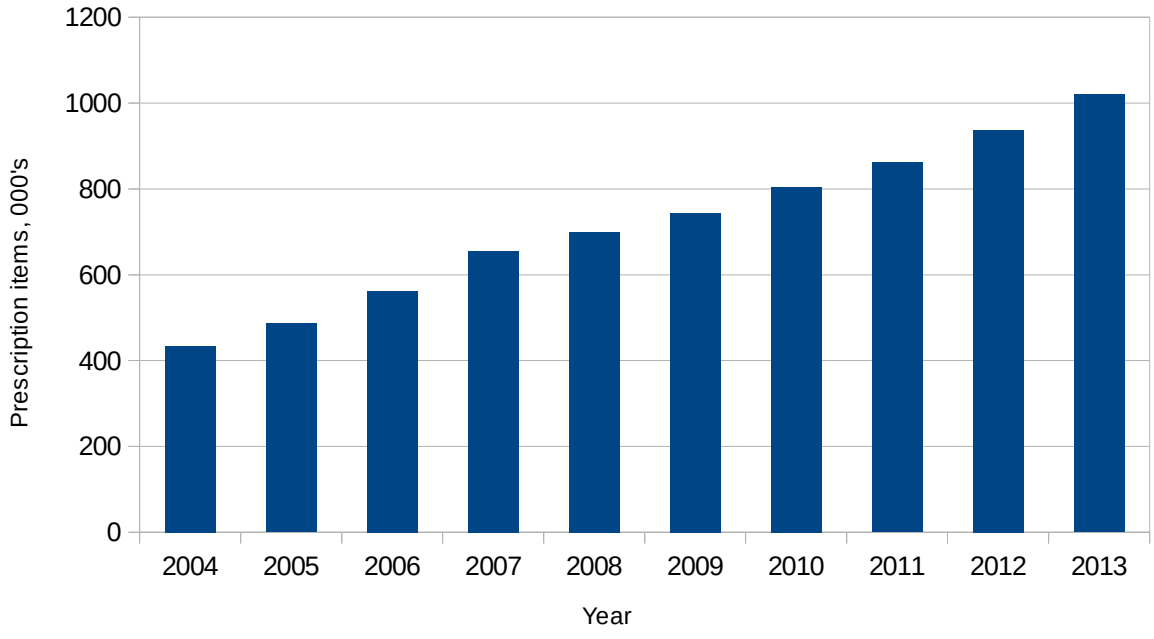
A psychiatric label is a sign not only of difference but of problematic difference. No wonder young people feel isolated by it and try to hide that they have been given this label. It would appear that rather than face up to the very considerable “stigma” caused to young people by ADHD labelling, and 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)).

Notes & References

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* This paper used a copy of the NICE Guideline which has been subsequently revised. The copy which this paper criticised is available here:

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).

2. *Ibid.* Section 10.18.2.1

3. *Ibid.* Section 10.18.3.1

4. *Ibid.* Section 2.4

5. *Ibid.* Section 5.14

6. *Ibid.* Section 5.3

7. Dr Peter Breggin. *Talking Back To Ritalin*. Perseus Publishing 2001. Chp. 3 pp 68-69

8. *Ibid.* Note 1. Section 5.8.1

9. *Ibid.* Section 5.10

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16. *Ibid*. Section 5.15.2 (F)

17. *Ibid*. Section 5.15.2 (G)

18. *Ibid*. Section 5.15.2 (H)

19. *Ibid*. Appendix 16. Section 1.1.6 (Appendix 16 is included in the main document).

20. *Ibid*. Sections 11.3, 11.4

21. *Ibid*. Section 11.3.3

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26. *Ibid*. Section 11.4.1

27. *Ibid*. Section 11.3.5

28. *Ibid*. Section 10.8.5

29. *Ibid*. Section 11.7.1.1

30. Santosh, P. J., Taylor, E., Swanson, J., *et al*.. *Refining the diagnoses of inattention and overactivity syndromes: a reanalysis of the multimodal treatment study of attention deficit hyperactivity disorder (ADHD) based on ICD–10 criteria for hyperkinetic disorder*. Clinical Neuroscience Research. December 2005.

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32. *Ibid*. Note 1. Section 12.5.3.3

33. *Ibid*. Section 10.18.3.4

34. *Ibid*. Section 11.6

35. *Ibid*. Section 10.6.1

36. *Ibid*. Section 4.4.3 Case Study E

37. *Ibid*. Note 7. Chp 1 p 7

38. *Ibid*. Note 1. Section 10.6.2

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http://www.mhra.gov.uk/home/groups/comms-ic/documents/websiteresources/con2031677.pdf

40. Dr Ilina Singh. *Clinical Implications of Ethical Concepts: Moral Self-Understandings in Children Taking Methylphenidate for ADHD*. Clinical Child Psychology and Psychiatry. April 2007.

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44. *Ibid*. Note 7. Chp. 1 p 6

45. *Ibid*. Chp. 1 p 15, Chp. 2 p 49

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52. Bereket, A., Turan, S., Karaman, M. G., *et al*.. *Height, weight, IGF-I, IGFBP–3 and thyroid functions in prepubertal children with attention deficit hyperacticity disorder: effect of methylphenidate treatment*. Hormone Research. March 2005.

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58. *Ibid*. Note 1. Section 10.6.5

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61. *Ibid*. Note 1. Section 10.8.2

62. See Note 32. for Section 3)

Note 32:

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.

http://www.bbc.co.uk/pressoffice/pressreleases/stories/2007/11_november/12/adhd.shtml

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http://online.wsj.com/article/0,,SB108249266648388235,00.html

Requires subscription. The article explains Lilly's programme for trying failed drugs for other purposes. The specifically relevant passage is:

“Many Lilly drugs have risen from failure. Evista, now a $1 billion-a-year drug for osteoporosis, was a failed contraceptive. Strattera, a hot-selling drug for attention deficit/hyperactivity disorder, bombed out as an antidepressant”.

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66. *Ibid*. Note 1. Section 10.8.1

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68. *Ibid*. Note 1. Section 10.8.5

69. *Ibid*. Section 10.7.5

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84. *Ibid*. Section 10.6.5

85. *Ibid*. Note 7. Chp 2 p 29. Dr Breggin is quoting the Physician's Desk Reference

86. *Ibid*. Note 1. Section 10.6.5

87. *Ibid*. Section 5.6.2

88. *Ibid*. Appendix 16. Section 1.7. .Appendix 16 is included in the main document. See Note 1.

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90. *Ibid*. Section 10.8.3

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