resulting children to be weak, sickly, stupid and cancer-ridden. But they aren't. A review by Tournaye (2009) concluded that the absolute risk of genetic anomalies from older paternity is low, and that "there is no clear association between adverse health outcome and paternal age".

And finally, any genetics which ignores the context of changing environmental factors is half-baked. A hugely important factor is dental mercury. In Chapter 3 here I show how just about all of this autism, schizophrenia, manic depressive, and more, can be accounted for as the consequence of the introduction of dental amalgam in the 19th century followed by its "improvement" from the 1970s with the even worse non-gamma-2 amalgams. Get rid of that dental mercury and just about all this disability caused thereby disappears, nothing whatsoever to do with genes being harmful per se, but merely genes conceivably making a person vulnerable to an abnormal environ-ment which would not be there anyway if fewer "distinguished experts" were liars.

The bottom line here is that the proper understanding of the genome and mutations does not at all correspond with the still-predominant assumptions of the outdated simplistic model. And too many people in autism genetics research are assuming that it does.

[P.S.: Ruben Arslan has commented: "You refer to me as "poor old Prof Arslan". I'm neither a Prof nor a Dr, I'm still a PhD student. The "poor" is quite right though." However, the very next month I noticed some other research of his reported in the *New Scientist*. Some "student".

He also informed me that that Arslan et al. 2014 "wrong" result has since been confirmed by "a much bigger study (D'Onofrio et al., 2014)...."]

(The main text of this chapter continues back at page 60.)

Chapter 3 starts on next page →

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3

A suppressed report of millions of (non-autistic) victims

"Academic journals and societies show an auto-immune response to information that should be the life-blood of medicine."
– Prof. David Healy, author of *Pharmageddon*

"Your paper is important"
– mercury expert Dr med. Joachim Mutter

Before reading this chapter I recommend that you read the first two chapters of this book. Otherwise you may come to it with considerable misconceptions which could make for difficult and unproductive reading here.

The main content of this chapter is a scientific paper. I wrote it with the intention of it being accepted in a scientific journal, and so you might find it rather turgid reading and with too many of those citations such as (Authorname, 2012) intruding into my florid prose. On the other hand one journal editor condemned it for (supposedly) appearing to be written like a newspaper article, so maybe there's hope for non-academic readers nevertheless.

You may be wondering whether you can have the competence to make any useful judgement of the soundness or credibility of this article. Wouldn't the experts perhaps point out all manner of hidden things wrong with it? But I am providing you with a special resource here. In the next two chapters, you can see the world's top experts telling me (off the record) the reasons why this article is such rubbish that you shouldn't even be informed of its existence anyway. I suggest that you study those critiques and my rejoinders to them, and (as is always necessary eventually) then decide for yourself who if anyone has the more credibility. I can't print the re-rejoinders from these experts because none have replied back. Perhaps you could write to these journals yourself to ask them why you shouldn't be persuaded by what I said in my own replies.

Scientific papers normally end with a list of the references cited. In this book I will transfer this paper's reference list into the list at the end of the book. But this article is unusual in that it contains an appendix which itself contains three further lists of

references. I will leave those in place just as they were present in the original documents contained in that appendix. Other than that, what follows after this paragraph is the most updated version of the manuscript I have sent to now eighteen journals. It is usual for scientific papers to begin with a summary called an "abstract". This gives an overview for those who don't already have the full text, but may be hard as a non-specialist to follow until you have read that full text, and you shouldn't let yourself get bogged down by this one here. Also this chapter contains some graphs of disability epidemiology. If you are not already a wizard with such graphs, you may find it useful to jump forward to the section of Chapter 6 which discusses some misuses and abuses of similar sorts of graphs. Lastly, the "p<" values stated herein indicate the probability of obtaining that result due to random chance.

(NOTE: AT NO POINT HAVE I EVER SAID THAT *ALL* AUTISM IS CAUSED BY MERCURY OR AMALGAMS – See Chapter 7!)

Autism, adult disability, and 'workshy': Major epidemics being caused by non-gamma-2 dental amalgams

Robin P Clarke

Abstract: It is unknown to most people that the dental amalgams which have been used as standard in recent decades, namely non-gamma-2 dental amalgams, have been substantially unlike those used before the 1970s, in that they constantly emit 20 to 50 times more mercury vapor than the older types. This is the first-ever study of health consequences of non-gamma-2. Following the changeover to non-gamma-2 amalgams, there promptly began a tenfoldish increase of autism, a tenfoldish change of ratio between late onset and early onset, a change from mainly genetic to mainly environmental, and a change from lifelong incurable to sometimes clearly recoverable. Exactly simultaneously there occurred a fourfoldish increase of claims for adult disability in the UK, with disabilities all or mostly of the nature that would be expected from chronic mercury poisoning (including mental disabilities and neurological disabilities). And similarly in the US. These timings cannot be dismissed as coincidence because there are no credible alternative explanations for the increases. Data strongly suggests that non-gamma-2 amalgams are currently by far the main cause of chronic disability in the UK, US, and other such countries, with about 10% of the UK working-age population disabled thereby.

An experiment on millions of dis-informed subjects

Dental amalgams in patients' teeth constantly emit mercury vapor, and that vapor is easily measureable. This has been known for decades as indicated in at least 18 published studies (Berglund, 1993; Berglund et al., 1988, Boyer, 1988; Brune et al., 1983; Clarkson et al., 1988; Ferracane et al., 1995; Mackert, 1987; Mahler et al., 1994; Moberg, 1985a, 1985b; Olsson et al., 1989; Olsson and Bergman, 1987; Patterson et al., 1985; Psarras et al., 1994; Svare et al., 1981; Vimy and Lorscheider, 1985a, 1985b, 1990).

And yet in stark contradiction of all this clearly established basic science, the UK's Chief Dental Officer (2009) has publicly asserted, as some supposed fact, that dental amalgams do *not* constantly emit any mercury vapor (or in his second thoughts on being challenged, at least "not measureably").

Such mercury vapor has been recognised for centuries as one of the most toxic of substances, causing various mental, neurological and physical disabilities. For more than a hundred years prior to the 1970s, strong condemnations were regularly issued against the use of amalgam in dentistry. These warnings were consistently ignored by health authorities, and dismissed with claims that there was no real evidence of harmfulness.

Much further evidence of harmfulness of dental amalgam has come to light in recent decades (Mutter, 2011; Hanson, 2004; Homme et al., 2014), not least in thousands of cases of improvement following amalgam removal which cannot be dismissed as merely anecdotal or placebo. Some relatively large-scale trials have been asserted to show amalgam safety, but they have been substantially flawed and in at least one case in reality showed harmfulness rather than safety (as explained by Mutter, 2011 and Homme et al., 2014).

In the 1970s a new type of dental amalgam was introduced as the new standard, partly on the basis that it was very much more durable, with far less tendency to corroding and crumbling. This new type was called non-gamma-2.

These non-gamma-2 dental amalgams constantly emit 20 to 50 times more of the toxic mercury vapor than the older types (Berglund, 1993; Boyer, 1988; Brune et al., 1983; Ferracane et al., 1995; Mahler et al., 1994; Moberg, 1985a, 1985b; Psarras et al., 1994). The amalgam constantly emitting this neurotoxic mercury vapor is located in a person's mouth, less than two inches from their brain, and in the pathway to the lungs (where 80% is absorbed at each inhalation). Any notion that the levels of mercury vapor caused by amalgams are very low has to be put in the context of the general outdoor levels being many times lower still at around 0.002 mcg/m3.

No safety testing was undertaken before or after it was introduced. Patients and the public in general have still not been informed of the change, let alone of the increased levels of mercury involved. No informed consent has been sought, and no warnings have been given of any possible harmfulness. Indeed, throughout the US it was actually made illegal for dentists to issue such warnings, and Hal Huggins and other dentists were struck off the register of practitioners for doing so.

In the UK, a number of untruths were adopted by the NHS and DH such as to prevent people being diagnosed with mercury toxicity and to thereby further reduce any concern about risk. The following untruths have been identified by the author, but it is unlikely that they have been the only ones.

1. Untrue assertion that "Chronic mercury poisoning is highly unlikely to present in a psychiatric setting".
2. Use of proven useless urine tests for supposed (dis-) diagnosis of chronic mercury poisoning.
3. Use of proven useless blood tests for supposed (dis-) diagnosis of chronic mercury poisoning.
4. Chief Dental Officer's untrue assertion that "no mercury vapor" emits from amalgams, or alternatively "not measureably".
5. Chief Dental Officer's untrue denial that amalgams are the main source of mercury in the body.
6. NHS Chief Executive's re-insistence on the untruth that dentists have capability for mercury diagnosis whereas doctors do not.

The existence of these untruths is authenticated via my Freedom of Information requests as documented partly in an Appendix hereto and more fully via http://tinyurl.com/dentmerc

Dates of introduction and usage

Non-gamma-2 amalgams are very much more durable than the previous types. Consequently, declining rates of amalgam installation would conceal an increase of prevalence of the amalgams in patients' mouths. And it is here expected that the key variable would be that rising prevalence rather than the declining rate of installations and replacements.

A number of US patents for non-gamma-2 were granted in the mid-1970s. The famous US dentist Hal Huggins states that the changeover to "high copper", i.e. non-gamma-2, occurred in 1976. In 1986 the ISO standard was changed retrospectively to incorporate them. The non-gamma-2 amalgams took over in the period 1975-79 in Denmark (Hansen et al., 1993). In Germany the use of the earlier types was banned in 1992, making the non-gamma-2 the only option. And according to the manufacturer's product sheet,

Dispersalloy is the most widely used amalgam with over 25 years of proven performance, i.e., since before 1979, but perhaps after their 1974 patent no. 3841860.

