

Blinded by neuroscience: social policy, the family and the infant brain

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Current social policy initiatives are promoting early intervention to improve the lives of disadvantaged children. Neuroscientific evidence is prominent in this discourse, creating the lustre of science, but too much has been taken on trust. In particular, the argument that the first three years are critical has created a now-or-never imperative to intervene before irreparable damage is done to the developing infant brain. A critique of current policy in the United Kingdom is provided here, drawing on counter-arguments from the policy discourse in the United States during the 'decade of the brain', updated with more recent research findings. Overall, we show that the infant brain is not readily susceptible to permanent and irreversible damage from psychosocial deprivation. Rather, plasticity and resilience seem to be the general rule. The co-option of neuroscience has medicalised policy discourse, silencing vital moral debate and pushing practice in the direction of standardised, targeted interventions rather than simpler forms of family and community support, which can yield more sustainable results.

Introduction

[B]abies are born with 25 per cent of their brains developed, and there is then a rapid period of development so that by the age of 3 their brains are 80 per cent developed. In that period, neglect, the wrong type of parenting and other adverse experiences can have a profound effect on how children are emotionally 'wired'. (Allen, 2011a: xiii)

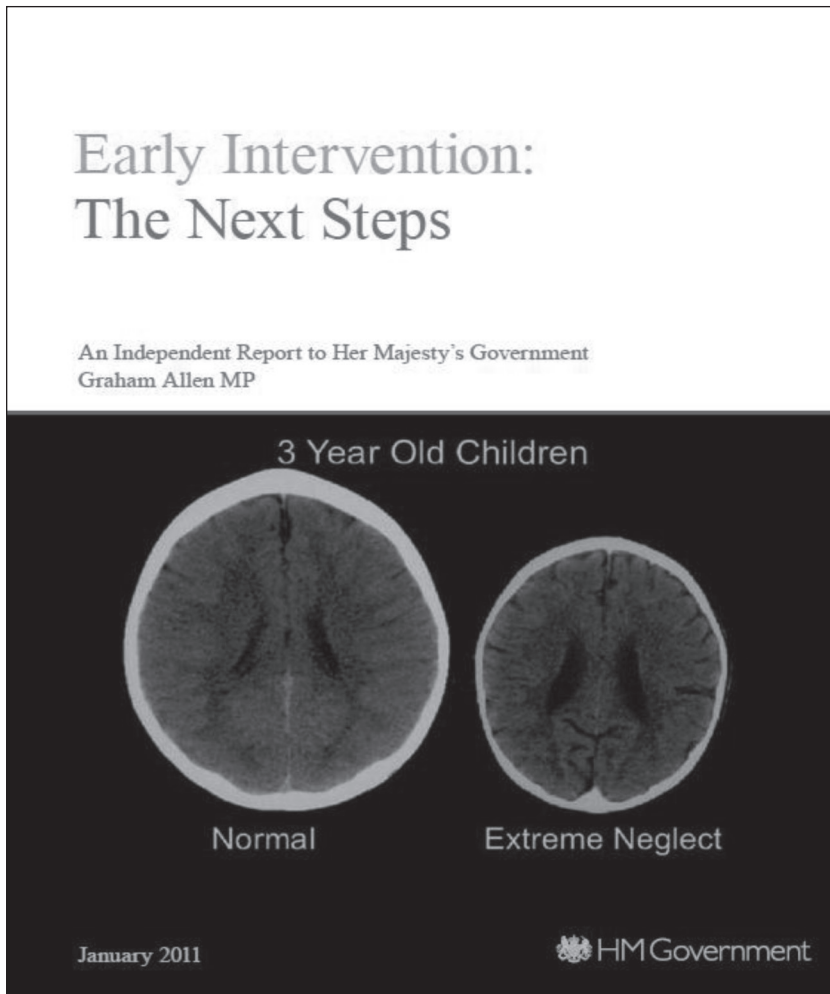
Social policy initiatives in the United Kingdom (UK) are currently promoting early intervention to improve the lives of disadvantaged children. The above epigraph is taken from the recently published report of Graham Allen, commissioned by the UK government in July 2010. Allen published his first report in January 2011; indeed, it was eagerly delivered 'ahead of time'. The neuroscience of the infant brain figures prominently; the brain is mentioned 59 times, and the cover carries dramatic images of an infant brain damaged by neglect (see Figure 1). Allen's second report retains the brain image on the cover, now joined by symbolic bars of gold emphasising the economic sense behind 'early intervention' (Allen, 2011b). Saliiently, the brain is not mentioned again; the case having been made, it is now simply a question of taking action. The report accordingly sets out various preferred ways to remedy the ills created by tardy intervention; privileged are a range of 'evidence-based', targeted programmes on 'parenting'. In its use of neuroscientific evidence to warrant claims

