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/

The drugs

i) The drugs

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

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

Notes:

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

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

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

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

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

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

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

ii) The myth of the paradoxical effect

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

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

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

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

> There appeared a definite 'drive' to accomplish as much as possible during the school period, and often to spend extra time completing additional work. Speed of comprehension and accuracy of performance were increased in most cases. [6]

> It appears paradoxical that a drug known to be a stimulant should produce subdued behaviour in half of the children. [7]

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

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

The idea that there was a “paradoxical effect” has continued in the ADHD discourse since the time of Bradley. It was well known from the Second World War onwards (at least) that

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

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

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

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

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

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

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

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

being able to offer a clear explanation of how they work.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

ADHD research studies which show some kind of brain abnormality or difference in “children with ADHD” are all based on averages across groups and statistical comparisons with the normals. In any one “clinical” case there is no test “for ADHD”. Which, anyway, “does not imply a medical or neurological cause”. [15] Therefore when a doctor prescribes drugs “for ADHD” she has *no idea* what is going on in her patient's brain. The prescription of these powerful drugs is based on a guess. The “chemical top-up theory” which is essentially the theory behind this guess states that “children with ADHD” have reduced dopamine levels and explains that the drugs raise the levels of dopamine. But since there is no diagnosis for ADHD and since it is all a matter of statistics some, at least, with the label will have perfectly normal brains - with no abnormality and no chemical deficiency. What will happen to their brains when they are given their top-up? This will be the equivalent of “healthy young people” taking stimulants such as amphetamines or cocaine. According to information published regularly by the government about the dangers of taking stimulants, they will be very seriously harmed. [16]

Let us imagine that a test for dopamine shortage or resistance to methylphenidate was developed and prescription of stimulants was only in these cases. It *still* wouldn't follow that prescribing methylphenidate would be a good idea. To assume that this would be a good idea requires a purely mechanistic view of a human being and the human brain. The implicit model in this process is that the human brain is like a bucket. If the level of a certain chemical in the bucket is 1% below the average level for people (“children”) of that age we should just top it up. Obviously human beings are much more complicated than this. The brain is complicated. The drugs can change one variable in the brain but there is no understanding of the whole and we cannot, therefore, be sure that changing this one variable with drugs is a suitable “treatment”. It may be that in the case of a

young person with increased resistance to methylphenidate and/or reduced levels of dopamine their brain produces just the right amount of dopamine for their particular brain structure. We don't know that this is not the case. The dopamine top-up theory is a convenient *folk-truth*, not science. It is difficult not to see it in terms of the marketing objective of selling more pharmaceutical products and raising the stock price of certain US pharmaceutical companies.

iv) The ADHD drugs market is large and growing

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

Year	**Number of prescription items dispensed '000 s**	**Net ingredient cost £ million**
2004	434	14
2005	486	19
2006	562	23
2007	655	26
2008	699	29
2009	744	31
2010	804	34
2011	861	39
2012	937	42
2013	1020	45

Notes:

a) Figures have been rounded to nearest whole number.

b) Source: NHS Information Centre [17]

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

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

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

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

The figures show that there has been substantial growth in the ADHD drugs market over the last 10 years. The market in England alone for all ADHD drugs in 2013 was £45 million. This included £32 million for methylphenidate. [17] This figure excludes private

prescriptions and drugs administered in hospitals so the true size of the market is actually larger.

The market for ADHD drugs in the US is vast by comparison. The US "medicates" more young people with methylphenidate per capita than the UK. The ratio was 1.25:1 in 2003 [18]. The population of the US is approximately approximately 6 times greater than that of England. As a very rough estimate this produces a figure for the US market for methylphenidate alone, (not the other ADHD drugs such as the amphetamines), of at least £240 million in 2013. This is compatible with the figure of US consumer sales of Ritalin (branded methylphenidate) in 1995 being USD 349.3 million, approximately £222 million, provided by marketing consultancy IMS America, quoted by Dr Peter Breggin. [19] One study gives a figure for the total worldwide market for all "ADHD medications" in 2003 as being USD 2.4 billion. [20]

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

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

<u>v) The drugs are extremely harmful</u>

Documented and typical side-effects of stimulant drugs include:

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

- Cardiac complications (rarely)