I have been unable to obtain any numerical data on usage or total prevalence of non-gamma-2 in people's mouths. The DH have told me they have no such records. And NHS dental records have not recorded the types of amalgam used. It is unlikely that any better information is available in other countries. But we can very reasonably assume that the overall prevalence of non-gamma-2 will have gradually, progressively increased in the decades following its introduction.

My epidemiological investigations

Having become aware of the changeover to non-gamma-2 amalgams, I decided to look to see if there might be epidemiological evidence of any consequences. It appears that no-one has ever done this before.

In respect of the following accounts it is important to understand that I have not cherry-picked selected data to prove any point, but instead have used all the best data readily available to me.

To avoid undue length here, the reader is referred to consult prior reviews of substantial important other data pointing to similar conclusions as those here, including Hanson (2004), Mutter (2011), Geier et al. (2010), Homme et al. (2014), and others not specifically cited.

Is mercury involved in causation of autism?

Before presenting the epidemiological findings it will be useful to first show the context of existing evidence from clinical studies on this question.

A number of reviews have suggested there is persuasive evidence that mercury is importantly involved in causing of autism (Geier et al., 2010; Bernard et al., 2001; DeSoto and Hitlan, 2010; Kern et al., 2012). And yet the evidence can be shown to be far more decisive than any of these suggest, and indeed beyond all reasonable doubt.

In any combinatory review of studies it is necessary firstly to rule out those which lack a sound rationale. A number of studies have used blood mercury or urine mercury as criterion measures, and yet it has been known for decades that these lack merit as indicators of chronic mercury toxicity. Indeed, the most prominent such study, Hertz-Picciotto et al. (2010), stated in its second-last

sentence that: "This report did not address the role of prenatal or early-life Hg exposure in the etiology of autism" [i.e., the study could not provide any evidence against causation by mercury].

Another danger in meta-reviewing of studies is the merging together of data which should be kept separate. In respect of mercury in autistics' hair, the most enlightening study is that of Majewska et al. (2010). They found that in younger children autism was associated with markedly decreased hair mercury ($p<0.01$), whereas in older children autism was associated with markedly increased hair mercury ($p<0.01$). If they had just lumped all their results together they would have got an entirely unwarranted "no difference" non-result instead. Viewed in the light of Majewska et al, all or most of the other hair mercury studies fall into a coherent pattern. There are several which have smudged together the different ages and therefrom invalidly declared non-results. Meanwhile others strongly reinforce the notion that there are real effects.

Holmes et al. (2003) obtained an eightfold difference of mercury in hair, with significance level of 1 in 250,000 ($p<0.000004$). Some commenters dismiss that study on a basis that it was done by opponents of mercury, and "therefore" their results may have been biased or fraudulent. But one would have expected any bias or fraud to result in a finding that hair mercury was increased in autistics, as that would have been in accordance with the standard rationale for diagnosing toxin exposure from increased hair measurements. They found instead 8-fold reduced levels, which strongly suggests that they were instead acting competently and honestly. Their rationalising notion of paradoxical reductions of measurements in mercury toxic subjects has since been supported by much other evidence that mercury sometimes impairs its own excretion.

A study in India (Lakhshmi Priya and Geetha, 2010) found 8-fold increased hair mercury ($p<0.001$). Another in Kuwait (Fido and Al-Saad, 2005) found 15-fold increased hair mercury ($p<0.001$).

Bradstreet et al. (2003) found that a challenge test with the chelating agent DMSA caused a release of mercury 3.15 times greater in autistic cases than in controls ($p<0.0002$). That is a 1 in 5000 probability that that excess mercury was just a fluke.

The probability of just these results listed above being all due to mere chance is 1/5000 x 1/250000 x 1/100 x 1/100 x 1/1000 x 1/1000, that is one in 12,500,000,000,000,000,000, vastly beyond the standard of proof ever required in any criminal prosecution.

And far more than one negative result is required to call into question one significantly positive result. There are far more ways of making a "negative" car that does not move than of making a

"positive" one that does. I and thousands of others have lived in the UK for many years and never seen the Queen in all that time, and yet that does not constitute significant grounds for dismissing the testimony of those who claim she has existed. If there were in reality no mercury-autism connection there should be a huge pile of "no-difference-found" results among which these high-significance results would be a small minority. But there is no such pile of null results to speak against the mercury-autism connection.

One could seek to interpret all those results with a notion that there could be an unknown factor which both causes autism and also harmlessly causes mercury to vary in hair and other tissues. But that notion is brought into question by the extensive commonalities between autism and mercury toxification (Kern et al., 2012). And it is completely demolished by the observations of the Autism Research Institute which has for decades been surveying the effectiveness of many potential treatments for autism. Of more than 80 treatments tested, the ARI has found that one of the most effective has been removal of mercury by careful chelation. And Blaucok-Busch et al. (2012) obtained highly-significant behavioral improvements even with the rather poor Hg chelator DMSA ($p<0.001$; $p<0.001$; $p<0.001$).

Meanwhile, three studies have been promoted as supposedly disproving any mercury-autism thesis. The study by Ip et al. (2004) was shown to be riddled with arithmetical errors, and in reality indicated that there was indeed a mercury connection (DeSoto and Hitlan, 2010). Likewise Soden et al. (2007) actually proved the opposite (DeSoto and Hitlan, 2010). And Hertz-Picciotto et al. (2010) stated in their own second-last sentence that their study did not address causation of autism by prenatal or early-life mercury exposure. Such falsely proclaimed studies are all that stands in supposed defiance of that astronomically large number calculated above. There is even more evidence that merits mention here but it would be superfluous. We can resolutely conclude that mercury is now a major cause of autism. [Updates: Autism association with prenatal SSRI use (Harrington et al., 2014) = amalgam causes both depression of mother and autism of baby. Widespread reports of seizures in 1/3 of autistics = perinatal mercury causes both autism and seizures (Szasz et al., 2002; Klinghardt, 1998).]

Increased autism?

In academic papers and elsewhere, certain myths about autism are constantly portrayed as self-evident truths, so they must be addressed here. Firstly, the human race does not divide into those "with" autism and those "without" it, or those "on the spectrum" and

those "not on the spectrum". Rather, there is a continuum of variation in the extent to which individuals are more or less autistic (in varying ways). Secondly, there is no scientific basis for a distinction between autism and Asperger's. It was merely a historical accident that Kanner and Asperger made simultaneous rediscoveries of the syndrome described by JL Down in 1887. Thirdly, there is no scientific basis for the routine references to autism as a "disorder". Autism can be severely disabling, is often terribly distressing, and may often be *a consequence of* a disorder (such as maternal infection), but rather than a disorder it is properly considered to be just atypicality (as is genius). [This is now more fully discussed in Chapter 2.]

Some researchers with decades of direct experience, such as Bernard Rimland and Lisa Blakemore-Brown, have been of the view that there has been a substantial increase of autistic behaviors, and not just increase of diagnoses.

[Update for this book: Significant further discussion of the increase "controversy" is contained in Chapter 2 in the section "The autism increase controversy" (page 68), just before the appendix to that chapter, and also majorly in Chapter 12 and pages 188-189.]

The NHS has published a report claiming to show that there has not been any increase, by supposedly showing the prevalence of autism among older adults to be the same as in children (Brugha, 2011). The report detailed the elaborate measures taken to ensure reliability of the autism assessments. And yet it gave no details at all of any measures taken to ensure the validity, that is the (infinitely more important) comparability of the diagnostic procedures as applied to adults relative to applied to children. The reason there were no such details is because there was no way of establishing such validity. And in absence thereof, such a study proves nothing about changing prevalence of autism. I myself have direct knowledge of two older persons given baseless diagnoses of autism by this same NHS that proclaims as expertise the untruths listed on a preceding page here.

The Autism Research Institute has a uniquely extensive historical database of cases. Figure 1 [here 3.1] is my re-plot of a graph published by the Autism Research Institute of its own records. Figure 2 shows my extraction from Figure 1 of the time-series of case ratios between late and early onset. Before 1980, onset at birth was twice as frequent as onset in the second year (i.e. regressive autism), whereas after 1990 the later onset rose to become five times more frequent than the onset at birth. The switch-over began at the end of the 1970s and was well under way by 1990. It closely related with the apparent increase of autism illustrated in Figure 3 and

elsewhere. Figure 3 shows the data from the California DDS 2003 report (2003), with the earlier 1999 report (1999) (1998 data) re-calculated to the same basis. [Note added to book chapter version:

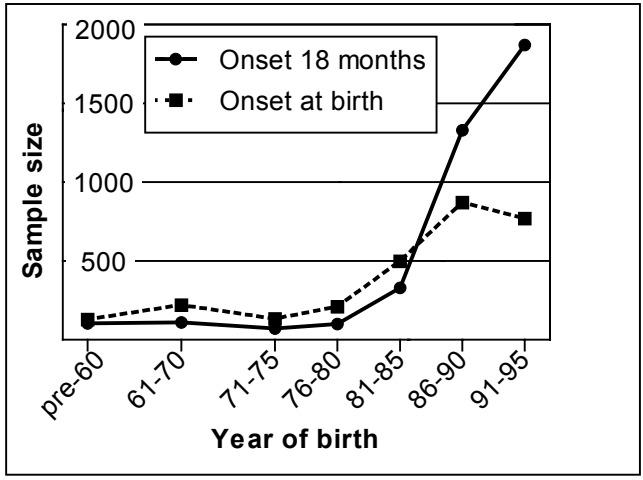

Fig. 3.1. "U.S. Cases: Autistic children who behaved normally before 18 months vs. those with no normal period." From Rimland (2000) (replotted)

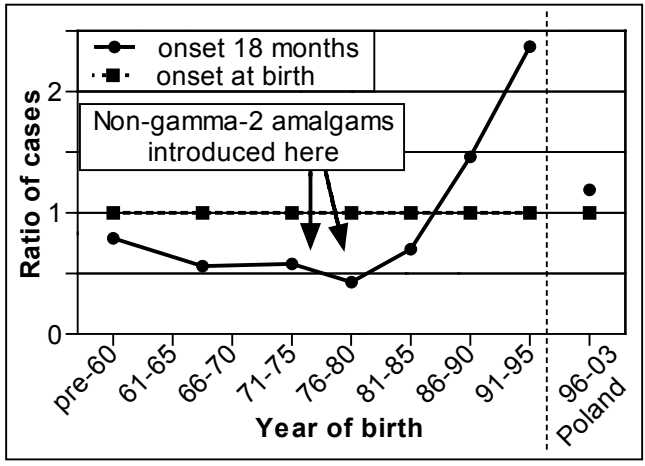

Fig. 3.2. Data from Figure 1 here used to show the changing ratio of cases in respect of age-of-onset. A further datum is from Mrozek-Budzyn et al. (2009) p.109.