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about the irreversible vulnerabilities of early childhood, and the proper responses of the state, Allen's first report is not alone. The now-or-never imperative, based on neurodevelopmental evidence of lasting damage, features across the gamut of current social policy. It is invoked, in more restrained terms, in Munro's (2011) review of the child protection system in England and in the recent family justice review (Ministry of Justice, 2011).

There is much to commend a 'progressive' agenda of help for the most disadvantaged children, and offering this at the earliest juncture; we are also sympathetic to neuroscience and what it may offer. It is the desire neither to help vulnerable children nor to draw on the best scientific knowledge that is in question here. Rather, we argue that the neuroscientific claims supporting current policy initiatives have received

Figure 1: The front cover of the first Allen report



Source: Allen (2011a)

little critical commentary. They appear to be operating as powerful ‘trump cards’ in what is actually very contentious terrain, suppressing vital *moral* debate regarding the shape of state intervention in the lives of children and families.

In this article, we interrogate the nature of the scientific claims made in key documents and the ideological thrust of policy that they have engendered. We examine Allen’s first report in detail first, before developing a more general critique of what Tallis and others have dubbed *neuromania*: ‘the appeal to the brain, as revealed through the latest science, to explain our behaviour’ (Tallis, 2011: 5; Legrenzi and Umilta, 2011). Bruer’s (1999) deconstruction of the ‘myth of the first three years’ will feature prominently in our argument, paving the way for a broader critical analysis of the ‘new’ brain science and its influence on policy. We contend that neuroscience is re-presenting an older ideological argument about the role of the state in family life in terms of a biologically privileged worldview. We suggest that there is a great deal of difference between ‘early intervention’ as defined in the Allen report and what Munro (2011: 69) refers to as ‘early help’, which includes a much wider range of family support activities. Neuromania, we conclude, is the latest of modernity’s juggernauts reifying human relations into ‘technical objects’ to be fixed by the state (Smith, 2002), which always ‘asks nothing better than to intervene’ (Ellul, 1964: 228).

Making the meme: ‘using our brains’

The neuroscientific strand of Allen’s (2011a) argument begins, as noted, with the imagery on the cover (Figure 1), and the case for early intervention quickly starts to take shape in Chapter 1. Paragraph 17 (p 6) prefigures the argument and its overwhelming ‘now or never’ urgency (emphasis added):

The early years are *far and away* the greatest period of growth in the human brain. It has been estimated that the connections or synapses in a baby’s brain grow 20-fold, from having perhaps 10 trillion at birth to 200 trillion at age 3... The early years are a very sensitive period ... after which *the basic architecture is formed for life* ... it is not impossible for the brain to develop later, but it becomes significantly harder, particularly in terms of emotional capabilities, which *are largely set in the first 18 months of life*.