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

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

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

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

> ASK FRANK: Speed (the 'street' name for amphetamines including dexamphetamine) can lead to agitation, panics or even a psychotic episode.
>
> RITALIN: Treatment emergent psychotic or manic symptoms, e. g., hallucinations, delusional thinking, or mania in children and adolescents without a prior history of psychotic illness or mania can be caused by stimulants at usual doses.
>
> ASK FRANK: Depending on how much you’ve taken, it can be difficult to relax or sleep.
>
> RITALIN: Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening.
>
> ASK FRANK: Speed [amphetamine] puts a strain on your heart, so it's definitely not advisable for people with high blood pressure or a heart condition – users have died from overdoses.
>
> RITALIN: Sudden death has been reported in association with CNS stimulant treatment at usual doses in children and adolescents with structural cardiac abnormalities or other serious heart problems
>
> ASK FRANK: Speed makes people feel wide awake, excited and chatty
>
> RITALIN: Ritalin is a mild central nervous system stimulant.

Both Novartis, the manufacturer of Ritalin, and the UK government are describing the adverse effects of taking the same type of drugs. The advice given by the British government about the use

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

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

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

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

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

Strattera is a relatively new drug used to “treat” “ADHD”. It has been on the market since 2004. The chemical substance is atomoxetine hydrochloride. Atomoxetine hydrochloride is not a stimulant. The selling point of Strattera therefore is that the risk of its escaping onto the black market, for illegal use, is reduced. From 2004 to 2013 its use by the NHS in England has grown by about 700%. During this period prescriptions for methylphenidate have risen by about 230%. Prescriptions for dexamfetamine have fallen by about 30%. Strattera is thus gaining market share. Like the discovery of the beneficial effects of stimulants on concentration in “disruptive children”, the applicability of atomoxetine

hydrochloride to treat “ADHD” was an accidental discovery. The drug failed as an anti-depressant and was re-purposed to “treat ADHD” “in children”. [2] Strattera's claim to be suitable “for ADHD” rests on the fact that a number of studies managed to show that ADHD “symptoms” were reduced by the substance. The authors of the NICE ADHD Guideline found 17 studies for Strattera which met their inclusion criteria. Of these, 16 were funded by Lilly, the company who makes Strattera. There was no funding data for the other one. [29] This then is not a medical-scientific process of discovery but a marketing launch. Indeed because there is no “medical or neurological cause” “for ADHD” there cannot be a medical-scientific process of research leading to a treatment, as there is, for instance for the HIV virus. All they can do is give the drugs to young people and count the reduction in symptoms, that is the reduction in the “disruptive” behaviours which constitute ADHD. A*nything* which reduces the “symptoms” / altered the behaviours would pass this test.

The average length of the 17 Strattera studies identified by NICE was 83 days. [29] ADHD drug studies tend to be in the short-term but ADHD drugs are typically prescribed in the long-term. This means that negative effects which occur over the long term are unlikely to have been considered.

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

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

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

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

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

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

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

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

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

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

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

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

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

In general terms the manufacturer of Strattera advises:

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

Which hardly sounds like much fun for a young person.

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

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

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

vi) It is about authority not medicine or science

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

> His school told Andrea Ruben faced exclusion unless he took drugs to control his behaviour. [40]

Another case is reported by the Guardian:

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

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

In the ADHD world the focus in on making the behaviour of the young person align with the expectations of adults. It is taken for granted that this is a valid goal. The shift to a “clinical” interpretation and framing serves to avoid any requirement for those on the side of authority to change so as to meet the *actual* needs of the young person, as they are. Once a “child” is said to “have ADHD” then a formulaic “treatment” is ordered. This may be drugging or a behavioural programme. Even if the intervention is a parenting programme this is still constructed as helping the parents to manage their “ADHD child's” behaviour, for example: “to optimise parenting skills to meet the above-average parenting needs of children and young people with ADHD”. [42] This sounds sympathetic but is based on the usual reification; “the children and young people with ADHD”. Everything is predicated on the problematised “child”. Whatever it is it is *his* problem. This is still an objectification of

the young person. The authentic relationship between adult and young person wherein actual needs might be met is obliterated under this manipulative framework. This location the problem *in* the child resembles moral narratives about “children” from the Victorian era. The “clinical” picture is superimposed on this essentially moral, and always potentially punitive, practice. The moral nature of psychiatry is expressed by Foucault:

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

An interesting example of the moral themes underlying the ADHD narrative is provided by Singh 2007 with a paper “Taking Methylphenidate for ADHD. Clinical Implications of Ethical Concepts: Moral Self-Understandings in Children”. [44] Sing 2007 is a confusing paper. Singh appears to believe that she is taking issue with a certain strand in dialogues which are critical of the ADHD drugging programme. These strands emphasise the “naturalness” of the individual and, apparently, promote an ethics of personal authenticity. Singh attributes the “natural” character of the “child” position to a writer D. Brock. Singh associates the point of view which emphasises the “uniqueness and individuality” of the self as a reference point for morality to an academic philosopher Charles Taylor. Singh believes that these views are used to develop an argument that ADHD drugging should not be allowed because it undermines these “innate dimensions” of the person. Singh however appears to have completely misunderstood the philosopher Charles Taylor. Singh writes that:

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

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

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

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

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

confused blend is then added a strong sense of “morality” which appears to be Dr Singh's own. In this morality being “bad” equates with “doing something wrong”. “Doing something wrong” appears to mean doing something for which a “child” might be told off by a parent or teacher.

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

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

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

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

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

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

Here is Singh explaining the “natural self” position:

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

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

> These assumptions of a core aspect of the self can be viewed as theoretically analogous to arguments for a genetic basis to personality and temperament in the field of clinical genetics. However, the genetic research on personality strongly suggests a gene–environment interaction: Genetic predispositions to temperament outcomes or to

> psychopathology can be triggered by environmental stressors; or the environment can have a protective function (Caspi et al., 2002). [44]

and

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

Dr Singh believes that in the above she has justified using the self-statements she manages to produce from her sample of 8-12 year olds as true characterisations of the actual nature of their "authentic" selves. However; the problem here is that "authentic self" is a philosophical concept. Singh may believe she can map ("analogous") ideas about personality from the fields of social psychology and genetics to this concept but she may be alone in this belief. She hasn't demonstrated or proved this mapping. On the whole the concepts belong in different types of discuses and cannot be so crudely mapped. In philosophy ideas about an authentic self are not "assumptions" as Dr Singh terms them. Or; if she wants to assert that philosophy is just non-empirical chatter which makes "assumptions" then she needs to demonstrate that. But, if she could do this, she would, to borrow a phrase from Charles Taylor, self-destruct her own argument which depends on the existence of an "authentic self". This is all confusion and Dr Singh has not established that persistently made self-statements are descriptive of the "authentic self" of a person. She could not because she is mixing discourses. Like most positivists Dr Singh has leapt from empirical science into the field of philosophical discourse, *collapsing* the philosophical concepts in the way. The starting point of positivism; that only empirically established facts have any meaning, is used to crudely destroy other discourses. Dr Singh can get a group of 8-12 year olds to say whatever she wants, but this is not a discussion about what constitutes the "authentic self" of a person. Nor even what constitutes the "authentic self" of a "child with ADHD".

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

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

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

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

> In order to adequately explore issues of gender, future research may need to oversample girls. [44]

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

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

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

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

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

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

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

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

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

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

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

> Another child, Mark, elaborates a similar narrative about the relationship of medication

to his good/bad self:

> Interviewer: If you had taken your tablets and you hit someone and hurt them on purpose, would you be a bad person?
> Mark: Mmmm. It would be a bit of both.
> Interviewer: Bit of both . . . in what way?
> Mark: Bad, and good then.
> Interviewer: Oh, cos you said it makes you feel good when you hit someone?

and

> You're saying that there's a bad part of you that the tablets can't make good?

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

In the dialogue above we can note that the term "bad person" is introduced by the interviewer to Mark. Mark accepts the term. The interviewer then follows up the advantage with "You're saying that there's a bad part of you....". The concept of "real you" is similarly introduced to Mark by the interviewer. This is not looking very "empirical". And indeed if this really was an "empirical" study about whether young people understand methylphenidate drugging as undermining their "authentic selves" it would appear that Mark has given a completely clear answer:

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

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

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

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

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

> Interviewer: When you've taken your tablets and you've done something wrong...
> Simon: Yeah?
> Interviewer: How do you feel inside?
> Simon: Bad.
> Interviewer: But when you haven't taken your tablets, and you do something

wrong...
Simon: [interrupts] I feel good about it! [emphatic]
Interviewer: Do you like that feeling of feeling good inside?
Simon: Yeah. Wait. What do you mean by 'good'? Do you mean doing something bad and I feel good inside?
Interviewer: Yeah.
Simon: No, I don't like that. I feel bad about myself. [44]

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

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

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

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

Tommy: That's me acting like a crazy monkey.
Interviewer: You're acting like a crazy monkey?
Tommy: Yeah, like this . . . ahha ahhh [monkey sounds].
Interviewer: So is that when you've taken your tablets, or when you haven't taken your tablets?
Tommy: Haven't. That's really fast.
Interviewer: And how does it feel to be this crazy monkey?
Tommy: Really quick.
Interviewer: Really quick.
Tommy: Ohhhahhha. Very movable, like that.
[Pause 6 seconds]
Tommy: Hmm. Do you feel like you can control this crazy monkey that you've become?