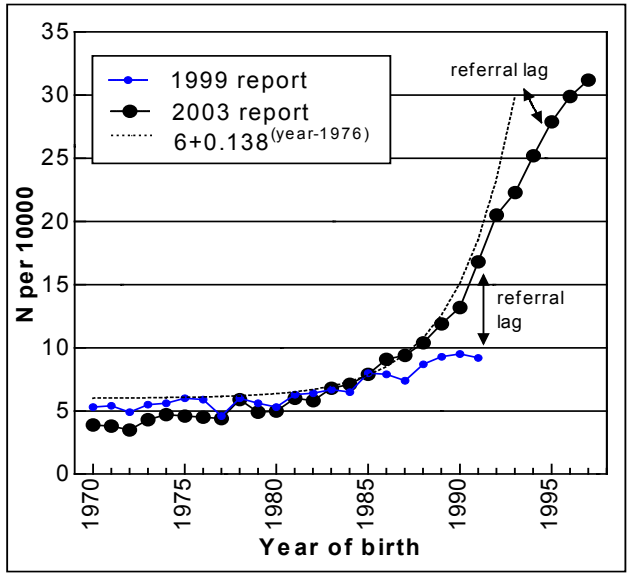

Fig. 3.3. Autism enrolment in California.

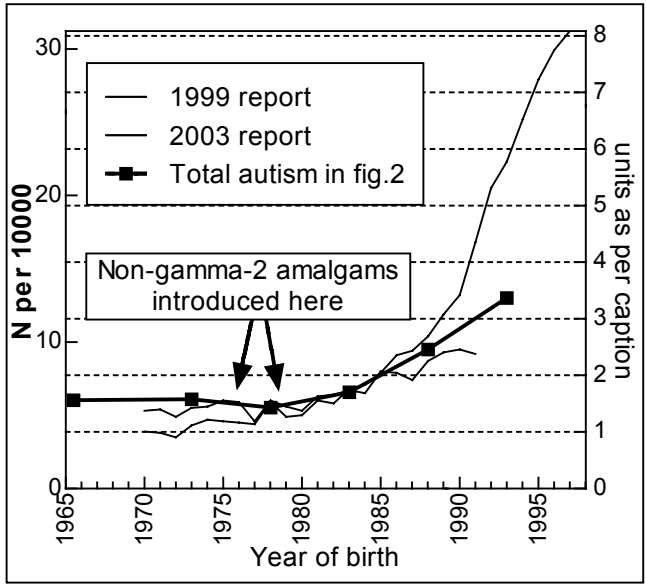

Fig. 3.4. Concurrence of California data of Figure 3.3 with total autism implied in Figure 3.2 if onset at birth is assumed to have constant incidence of one unit.

Figure 3 also shows what mathematicians call an *exponential increase* curve; basically it gets increasingly steeper exactly in proportion to the higher it gets.]

In Figure 4 I have added together the two series of Figure 2 such as to give a nominal "total autism" curve based on an assumption that onset at birth has had constant prevalence during those years, and that early onset cases plus late onset cases equals total autism. Figure 4 shows that the increase curves of Figure 3 peculiarly coincided in time with the ratio-change curve of Figure 2. This enables substantial confidence that the conceptually independent Figures 2 and 3 are tracking exactly the same causal phenomenon.

The late-onset, regressive autism is much more difficult to overlook than the at-birth autism, as parents are baffled by the regression of their children. Any under-awareness would not have been concentrated on those late-onset cases. And yet it is those which have increased about tenfold, not the more overlookable early-onset. So this data argues against interpretation in terms of mere changing of awareness or diagnostic thresholds. And it cannot be dismissed as due to demise of the diagnosis of "childhood schizophrenia", because ARI's survey questionnaire asked about age of onset rather than presumed about it, and indeed the ARI was neutrally called the "Institute for Child Behavior Research" until 1991.

These curves strongly suggest that the autism increase was caused by something that started having an effect on children around the end of the 1970s and also caused a tenfold change of ratio of late-onset cases relative to early onset.

An overview of autism trends in the US and UK found essentially the same trends of increase in both areas and in respect of both autism and "autism spectrum disorders" (Blaxill, 2004). Information about other capitalist countries has been less systematic, but generally similar trends appear to prevail. In respect of Sweden, Gillberg's three prevalence studies in Gothenburg (Stehr-Green et al., 2003) could have been plotted into Figure 3, but they would have collided impressively with the California data. The data of Denmark is rich in potential for confusion but the careful analysis by Goldman and Yazbak (2004) shows an increase from at least about 1987 onwards. Likewise, the general observation in the other countries is that there has been an increase in recent decades. And the age-of-onset data in Figure 2 follows the same pattern too. (The notion of Bernard (2003) that autism decreased in Denmark after removal of mercury from vaccines is misfounded for various reasons partly explained by Hviid (2004).)

So there is here a simple thing to be explained, seemingly beginning around the end of the 1970s.

Some years ago there was published "A theory of general impairment of gene-expression manifesting as autism" (the antiinnatia theory). It remains unchallenged in reasoning and evidence, and unrivalled as the only comprehensive fully satisfactory explanation of the supposed mystery of autism. Martha Herbert has recently been arguing that autism is not a brain/behavior condition but rather "whole body", and also not essentially genetic or developmental and fixed. But the antiinnatia theory already embodied all those notions decades ago.

The theory also specified circumstances in which autism would change from a mainly genetic condition to a mainly environmental one. Autism has now indeed markedly changed to a mainly environmental causation (Hallmayer et al., 2011).

That antiinnatia theory paper made no mention of mercury or an increase of autism (which was only vaguely becoming apparent at the time of writing it). But it did explicitly explain why molecules which randomly, dose-dependently bind to DNA and thereby reduce gene-expression would thereby cause autism. Mercury is now known to do exactly that binding and reducing at levels far below those producing other toxic effects (Ariza et al., 1994; Goyer, 1991; Rodgers et al., 2001; Walter and Luck, 1977).

A preceding section here has shown the decisive recent evidence of major involvement of mercury in many autistic cases. And thimerosal in vaccines cannot have been a main source of that mercury, for reasons explained in [Chapter 6]. So the question arises of:

where else is the source of the mercury that is now so strongly associated with most autism.

An update review of the antiinnatia theory was subsequently written, and showed confirmation of various peculiar predictions [Update: including Clarke (2015)], and explained the amalgam-autism causation more fully. But almost all medical researchers have a false presumption about theories, whereby "skepticism" (in reality a prejudice against new ideas) is supposedly a characteristic of intellectual superiority (Eysenck, 1995). And "peer review" systems block from effective publication any ideas that are more than routinely original (Eysenck and Eysenck, 1992).

Because readers are deprived of that update review I will outline here just a few of its important points. (1) Many mothers keep their infants close by at all times, and many people keep their homes very unventilated, even installing draught-proofing. The new prediction that autism would be associated with lack of ventilation

(of the mercury vapor breathed out by parents or carers then inhaled by infants) has already found significant accidental confirmation (Waldman et al., 2008). (2) The antiinnatia theory points to causation not so much like an overdose "hammer-blow" but rather more like a sustained suppression of genetic data, and thus the every-day inhalation of mercury would be much more impactful than occasional large injections. (3) The tenfold change to predominantly later onset is explained by gradual accumulation when infants regularly inhale the vapor. (4) Any persons who dismiss the antiinnatia theory must logically be supporting one of a handful of utterly absurd alternatives, and this author requests that such "skeptics" kindly state which ones they find so credible: (i) "anti-innatia factors don't tend to produce biological advantageousness"; (ii) "they don't exist anyway despite their experimental demonstration" (genuine flat-earthers will prefer that one); (iii) "they would not tend to become excessive"; (iv) "excess would not manifest as autism".

Some studies have found positive associations between maternal dental amalgams and autism (Holmes et al., 2003; Geier et al., 2009). There have also been some seemingly conflicting findings, such as Adams et al. (2008) compared to Holmes et al. (2003). But rather than concluding from these that the whole mercury or amalgam theories are unsound, or that there has been falsification or error, we may better understand them as reflecting a fact that autism is far from being simply "a novel form of mercury poisoning", and instead other factors impact in ways not yet known. Even the causation of autism by amalgam vapor alone would be complicated by variables of ventilation, parental habits, galvanic contacts in the mouth, genetics and epigenetics, intake of protective selenium, and other intakes and exposures. That complexity could explain why small cross-sectional studies have given inconsistent results. And meanwhile the time-series data shown in the charts here reflects varying levels of non-gamma-2 applied to whole populations, such that all those confounding variables are evened out, which explains why they show a clear association with the growing prevalence of the non-gamma-2 in adults' mouths.