Chapter 2, ‘Using our brains’, is devoted to detailed elaboration of the neuroscientific evidence. Its epigraph reiterates the diminishing capacity of the brain ‘to adapt and change throughout life’. A footnote cites three journal papers, the titles of which are as follows: ‘Adaptive auditory plasticity in developing and adult animals’, ‘Cortical plasticity: from synapses to maps’ and ‘Experience-dependent plasticity in the adult visual cortex’. Given the burgeoning corpus of neuroscience research, this is a puzzling selection. All papers clearly relate to ‘plasticity’, two explicitly in the context of the *adult* brain. Such plasticity would seem to contradict the idea of a brain ‘formed for life’ (and this is what the neuroscience shows, as we shall see) but Allen presses on undeterred. Into the neuroscience warp is soon woven the web of attachment theory. The importance of secure attachment is invoked:

Children develop in an environment of relationships... From early infancy, they naturally reach out to create bonds, and they develop best when caring adults respond in warm, stimulating and consistent ways. This secure attachment with those close to them leads to the development of empathy, trust and well-being. (2011a: 13)

Predictive claims quickly follow regarding the long-term effects of such early attachment patterns, especially the beneficial effects of secure attachment and the dire impact of the failure to cement such bonds:

Recent research also shows insecure attachment is linked to a higher risk for a number of health conditions, including strokes, heart attacks ... people with secure attachment show more healthy behaviours such as taking exercise, not smoking, not using substances and alcohol, and driving at ordinary speed. (2011a: 15)

Two studies are cited as the basis for these ominous claims. But again the evidence cited is perplexing. These are not studies of children, but adults; both use 'attachment style' as a way of measuring the adult personality with self-report questionnaires. Neither study shows, nor purports to show, any link between early childhood experiences and problems later in life. In subsequent paragraphs, damaged emotionality and damaged brains are soon united, and the perpetrator of all this devastation is unflinchingly denounced. Parents are to blame:

Parents who are neglectful or who are drunk, drugged or violent, will have impaired capacity to provide this social and emotional stability, and will create the likelihood that adverse experiences might have a negative impact on their children's development ... the worst and deepest damage is done to children when their brains are being formed during their earliest months and years. (2011a: 15)

Blaming parents is nothing new in contemporary family policy (Furedi, 2001), although Allen adds a novel twist. Returning to the cover image, of the neglected brain, how else can the report be read, other than to show the damage done by irresponsible parenting: figuratively, you can see the images being jabbed in the feckless mother's face – look at what you're doing to your child!

What then of these images and their potent moral rhetoric? The report attributes them to the Child Trauma Academy (www.childtrauma.org), a child advocacy organisation run by Bruce Perry. A reference is given to an article by Perry (2002) published in *Brain and Mind*, a 'Transdisciplinary Journal of Neuroscience and Neurophilosophy' with questionable credentials.¹ The critical section of the paper (p 92) is less than a page long, with but the sketchiest of methodological detail; no clinical histories are given. Two main groups of cases are distinguished: children suffering from 'global neglect' (minimal exposure to language, touch *and* social interaction) and children suffering from 'chaotic neglect' (the sort of neglect routinely encountered

in UK services). Measuring head size as a proxy for brain size, Perry's main result was that the head sizes for the globally neglected children were extremely abnormal, whereas those suffering from chaotic neglect were within the normal range. Brain imagery then takes its bow:

Furthermore in cases where MRI [magnetic resonance imaging] or CT [computerised tomography] scans were available, neuroradiologists interpreted 11 of 17 scans as abnormal from the children with global neglect (64.7%) and only 3 of 26 scans as abnormal from the children with chaotic neglect (11.5%). The majority of the readings were 'enlarged ventricles' or 'cortical atrophy' (see Figure 1). (Perry, 2002: 92)

Figure 1 is none other than the cover image of Allen's report. Without details of the case history for the neglected child, such an image is meaningless; perhaps the child was the subject of massive birth trauma, or some congenital condition. We simply do not know. Returning to Allen's report, the following excerpt summarises the final step of his neurobiological argument:

Different parts of the brain develop in different sensitive windows of time. The estimated prime window for emotional development is up to 18 months, by which time the foundation of this has been shaped by the way in which the prime carer interacts with the child. . . . Infants of severely depressed mothers show reduced left lobe activity (associated with being happy, joyful and interested) and increased right lobe activity (associated with negative feelings).