Tommy: Not quite . . . well, you can't really tell . . . if I'm going to be able to control it. Cos sometimes you can control it, and sometimes you can't. Sometimes when my mother says, 'Stop!' I can do it, and sometimes when my mom says, 'Stop!' I carry on doing it.
Interviewer: Why do you think that is, that you can stop sometimes and not others?
Tommy: Cos I think your brain sends messages inside the body.
Interviewer: So your brain says . . .?
Tommy: Carry on because I don't have any tablets.
Interviewer: Oh, your brain says, 'Carry on because you don't have any tablets'.
Tommy: Yes.
Interviewer: Do the tablets stop your brain from making you behave that way?
Tommy:Yes.

Singh interprets this as Tommy attributing to methylphenidate the power to give him self-control. An alternative interpretation would understand this in terms of drug dependency. Recall that Tommy may have been told by his parents that he is being given the drugs to control his "naughtiness". In as much as he has come to believe that being "naughty" or "not naughty" is something which is controlled by being on or off tablet he may allow himself to be "naughty" when he is "off tablet". Thus he develops a dependency on drugs. Telling young people that it takes drugs to stop them being "naughty" will discourage them from learning how not to be "naughty" without drugs.

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

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

But she isn't completely sure about this:

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

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

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

and

> These binary representations of the self/behaviour on and off medication make up one level of discussion in interviews with children. These representations are notable in that they appear to demonstrate a lack of cognitive sophistication in these children; the tendency to structure descriptions of the self and behaviour as unintegrated opposites is identified with the cognitive skills of 5–7-year-olds (Griffin, 1992; Harter, 1999). However, these representations should probably not be taken to reflect cognitive immaturity in these children; rather, they are better explained by the structure of the question children were responding to when taking their photographs... [44]

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

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

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

But, if your case depends on an explanation about “children's self-understandings” surely it does

matter if what the “children” say in the interviews is what they think, or what their parents have told them?

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

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

This reporting by young people of the negative effects methylphenidate has on them does not seem to influence the “potential” clinical recommendations formed from the research which recommend more consistent dosing (extending “dosing” at week-ends and holidays and not just during the school-week) [44]. Approaches such as this while appearing to “consult” young people are not really doing so seriously. The young people are not asked the meaningful question “would you like to stop taking these tablets now?”, as a real question where if they answered “yes” then they would stop being given the drugs. They are being asked for their views about a given situation, organised by all the adults around them which they know is going to continue whatever they say. Like all young people being abused by adults these young people will give adaptive answers. Such “consultation” exercises with “ADHD children” consistently produce the same answer. The “children” approve of their “medication” and voice a quiet, permitted, protest about the “side-effects”. That won't help them because clinicians like Dr Singh have already decided that methylphenidate has a “tolerable side-effect profile”. [44]

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

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

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

that through improving short-term attentiveness methylphenidate can help young people reflect on their behaviour in a more focussed way. Thus, perhaps, they really do start to develop more appropriate thinking about being “naughty”. (That is, thinking which is in line with Dr Singh's moral system). But even if this is accepted it does not follow that “medicating” is vindicated. Behavioural interventions (or indeed other types of intervention or response) may well achieve the same result without any of the side-effects that young people report from “medication”.

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

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

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

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

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

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

> These medications are not a permanent cure for ADHD, but they can help someone with the condition concentrate better, be less impulsive, feel calmer, and learn and practise new skills. [48]

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

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

The drugs can “reduce symptoms”. The “symptoms” are behaviours which are “disruptive and

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

vii) Summary

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

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

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

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

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

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Section 5.9

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29. *Ibid*. Appendix 17.5

https://www.nice.org.uk/guidance/cg72/evidence

30. Lilly Corporation Information sheet about Strattera

http://pi.lilly.com/us/strattera-pi.pdf

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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.

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