Increased adult disability?

In 2010 I heard on BBC Radio a claim by a government minister that "There certainly hasn't been a threefold increase of disability". This suggested to me that perhaps there had indeed been an increase of adult disabilities, threefold or even greater.

On investigating this possibility, the most extensive data I could obtain was a chart on page 9 of pathways-presentation.pdf,

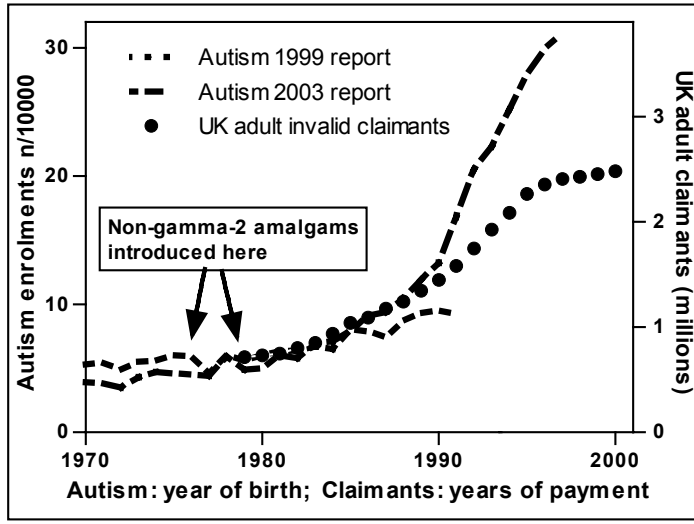

Figure 3.5. Autism enrolments (DDS) in California compared with UK adult invalidity benefits claims granted (excluding short-term lower-rate cases and excluding claims denied for policy reasons of "caseload growth now controlled")

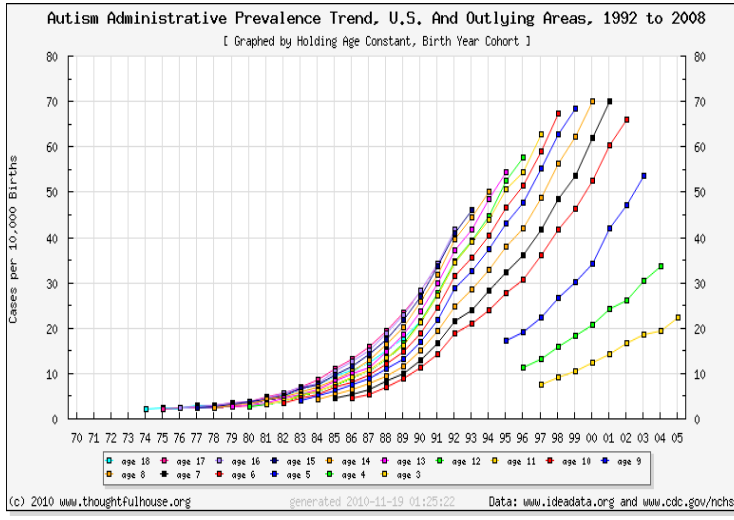

Figure 3.6. Autism enrolments under the Individuals with Disabilities Education Act (IDEA)

(DWP, no date a) and online data timeseries (DWP, no date b) from the DWP's website.

I then took the Figure 4 chart from my (long-obstructed) autism theory update review draft, and removed my data-series derived from age-of-onset ratio-change, leaving only the two data-series of autism enrolment in California. I then added in the data of granted invalidity benefits from the DWP's chart. I used only zero-baselines, so as to not to misuse the statistics to create artificial alignments. All I did was set the righthand scale such that the first datum of the invalidity benefits data was level with the autism data at that same year, 1979.

This showed a close relationship of timing between the two, as shown in Figure 5 here.

At this point it will be useful to show you a second chart of the autism increase, this time a different administrative database (IDEA rather than DDS) and covering the whole US, namely Figure 6. This is a more complex graph, with each data-series representing a different age at recording of the cases.

With the increasing of age, fewer children from any particular birth-year cohort remain undiagnosed. So in respect of each year on Figure 6, the highest datum is the most accurate estimate to date of the real underlying level. And the falling off at the top of the latest years is due to diagnoses not yet being made.

The first important thing that this chart of IDEA data shows is that the increase has been a remarkably uniform exponential sort of curve, with just a moderate decrease of slope after 1992. The other curves, from the California DDS data, can be understood as showing what would be a similarly uniform exponential, but distorted by noticeable "noise" due to smaller samples or mislaid records.

Another important thing to understand about all these curves is that we are not here counting clear distinct things like apples or oranges. The number of people granted invalidity benefits in a particular year is a precisely-known integer, but the underlying number of people who were more or less "disabled" is necessarily a debatable, fuzzy one. Likewise with the autism numbers, and this goes some way to explaining why these two autism databases (DDS and IDEA) show significant discrepancies, most obviously in the starting levels before the increase. So we must understand that none of these curves document validly exact measurements of the underlying pathologies in their vertical scales. And therefore we should not be looking for particularly close alignments in the vertical scales; and if we do find such precision it should be considered largely a fluke. Also there is a lack of numerical data on usage or total prevalence of non-gamma-2 in people's mouths.

But these charts nevertheless do give an accurate documentation on the horizontal scale, of the timing of the increases and of the form of the increases (i.e. not one big jump over a couple of years). And the four series (DDS, IDEA, onset ratio, and invalidity claims) show closely similar timing, of an increase that was gently starting off just before 1980, and then accelerating rapidly through the 1980s and well into the 1990s.

Meanwhile, there is also a weight of other facts attesting to the reality of an increase of invalidity.

The symptoms of chronic mercury vapor poisoning have been known for centuries, and include most especially all manner of mental and neurological disturbances, but also a variety of other symptoms. The wide variability of the presentation is easily understood in terms of the effects of mercury as a general anti-antioxidant, and as an antagonist to zinc thereby disrupting hundreds of enzymes, and also binding to the body's own proteins thereby causing the immune system to identify them as alien and thereby producing auto-immune reactions.

Page 14 of pathways-presentation.pdf gives an analysis of diagnoses of the claimants. It shows that 83% of cases are accounted for by those categories especially readily attributable to amalgam illness:

Mental disorder	35%
Nervous system	10%
Musculo-skeletal*	22%
Others**	16%

(* Which could be mostly fibromyalgia, a modern "mystery" illness commonly sharing features of typical amalgam illness and often cured by amalgam removal.)

(** An all-too-likely official label for cases of the amalgam illness which officially does not exist.)

[Book update: In David Brownstein's book *Overcoming thyroid disorders*, he quotes Dr Derry saying: *"Chronic fatigue and fibromyalgia were non-existent before 1980. So where did these two new diseases come from?"* Errm.... no idea, please tell me, folks.]

Further evidence supports the reality of the increase. I web-searched for the minister's words "been a three-fold increase in disability" and found instead that in Finland 1987-1994 there was a threefold increase of disability pensions granted in respect of affective disorders (mainly depression) (Salminen et al., 1997), which is one of the most common effects of amalgam illness.

And the disability claimants are now being regularly characterised by ministers and propagandists as "workshy", "bogus", or merely making a "lifestyle choice" of fraudulent leisure.

In 2010, the government minister Mark Harper declared on BBC Any Questions that "There are definitely some people in this country—and everyone in every community knows who they are—who are perfectly able to work, and don't." and then reiterated with "Everybody knows them, able-bodied people with no barriers to work who choose not to."

Another government minister, George Osborne, asserted that there were a sizeable number for whom claiming disability was a "lifestyle choice".

Meanwhile we are also being told that immigrants are substantially more hardworking than the natives of the UK, who appear by contrast to be "workshy". And indeed employers confirm such a difference.

In the real world of disability, the effects of adult mercury vapor poisoning can be far from obvious to "everyone in every community". As stated in the book *Amalgam Illness* by Andrew Hall Cutler, at page 78, "Extremely poisoned patients do not look as sick as they are …. they make adequate adrenaline during the stressful time and perform. Then they collapse for a long time while nobody is around." And at page 13, "One very important note: the patient looks a lot healthier than he is…..It is important to keep in mind that the patient may look well during appointments and yet be unable to conduct day-to-day activities, as well as be experiencing great discomfort on an ongoing basis."

And note also the following 1926 account by the famous chemist Alfred Stock of his own mercury vapor poisoning. Note how easily it could be "known" to be "workshy" were it not that the author was a notable professor.

"Mental weariness and exhaustion, lack of inclination and ability to perform any, particularly mental, work, and increased need for sleep. …. My memory, which had previously been excellent, left more and more to be desired and became worse and worse until, two years ago, I suffered from nearly complete memory loss….. I forgot the content of the book or theater play I had just read or seen as well as my own work, which had been published. ….. Obstacles, which formerly I would have overlooked smilingly (and am overlooking again today), seemed insurmountable. Scientific work caused great effort. I forced myself to go to the laboratory without being able to get anything useful accomplished in spite of all efforts. Thought came laboriously and pedantically. I had to deny myself working on solutions to questions beyond the nearest tasks at hand. The lecture that used to be a pleasure became a torture. The preparations for a lecture, the writing of a dissertation, or merely a simple letter caused unending effort in styling the material and

wrestling with the language." (translated by Birgit Calhoun)(Stock, 1926)

You can see from all the above that the characteristics ascribed to the allegedly bogus claimants are characteristics of mercury poisoning. With this understanding we can even account for the peculiar observations that workers from Eastern Europe and from more distant countries are found to be more "hardworking" than the native British, who by contrast are accused of being "workshy". In fact a whole peculiar myth about normal human nature has been deployed here. Any normal healthy person, yourself for instance, would positively want to go out and do things rather than just lie in bed or slump in front of the tv all day every day. The normal healthy person would experience the latter prospect as more like a form of imprisonment than as an agreeable "lifestyle choice".