If the predominant early experience is fear and stress, the neurochemical responses to those experiences become the primary architects of the brain. Trauma elevates stress hormones, such as cortisol. One result is significantly fewer synapses. Specialists viewing CAT scans of the brains of abused or neglected children have likened the experience to looking at a black hole. In extreme cases the brains of abused children are significantly smaller than the norm. (Allen, 2011a: 16)

Those damaged brains again. For the claim of lasting damage from fear, stress and trauma, Allen cites no specific scientific support. A significant body of work does, however, exist on the possible damage caused by post-traumatic stress disorder, reviewed by Wang and Xiao (2010). Although there is evidence of reduced volume in one brainstem structure (the hippocampus), the seminal research involves war veterans, not children; follow-up studies have not shown lasting hippocampal damage, and the scant imaging research on children has failed to find such impact. A recent authoritative review (McCrory et al, 2012) comes to much the same conclusion regarding the hippocampus, and another much-mentioned brainstem structure, the amygdala; only under conditions of prolonged rearing in orphanages is diminished brain size evident (see below).

Digging into the specific (frontal) lobe evidence invoked by Allen, he cites a paper by Dawson et al (1994), which reviews psychophysiological studies of the children

of depressed mothers. Dawson's evidence, however, actually goes in the opposite direction to that claimed in the Allen report. Referring to a study on the reactions of children when mothers left the room: 'the infants of symptomatic mothers exhibited an unexpected pattern of greater left than right activation during the maternal separation condition' (Dawson et al, 1994: 772). More 'positive' emotion it would seem. In truth, there is a vast gallimaufry of neuroscience research, but little settled knowledge. Evidence for policy making does not simply repose in journals 'ready to be harvested' (Greenhalgh and Russell, 2006: 36). Rather, it is 'rhetorically constructed on the social stage so as to achieve particular ends' (Greenhalgh and Russell, 2006: 37). This seems an apt enough description of Allen's modus operandi. Although 'journal science' is invoked, he seems not much interested in what it actually says. This is 'prejudice masquerading as research' (Furedi, 2001: 155), of science being enrolled to legitimate an a priori ideological position favouring a larger arena for public intervention in the lives of families.

The myth of the first three years

I, George Bush, President of the United States of America, do hereby proclaim the decade beginning January 1, 1990, as the Decade of the Brain. I call upon all public officials and the people of the United States to observe that decade with appropriate programs, ceremonies, and activities. (George Bush, Presidential Proclamation 6158, July 17th, 1990, <http://www.loc.gov/loc/brain/proclaim.html>)

In child welfare policy, as in many other areas, the UK has been led by prior developments in the United States (US). In the 'Decade of the Brain', US education policy was strongly influenced by neuroscience, in particular the idea that the first three years of a child's life are critical. In a withering critique, Bruer (1999) traces these policy developments, showing how they were shaped by the early intervention campaign groups and their misuse of science. Bruer charts a slew of programmes in education, welfare and healthcare, highlighting their neurodevelopmental foundations: 'The findings of the new brain science have become accepted facts, no longer in need of explanation or justification, to support childcare initiatives' (1999: 61).

In this section, we draw on Bruer's deconstruction, updating and developing various points. Bruer identifies three neuroscience strands running through the policy discourse:

- the early years represent a period of 'biological exuberance' in brain development, characterised by an explosive growth in synaptic connectivity;
- this constitutes a once-and-for-all 'critical period';
- more stimulating environments can boost 'brain power'.

Woven together, these strands created a potent neurobiological meme, but Bruer argues that the critical importance of the first three years is a myth: it is powerful because

it promises to solve so many social problems, but it is based on oversimplification and misinterpretation.

Let us now follow Bruer's argument. First, the synaptic strand. What does the scientific record actually say? The picture is not straightforward. Counting synapses is a technically tricky and expensive business, involving the laborious analysis of tissue specimens. It is unsurprising that there are very few such studies, and these involve mainly cats and monkeys. Initial synaptic exuberance in the early years is only part of the story. There is a second stage in which the number of synapses reaches a plateau, followed by synaptic pruning in which densities decline to adult levels. The time-course also varies by brain area. The argument that there is a simple, proven connection between brain power and synaptic profusion is not sustainable. It is abundantly clear that more synapses does not mean greater intellectual prowess in any simple way. Indeed, just at the point in adolescence when humans begin to master increasingly complex bodies of knowledge, their synapses are undergoing mass elimination.