[[Update for this book: Here are the words of Frank Field MP speaking on BBC Any Questions (Field, 2012):

> "London's got the second highest youth unemployment, and yet it is the mecca for immigrants to come in and work. Now why is it that our schools produce people who cannot work or don't work, as opposed to other people who at the very same time have work as part of their dna and the best thing in the world they want to do is to actually work? (loud applause)." [He then answers in terms of lazy racist white people, presumably with inferior dna, before continuing....]
>
> "It doesn't take much money to get the kids to school on time, washed, clothed, breakfasted, and to school on time, and it is worrying that something is happening here..." [Indeed, and my own inability to get to the grammar school on time had nothing to do with my family's shortage of money either.]

And here are words about chronic fatigue syndrome from the book *Plague* (Heckenlively & Mikovits, 2014):

> "If this had been going on in the fifties and sixties, even if we had discarded it as psychiatric, it would have been written about, and [yet] it's not in the literature." ..... "How could we have possibly missed this disease for all these years?". ....
>
> "....by the mid-1980s, distressed doctors and desperate patients had turned the disease into the top category of inquiry at both the CDC and public health departments ...."
>
> "Aided by a passive lay press, government scientists have sought to dismiss the disease by labeling sufferers with all manner of deficiencies and malevolent motives. That list has included malingering and cheating welfare systems, ... or people who [had] read about the disease and "wanted to have it".

> "By 2009.... patients were denied not merely medical care, payouts on disability claims, and the emotional support that might have been forthcoming from family and friends had they suffered from a "real" disease,..... If they were children, they were denied educations ...." [like don't I know myself, and see Chapter 8 here]]]

And furthermore, in reality almost all people are desperate to avoid becoming categorised as disabled, going to near-psychotic lengths of denial in the opposite direction. Few people would be pleased to be in any social context, with no better answer to a common question than: "I've been chronically disabled for the past five years (mentally rather than physically of course)." Virtually no-one in any society treats mentally disabled people as even near to being social or intellectual equals of themselves (in terms of marriage or educational opportunities for instance) (notwithstanding their pretentions to otherwise).

An even greater catastrophe?

Notably in line with the UK data, recipients of disability benefits in the US (SSI/SSDI) also increased more than twofold between 1987 and 2007.

Here are some further facts. Figure 5 indicates a levelling off at 2.5 million claimants from 1995 onwards. But this must be put in the context of the words of the DWP document those figures came from. It was an internal discussion document about the "Personal Capability Assessment", and its page 11 was headed "Caseload growth now controlled". Translating those words from Officialese, they mean that there has been political resistance to the growth of disability claims, and that many thousands of persons genuinely disabled by DH recklessness have been denied the disability benefits they needed for survival, and that if the graph had reflected the real increase of disability it would not have levelled off, but instead would probably have surpassed more like 4 million by 2000 (which is about 10% of the UK's working-age population). [Update August 2015: "Statistics [reluctantly] released by the DWP on Thursday revealed that 2,380 people died between December 2011 and February 2014 within 14 days of being taken off Employment and Support Allowance because a Work Capability Assessment had concluded they were able to work." (Butler, 2015).][Update November 2015: 590 additional suicides linked to WCA reassessments (Barr et al., 2015; Benefits and Work, 2015).]

And yet more. Four of the most characteristic symptoms of chronic mercury vapor poisoning are fatigue, depression, sleep disturbances, and poor memory. And surveys in recent years have

found that now a gigantic proportion of the NON-claimant population have these very symptoms, as follows.

Depression:

A survey of 2000 women and girls in England and Wales found 63% had been affected by mental health problems, having "a devastating impact on their lives", and "48% experiencing mental health problems had stayed in bed or not left the house for a long period as a result" (Platform 51, 2011). Meanwhile, Colin Walker of Mind said his organisation's research showed men and women experienced mental health problems such as depression and anxiety in roughly equal numbers (Hill, 2011).

Insomnia:

A report from the Mental Health Foundation (2011) states: "Only 38% of survey respondents (2522 people) were classified as 'good sleepers', whilst 36% were classified as possibly having chronic insomnia (2414 people). …. Other estimates of insomnia have put the total figure at around 30% of adults, …. although rates depend upon the criteria used to define it. Of the people reporting insomnia in the survey, over 30% have had insomnia for 2–5 years, and over 25% for over 11 years (figure 4)."

The figure 4 in question then shows a distinctly bimodal distribution, in which the larger, longer-term, mode can be reasonably attributed to the effects of the dental amalgam toxicity.

Fatigue:

In a survey by Pharmaton (2010) in the UK, 24% said they are mentally or physically exhausted every day, 45% say they miss socialising due to tiredness, and 60% of the young are too tired to socialise, compared to 40% in 2002. And that is in line with the widespread experience that immigrants from less-developed countries are substantially more "hardworking" than those who have grown up in the UK, who are conversely "workshy" as discussed on a preceding page here.

Memory:

Almost everyone nowadays wishes they had "better" memory, by which they mean more remembering rather than less. And yet contrary to the common assumption, memory is not something which natural selection would always be pressuring for more of (such as health or beauty). On the contrary, some persons (e.g. Solomon Shereschevsky) have had more memory than was actually useful for them. And history attests to the powerful memorising abilities of our ancestors.

(This chapter continues on the next page.)

Update 1

All the preceding evidence here was suggesting to me an obvious further question, namely whether the original introduction of amalgams in the 19th century had caused an earlier increase of mental disabilities to the baselines shown here. Subsequent to my writing all the preceding, I learnt of the detailed historical review by Torrey & Miller (2002) of what was then called "insanity", and the time-series graphs therein (at pages 94, 152, 188, 271, and frontispiece). In Figure 7 here I have re-plotted their data along with dates relating to the introduction of amalgam. Their book makes no mention of amalgam, or dentistry, nor of mercury as a possible cause of that increased morbidity. And yet their graphs show that rates of mental disability steadily increased from the original introduction of amalgams till a century later, by fourteenfold in Ireland and Canada, elevenfold in the US, and fivefold in the UK. These increases occurred in the context of vociferous contemporary condemnations of the use of amalgam due to its causing of mercury toxicity disabilities. The ASDS disbanded and the ADA replaced it because too many dentists preferred making quick profits from poisoning their patients with fillings deceitfully referred to as "silver".

Two curious observations on Figure 7. Firstly, the starting level being much higher in England/Wales, which could be because England was the first industrialised country, and with the main fuel both in houses and factories being mercury-emitting coal, besides which mercury was used for other purposes (such as hat-making). And indeed there is much reference in Torrey & Miller to insanity having been considered "the English disease".

Secondly, the ending level being much higher in Ireland, which could be because Ireland gets high rainfall from the Gulf Stream and consequently people are much more indoors and hence breathing in the amalgam mercury (as per my citation of Waldman et al earlier here). These two reality-harmonious observations suggest that these statistics reflect real increases rather than what some might construe as just some speculated mysterious spontaneous increase of awareness of what was then called insanity.

And the Preface of their book states: "It has now been almost thirty years since one of us—E. Fuller Torrey—submitted a paper for publication suggesting that epidemic insanity was a recent phenomenon. .... The paper was summarily rejected by all journals .... and it was never published.... ".

And then even my own copy of their extraordinary book had come from being dismissed from a library in Illinois.

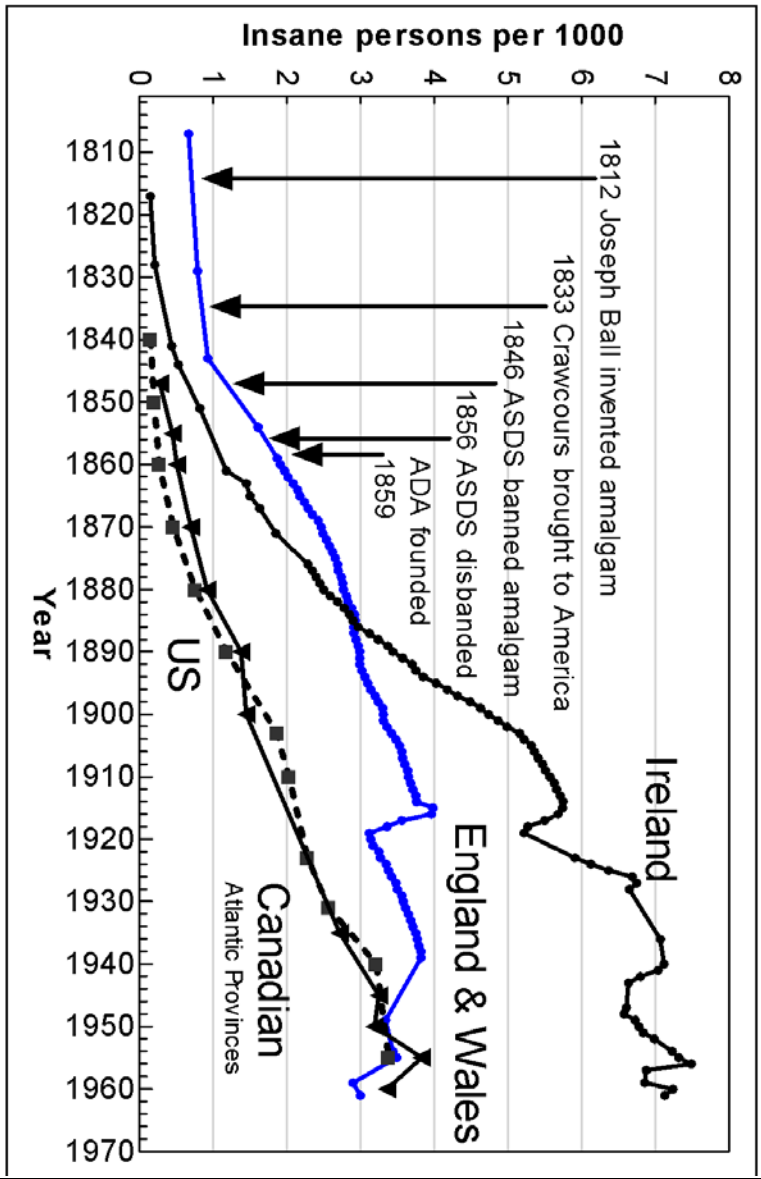

Figure 3.7. Insane persons in relation to the history of amalgams

[Update 2

Since about 2012 there has been a very peculiar and "mysterious" change to the appointments systems used in the UK by the clinics of NHS GPs. The new systems require the patients to phone in at 8.15–8.30 am or else wait to phone in next day at 8.15–8.30. If a patient missing the deadline asks to be listed for the next day, they just get a blank reasonless reply that "We can't do that", as if the ability to write down a note on a piece of paper has somehow been lost by receptionists.

On the NHS Choices website you can see countless people complaining about this appointments system and how it was so much better for many years before it was thus "improved". So why make this weird change which no one asked for and many have been complaining bitterly about? Why "fix" something that wasn't broken anyway, and then only with a hugely inconvenient and annoying disimprovement rather than an improvement?

Well here's the answer to this mystery. Basically the new systems are designed to get rid of the mercury-poisoned people who (speaking from *far* too much personal experience) (a) cannot wake up in the morning, (b) even if they are awake, still cannot remember to phone at just that time, (c) are too exhausted to keep dialling and likely phone-phobic as well. So the system successfully disappears all the mercury-poisoned would-be patients who would be complaining of Tired All The Time (TATT, the most frequent complaint received by GPs which of course they can do nothing honest about anyway as the MHRA has decreed that "amalgams are harmless"). Thereby are got rid of those who raise tedious questions about mercury poisoning (as I did) or who get angry at the constant shifty nonsense they encounter from the medical professionals. And furthermore, because they fail to get registered for an appointment, their reports of illness do not even exist in legal terms. Thereby is the epidemic detailed in this book pretended into non-existence by this new appointment system perfectly designed for exactly that purpose.

Chapters 8 and 9 will show you more on the huge deceitfulness shown against a severely disabled person very obviously poisoned by amalgams, and despite years of informative efforts about the matter.**]**

Conclusions and Predictions

It is important to bear in mind here the further supporting data reviewed by Mutter (2011), Hanson (2004), Geier et al (2010), Homme et al (2014), and others.

There are roughly two alternative viewpoints which may be reached from the data presented here. On the one hand there is a notion which entails that:

(1) The heavy involvement of mercury in modern autism has nothing to do with the largest source of mercury input but instead is due to some other mysterious source or process.

(2) And these graphs and other observations are mere coincidences in time.

(3a) And either some mysterious unknown substance caused all these disabilities just so as to resemble the mercury symptoms that Mutter, Hanson, Geier, etc., have long been predicting anyway *on entirely different evidence*, and just happened to coincide at the right time to neatly confuse the author.

(3b) Or there has been either a huge moral degeneration into "workshy" or else millions of people have enthusiastically embraced a "lifestyle choice" of living like a prisoner combined with the social leper dis-status of being mentally disabled, and furthermore these shirkers by some fluke just happened to be getting diagnosed with mercury symptoms even though they knew nothing about mercury toxicity, and by further impressive fluke so closely coincided with the increases of autism diagnoses and non-gamma-2 prevalence. And these "workshy" millions are somehow descendants of the people who hand-built the huge medieval cathedrals in a cold wet small island and then went on to create the largest empire (of hardworking foreigners) in history.

(4) And a many-fold increase of mercury burden has not had any harmful effects on the millions thus burdened.

(5) And the change of autism from life-long genetic to environmental and recoverable is just another of these mysteries.

(6) And those gross untruths from the NHS just happened by fluke to all relate to preventing people getting diagnosed with mercury poisoning (two evidence-defying pseudo-tests, the "birds are highly unlikely to have wings" nonsense, the "see a dentist instead" - "see a doctor instead" nonsense, the review of my non-dental problems complaint exclusively by a dental panel with no toxicological or neurological expertise, the NHS's own pseudo-study to pretend away the autism increase, and the Chief Dental Officer's evidence-defying insistence that no mercury vapor comes off anyway).

(7) And merely by yet another fluke Torrey's graphs confirmed my suspicion that there would have been a previous increase of mental disabilities following the original introduction of amalgam 150 years earlier.

(8) And merely by yet another fluke there is that observation that most mental disorders start in the 12-25 age-range.

Alternatively there is a notion that non-gamma-2 amalgam has been the main cause of a tenfoldish increase of autism and a fourfoldish increase of adult disability including so-called "workshy". It is the view of this author that this latter interpretation of the data strains credibility very much less than the former. It is hardly a surprising discovery given what Mutter, Hanson, and others have previously predicted on entirely different evidence already.

And likewise the data of an increase in the 19th century cannot be lightly dismissed as "merely" coincidence. Some such increase was to be suspected by inference from the later non-gamma-2 data; it is scientifically explainable in terms of known mercury toxicity; and indeed it was very much pre-warned of already by ASDS members 170 years ago. And the ADA then adopted the propaganda language of "silver amalgams" by way of the ongoing cover-up. And I obtained that data from a very detailed review book which did not even mention dental or amalgam, so can hardly be dismissed as some sort of cherry-picking.

Editors of putatively scientific medical journals have a duty to ensure that the public is not being kept unaware of evidence of possible serious harm from standard medical practices. It is a serious breach of ethics for such evidence as contained here to be refused publication other than for rigorously justified reasons. If there really are any serious faults in the case presented here, they should be openly published in the scientific literature rather than used as mere excuses to prevent the evidence being raised in the first place.

It is here predicted that these increases will tend to correlate together in comparisons between different nations, due to the common causality. It is predicted that these epidemics will only be reversed by reduction of prevalence of non-gamma-2 in victims' mouths. And meanwhile the risk of autistic disability can be reduced by ensuring adequate ventilation (in practice with a through draught at breathing-level).

[[**Update 3**

Subsequent to all the preceding I have noticed the words of Professor Stephen Wood, as follows:

> "My research aims to understand the health paradox of adolescence – the years between 12 and 25 are a time of great physical fitness, yet this is the period during which 75% of all mental disorders have their onset. Why should this be the case? Clearly changes in the brain are likely culprits, but how they interact with genetic and environmental factors to produce illness is unclear." (Wood, 2015)

But I suggest that there is not really much paradox or unclarity here (in the context of the graphs on the preceding pages). It could be simply that medical experts start installing those great lumps of harmlessly neurotoxic mercury into the mouths of people two inches from their brains shortly before that age.

In 2012 Professor Wood got a grant of £818,000 from the MRC for this research on adolescent mental problems. The entirety of *all* my own research to date has been funded to the tune of £0.]]

Additional Files

Additional File 1.htm: Four Freedom of Information requests (outlined in the Appendix). [www.tinyurl.com/dentmerc]
Additional File 2.pdf: Mercury in vaccines as alleged cause of autism increase. [see Chapter 6]
Additional File 3.pdf: Prior responses from journals. [Chapter 5]
Additional File 4.pdf: Prior responses from Neurotoxicology journal. [Chapter 4]

[Update: One in six American adults taking psychiatric drugs (Moore and Mattison, 2016).]

[Update: "About 300,000 [UK] people with a long-term mental health problem lose their jobs each year, a review commissioned by Theresa May has found." (Siddique, 2017)]

Appendix: Four Freedom of Information requests

(more fully documented via Additional File 1.htm) or
http://tinyurl.com/dentmerc

Why "Chronic mercury poisoning is highly unlikely to present in a psychiatric setting."?

Mr Clarke made this Freedom of Information request to
Birmingham and Solihull Mental Health NHS Foundation Trust
RESPONSE TO THIS REQUEST IS LONG OVERDUE
6 September 2011

Dear Birmingham and Solihull Mental Health NHS Foundation Trust,

1. Given that it has been well-known for years, decades, and even centuries, that among the most characteristic symptoms of chronic mercury poisoning are nervousness, shyness, depression, agitation, fatigue, impaired memory, lack of concentration, and indecision (as per abundant documentation indicated below):
Why did the BSMHFT (Birmingham and Solihull Mental Health Foundation Trust) state this year in a FOI reply that "Chronic mercury poisoning is highly unlikely to present in a psychiatric setting."?
2. What scientific or evidential basis existed to justify such a statement?
3. Who in the BSMHFT gave that answer, and from where did they derive that conclusion? Where did the notion originate?

Yours faithfully,
Mr Clarke

DOCUMENTATION:

Numerous studies and reports exist, for example:
Alfred Stock 1926: "Mental weariness and exhaustion, lack of inclination and ability to perform any, particularly mental, work, and increased need for sleep..... nearly complete memory loss..... Obstacles, which formerly I would have overlooked smilingly, seemed insurmountable.... merely writing a simple letter caused unending effort...."
BMJ 287:1961 (1983) Did the Mad Hatter have mercury poisoning? HA Waldron: "The principal features of erethism were excessive

timidity, diffidence, increasing shyness, loss of self confidence, anxiety, and a desire to remain unobserved and unobtrusive. The victim also had a pathological fear of ridicule and often reacted with an explosive loss of temper when criticised."
1899 Tuthill: "makes a mental wreck of its victim".
1974 J Am Dent Soc 98(4),904: "symptoms include ….
self-consciousness, embarrassment without justification,
disproportionate anxiety, indecision, poor concentration, depression,
irrational resentment of criticism, and irritability."

TOXICOLOGICAL PROFILE FOR MERCURY. U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service Agency for Toxic Substances and Disease Registry March 1999 Page 276:
Neurological Effects. The nervous system is the primary target organ for elemental and methylmercury-induced toxicity. Neurological and behavioral disorders in humans have been observed following inhalation of metallic mercury vapor and organic mercury compounds, ingestion or dermal application of inorganic mercury-containing medicinal products (e.g., teething powders, ointments, and laxatives), and ingestion or dermal exposure to organic mercury-containing pesticides or ingestion of contaminated seafood. A broad range of symptoms has been reported, and these symptoms are qualitatively similar, irrespective of the mercury compound to which one is exposed. Specific neurotoxic symptoms include tremors (initially affecting the hands and sometimes spreading to other parts of the body), emotional lability (characterized by irritability, excessive shyness, confidence loss, and nervousness), insomnia, memory loss, neuromuscular changes (weakness, muscle atrophy, and muscle twitching), headaches, polyneuropathy (paresthesias, stocking-glove sensory loss, hyperactive tendon reflexes, slowed sensory and motor nerve conduction velocities), and performance deficits in tests of cognitive and motor function (Adams et al. 1983; Albers et al. 1982, 1988; Aronow et al. 1990; Bakir et al. 1973; Barber 1978; Bidstrup et al. 1951; Bluhm et al. 1992a; Bourgeois et al. 1986; Chaffin et al. 1973; Chapman et al. 1990; Choi et al. 1978; Cinca et al. 1979; Davis et al. 1974; DeBont et al. 1986; Discalzi et al. 1993; Dyall-Smith and Scurry 1990; Ehrenberg et al. 1991; Fagala and Wigg 1992; Fawer et al. 1983; Foulds et al. 1987; Friberg et al. 1953; Hallee 1969; Harada 1978; Hook et al. 1954; Hunter et al. 1940; Iyer et al. 1976; Jaffe et al. 1983; Jalili and Abbasi 1961; Kang-Yum and Oransky 1992; Karpathios et al. 1991; Kutsuna 1968; Langauer-Lewowicka and Kazibutowska 1989; Kutsuna 1968; Langolf et al. 1978; Langworth

et al. 1992a; Levine et al. 1982; Lilis et al. 1985; Lundgren and Swensson 1949; Matsumoto et al. 1965; McFarland and Reigel 1978; Melkonian and Baker 1988; Miyakawa et al. 1976; Ngim et al. 1992; Piikivi and Hanninen 1989; Piikivi and Tolonen 1989; Piikivi et al. 1984; Roels et al. 1982; Sexton et al. 1976; Shapiro et al. 1982; Snodgrass et al. 1981; Smith et al. 1970; Tamashiro et al. 1984; Taueg et al. 1992; Tsubaki and Takahashi 1986; Verberk et al. 1986; Vroom and Greer 1972; Warkany and Hubbard 1953; Williamson et al. 1982). Some individuals have also noted hearing loss, visual disturbances (visual field defects), and/or hallucinations (Bluhm et al. 1992a; Cinca et al. 1979; Fagala and Wigg 1992; Jalili and Abbasi 1961; Locket and Nazroo 1952; McFarland and Reigel 1978; Taueg et al. 1992). Although improvement has often been observed upon removal of persons from the source of exposure, it is possible that some changes may be irreversible. Autopsy findings of degenerative changes in the brains of poisoned patients exposed to mercury support the functional changes observed (Al-Saleem and the Clinical Committee on Mercury Poisoning 1976; Cinca et al. 1979; Davis et al. 1974; Miyakawa et al. 1976).

The characteristic symptoms of chronic mercury vapour are also documented in innumerable other studies and sources and case histories:

a) “References documenting symptoms to mercury exposure” published by the International Academy of Oral Medicine and Toxicology, www.iaomt.org ; the first seven in their list are all very familiar as major symptoms of this inquirer, namely irritability, anxiety/nervousness, loss of memory, inability to concentrate, lethargy/drowsiness, insomnia, mental depression/ despondency/ withdrawal; plus also very familiar, 9: muscle weakness, 11: tremors of hands, legs, eyelids, 12: decline of intellect, 13: loss of self-confidence, 16: bleeding gums, 18: loosening of teeth, etc.

b) Mats Hanson “Effects of Amalgam Removal on Health; 25 studies comprising 5821 patients” lists the main removal findings as “fatigue, anxiety/depression, muscle pains, headache, concentration problems, joint problems, metal taste, mouth symptoms, vertigo/dizziness, gastrointestinal problems, memory disturbances, problems with sight, irritability, sleep disturbances, heart problems, skin problems, allergies, problems with hearing, numbness, infection-prone (bold added here to indicate this inquirer’s most notable symptoms in that list).

c) Extensive further documentation of causation of these same symptoms can be seen in excerpts here appended from www.flcv.com/depress.html and www.flcv.com/amalg6.html.

Excerpt from http://www.flcv.com/amalg6.html

Bernard Windham compilation of references re amalgam removal cases

[….]

VI. Results of Removal of Amalgam Fillings

[…] There are extensive documented cases (many thousands) where removal of amalgam fillings led to cure or significant improvement of serious health problems such as: [excerpts here:]
epilepsy (5,35,309,229,386e,557),
dizzyness/vertigo
(8,40,95,212,222,229,233bcdgh,271,322,376,453,525c,551,552),
523,525c,538,551, 552,556,557,583),
insomnia (35,62,94,212,222,233ag,271,317,322, 376,525c,583),
MS 2,94,95,102,163,170,212,222,229,271,291,302,322,369,
469,485,34,35c,229,523, 532),
ALS (97,246,423,405,469,470,485,535,35),
Alzheimer's (62,204,251c,386e,535,35),
Parkinson's/ muscle tremor (222,248,228a,229,233f, 271,322,
469,557,212,62,94,98,35),
Chronic Fatigue Syndrome
(8,35,47f,60,62,88,185,212,293,229,222,232,233abcdfgh,271, 313,
317, 322,323,342, 346, 369,376,386de, 440, 469,
470,523,532,537,538, 551,552,556,557,595),
nausea (525c),
neuropathy/paresthesia (8,35,62,94,163,212,222,322,556,557),
memory disorders (8,35,94,212,222,322,437,440,453,552,557,595),
depression
(62,94,107,163,185,212,222,229,233bcfh,271,294,285e,317,322,376,3
86de,437,453,
465,485,523, 525c,532,538,551,556,557,583,595,35,40),
anxiety & mental confusion
(62,94,212,222,229,233abcfgh,271,317,322,440,453,525c, 532,551,
557,583,35,57),
neuropathy/paresthesia (8,35,62,94,163,212,222,322,556,557),

Why SWBHNHST uses well-known useless quack tests of dental amalgam mercury poisoning?

Mr Clarke made this Freedom of Information request to Sandwell and West Birmingham Hospitals NHS Trust

RESPONSE TO THIS REQUEST IS LONG OVERDUE

23 September 2011

Dear Sandwell and West Birmingham Hospitals NHS Trust,

1. Given that it has been well-established and well-known for decades that blood mercury level and urine mercury level are useless as indicators of chronic mercury toxification (as documented below)....

Why did the toxicologists of the SWBHNHST / the City Hospital in Birmingham propose in 2010/11 these well-known useless tests in respect of a patient presenting substantial evidence of being disabled by dental amalgams?

2. What scientific or evidential basis existed to justify such proposals?

3. From where did the SWBHNHST toxicologists get that notion of usefulness of those blood and urine tests? Where did that notion originate?

4. What worthwhile purpose could be served by those tests given that the patient already had reported extraordinarily high mercury vapour measurements of 460 mcg/m3 (unprovoked, open mouth) (a world record level, about 100x higher than typical levels)?

DOCUMENTATION:

Goldwater et al. (1964) stated:

> "Those investigators who have studied the subject are in almost unanimous agreement that there is a poor correlation between the urinary excretion of mercury and the occurrence of demonstrable evidence of poisoning."

and a joint statement of the National Institute of Dental Health and the American Dental Association (NIDH/ADA, 1984) stated in 1984 that: "The distribution of mercury into the body tissues is highly variable and there appears to be little correlation between levels in urine, blood or hair and toxic effects." And later studies have further confirmed that conclusion. Even with normal or low mercury levels in blood, hair and urine, high mercury levels are

found in critical organs such as brain and kidney (Danscher et al., 1990; Drasch, 1997; Hahn et al. 1989, 1990, Hargeaves et al., 1988; Lorscheider et al., 1995; Opitz et al., 1996; Vimy et al., 1990; Weiner & Nylander, 1993). Drasch et al. (2001, 2002, 2004) found that 64% of individuals occupationally exposed to mercury vapor and having typical clinical signs of mercury intoxication had low mercury levels in blood. A more recent autopsy study again confirmed the lack of correlation between inorganic (e.g. dental) mercury levels in urine or blood and mercury levels in brain (Björkman et al. 2007).

Bjorkman L, Lundekvam BF, Laegreid T, Bertelsen BI, Morild I, Lilleng P. 2007. Mercury in human brain, blood, muscle and toenails in relation to exposure: an autopsy study. Environ Health 6:30.

Danscher G, Hørsted-Bindsley P, Rungby J. 1990. Traces of mercury in organs from primates with amalgam fillings. Exp Mol Pathol 52:291-299.

Drasch G, Wanghofer E, Roider G. 1997. Are blood, urine, hair, and muscle valid bio-monitoring parameters for the internal burden of men with the heavy metals mercury, lead and cadmium? Trace Elem Electrolyt 14:116-123.

Drasch G, Böse-O'Reilly S, Beinhoff C, Roider G, Maydl S. 2001. The Mt. Diwata study on the Philippines 1999 - assessing mercury intoxication of the population by small scale gold mining. Sci Total Environ 267:151-168.

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Drasch G, Böse-O'Reilly S, Maydl S, Roider G. 2004. Response to the letter of the Human Biomonitoring Commission. Int J Hyg Environ Health 207:83-184.

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Hahn LJ, Kloiber R, Leininger RW, Vimy MJ, Lorscheider FL. 1990. Whole-body imaging of the distribution of mercury released from dental fillings into monkey tissues. FASEB Journal 4:3256-3260.

Hargreaves RJ, Evans JG, Janota I, Magos L, Cavanagh JB. 1988. Persistant mercury in nerve cells 16 years after metallic mercury poisoning. Neuropath Appl Neurobiol 14:443-452.

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NIDH/ADA Workshop on Biocompatibility of Metals. 1984. J Am Dent Assoc 109, September 1984.

Opitz H, Schweinsberg F, Grossmann T, Wendt-Gallitelli MF, Meyermann R. 1996. Demonstration of mercury in the human brain and other organs 17 years after metallic mercury exposure. Clin Neuropath 15:139-144.

Vimy MJ, Takahashi Y, Lorscheider FL. 1990. Maternal-fetal distribution of mercury (203 Hg) released from dental amalgam fillings. Am J Physiol 258:939-945.

Weiner JA, Nylander M. 1993. The relationship between mercury concentration in human organs and different predictor variables. Sci Tot Environ 138:101-115.

Yours faithfully,
Mr Clarke

Seriously misleading falsehoods about dental amalgam by Chief Dental Officer Barry Cockcroft

Mr Clarke made this Freedom of Information request to Department of Health

RESPONSE TO THIS REQUEST IS LONG OVERDUE

1 November 2011

Dear Department of Health,

The Chief Dental Officer Barry Cockcroft declared (on ITV, see link below) that no mercury vapour is emitted from dental amalgams, or --on second thoughts-- at least "not measureably". Yet this is violently at odds with the real bleedingly obvious long-established facts of the matter, as per documentation below.

He further queried the point that dental amalgam is the main source of mercury exposure in humans. Again this flies in the face of the known evidence. As per documentation below.

In the context of the above, would you please tell me:

1. On what scientific basis did Barry Cockcroft assert that no measurable mercury is emitted from amalgams?

2. On what scientific basis did Barry Cockcroft assert that dental amalgam is not the main source of mercury exposure?
3. In the absence of such a scientific basis, why did Barry Cockroft make these very seriously misleading assertions on the major ITV Tonight program?
4. Do you appoint utter incompetents/liars/idiots to your most senior positions as a matter of deliberate policy or did you make some mistake in the case of Barry Cockcroft?
5. Why is Barry Cockcroft still the Chief Dental Officer more than two years later?
6. Why do you everywhere keep the public in the dark about those two very important most basic points of amalgam toxicity (that measureable amounts of mercury are constantly emitted and that that is the main source of mercury exposure)?

Yours faithfully,
Mr Clarke

DOCUMENTATION:
This video:
http://www.youtube.com/watch?v=mMI_em8UPo4
shows at 5-7 minutes: (a) three measurements (9.93, 2.58, 1.66); (b) CDO Barry Cockcroft declaring that the measurements are impossible; (c) a further measurement (2.44).

> Here are TEN studies from twenty years earlier, of the measurements that Chief Dental Officer Barry Cockroft says are impossible.

Svare, C.W., Peterson, L.C., Reinihardt, J.W., et al. (1981): The effect of dental amalgams on mercury levels in expired air. J Dent Res 60:1668-1671.

Patterson, J.E., Weissberg, B.G., Dennison, PJ. (1985): Mercury in human breath from dental amalgams. Bull Environ Contam Topical 34:459-468.

Vimy, M.J., Lorscheider, F.L. (1985): Serial measurements of intra oral air mercury: estimation of daily dose from dental amalgam. J Dent Res 64:1072-1075.

Berglund, A., Pohl, L., Olsson, S., Bergman M. (1988): Determination of the rate of release of intra-oral mercury vapor from amalgam. J Dent Res 67: 1235-1242.

Vimy, MJ., Lorscheider, FL. (1985): Intraoral air mercury released from dental amalgam. J Dent Res 64:1069-1071.

Clarkson, TW., Friberg, L., Hursh, JB., Nylander, M. (1988): The prediction of intake of mercury vapor from amalgams. In: Clarkson, TW., Friberg, L., Nordberg, GF., Sager, P.R. editors. Biological Monitoring of Toxic Metals, New York. Plenum Press: 247-260. ·

Vimy, M.J., Lorscheider, F.L. (1990): Dental amalgam mercury daily dose estimated from intra oral vapor measurements: a predictor of mercury accumulation in human tissues. J Trace Elem Exp Med 3:111-123.

Mackert, J.R., Jr. (1987): Factors affecting estimation of dental amalgam mercury exposure from measurements of mercury vapor levels in intra oral and expired air. J Dent Res 66:1775-1780.

Olsson,, S., Berglund, A., Pohl, L., Bergman, M. (1989): Model of mercury vapor transport from amalgam restorations in the oral cavity. J Dent Res 68:50~508.

Olsson, S., Bergman, M. (1987): Intraoral air and calculated inspired dose of mercury [Letter]. J Dent Res 66:1288-1289.

And here are EIGHT more studies from more than fifteen years ago, comparing these impossible measurements for differing types of amalgam:

Mahler DB, Adey JD, Fleming MA: Hg emission from dental amalgam as related to the amount of Sn in the Ag-Hg (g1) phase. J Dent Res 1994, 73:1663-1668.

Berglund A: An in vitro and in vivo study of the release of mercury vapor from different types of amalgam alloys. J Dent Res 1993, 72:939-946.

Boyer DB: Mercury vaporization from corroded dental amalgam. Dent Mater 1988, 4:89-93.;

Psarras V, Derand T, Nilner K: Effect of selenium on mercury vapor released from dental amalgams: An in vitro study. Swed Dent J 1994, 18:15-23.

Ferracane JL, Adey JD, Nakajima H, Okabe T: Mercury vaporization from amalgams with varied alloy composition. J Dent Res 1995, 74:1414-1417.

Moberg LE: Long-term corrosion studies in vitro of amalgams and casting alloys in contact. Acta Odontol Scand 1985, 43:163-177.

Moberg LE: Corrosion products from dental alloys and effects of mercuric and cupric ions on a neuroeffector system [dissertation]. Stockholm; 1985.

Brune D, Gjerdet N, Paulsen G: Gastrointestinal and in vitro release of copper, cadmium, indium, mercury and zinc from conventional and copper-rich amalgams. Scand J Dent Res 1983 Feb, 91(1):66-71.

Finally, references for amalgam being the main source of mercury:

-Criteria 118 WHO 1991 states that amalgam is up to 6x the other sources combined;

-Aposhian HV, Environ Health Perspect 1998: – 2/3 comes from amalgam.

-Richardson GM. Assessment of mercury exposure and risks from dental amalgam. Health Canada 1995. Tolerable Daily Intake is exceeded in adults with 4 or more amalgams.

Dentist training in diagnosis of mental/physical symptoms of mercury

Mr Clarke made this Freedom of Information request to University of Birmingham
The request was **successful**.
26 October 2011

Dear University of Birmingham,

I have been informed by an NHS Chief Executive that dentists have the capability to diagnose chronic systemic mercury poisoning whereas GPs do not (and thus the GP was correct in telling me to instead see a dentist about my fatigue and mental and other problems).

In that connection, could you please tell me the following.

1) What training in this diagnosis do your dental students receive?
2) What methods do they use in this diagnosis?
3) Why have all the dentists I have consulted invariably insisted that they do not have any capability of making such diagnosis and insist that I have to seek it from a doctor instead?

Yours faithfully,
Mr Clarke

1) What training in this diagnosis do your dental students receive?

Undergraduate dental students at the University of Birmingham are **not** taught to diagnose chronic systemic mercury poisoning.

2) What methods do they use in this diagnosis?

Undergraduate dental students at the University of Birmingham are not taught to diagnose chronic systemic mercury poisoning and therefore the question is not relevant.

3) Why have all the dentists I have consulted invariably insisted that they do not have any capability of making such diagnosis and insist that I have to seek it from a doctor instead?

Under the Freedom of Information Act 2000 the University of Birmingham is only required to provide information that it holds rather than to express an opinion; therefore the University can provide only limited information in respect of this part of your request

The diagnosis of systemic poisoning clearly falls into the area of clinical toxicology and therefore would require a diagnosis from a medically qualified specialist. A general dental practitioner would not have the knowledge or skills to exclude all other systemic possible causes of the symptoms an individual was suffering without considerable postgraduate training. Therefore only a limited number of dentists would have this capability.

References

The references cited in this paper would normally be listed at the end here in a scientific paper but as this is incorporated in a book chapter they have here been merged with the list at the end of the book instead.

"the most insidious myth, increasingly pervasive, is that the poor are workshy, scrounging"

– Labour former UK Prime Minister Gordon Brown, November 2015

"Everyone in receipt of benefits is not a scrounger."

– Conservative former UK Prime Minister John Major, November 2015