The other elements of the myth do not stand much stress testing either. The second strand of the myth is the idea that there are critical periods (or sensitive periods as they are now known) in the brain's development. Here the iconic neuroscience is that of the Nobel Prize winners, Hubel and Weisel, on the visual cortex of cats, which showed that kittens deprived of input in one eye remained permanently blind in that eye. This is evocative stuff: the image of 'blind little kittens pathetically groping for a ball of yarn' really does 'ratchet up the guilt' (Bruer, 1999: 102–3). Again, all is not what it seems: what is actually happening is not so much loss of capability for the deprived eye, but the annexing of this surplus capacity by the functioning one. Tellingly, when both eyes are closed at birth, experiments have shown no permanent damage. So, what the neuroscience actually shows is a highly plastic and adaptable brain. Bruer goes on to reinforce this through research on language acquisition, where critical periods are the exception; given the capacity of the human for lifelong learning why, he asks, would anyone want to construct critical periods as the norm?

Research on sensory deprivation in animals is often invoked to bolster the myth, and attachment theory is also recruited in this context, as in Allen's report. However, the evidence at the time of Bruer's book fails to demonstrate any causal connection between secure attachment and specific parental behaviours, or that attachment patterns, once formed, are stable and set forever. Reliable predictions can be made only in *situations where childrearing conditions have remained the same*. Recent attachment research supports this position. Levendosky et al (2011) looked at the impact of domestic violence and income on attachment patterns at ages one and four. The study concluded that attachment was unstable for 56% of the sample, and that positive changes in attachment were related to lower domestic violence or rising income, and vice versa.

The third neurobiological strand of the myth is succinctly encapsulated in the following quote from a key pamphlet cited by Bruer (1999: 144): 'Early experiences can have a dramatic effect on this brain wiring process, causing the final number of synapses in the brain to increase or decrease by as much as 25 percent.' The canonical neuroscience supporting this is that of Greenough and colleagues (see Bruer 1999: 145–52) who studied the effect of three types of environment on rat brain development:

the rat in a small cage by itself (isolated), in a larger cage with companions (social) and in a larger enclosure with obstacles, toys etc (complex). Examining the brains of rats raised for 30 days post-weaning in these environments, roughly 20–25% more synapses were found in the visual cortex of the ‘complex rats’. So far so good for the myth, but let us look closer. Yes, there were more synapses for the ‘complex rats’; however, the difference was much less for non-sensory areas of the brain, the frontal lobes in particular, which are supposedly associated with higher cognitive functions. Bruer also notes that the weanlings were the equivalent of 50 days old after their period of deprivation, well into advanced childhood in human terms. The conditions experienced by the rats were also somewhat extreme compared to their life in the wild, even in the complex condition. Reading ‘Palo Alto’ for complex and ‘South Bronx’ for isolated is totally absurd, quips Bruer (1999: 146). Further experiments showed that increases in dendritic density as a result of ‘enriched experience’ can be shown at any age: adult rats raised in ‘deprived’ conditions and then placed in a complex environment at 120 days had the same synaptic density as the weanlings of the earlier studies.

Bruer moves on to consider the impact of early intervention programmes for children. In the myth literature of the time, two programmes stood out: the North Carolina Abecedarian Project and Infant Health and Development programme, a national study. The first of these was targeted at 100 high-risk, low-income families and children were divided into two main groups: an ‘untreated control group’ and the intervention group, who received intensive support from four months old until they entered school five years later, and continued thereafter. Early in the evaluation, IQ differences of between 10 and 18 points were observed between the two main groups, but these rapidly diminished over time; by the age of 15 the advantage was 4.6 points, and this despite an eight-year programme of tailored, intensive support. Similarly equivocal results derived from the Infant Health and Development programme. But even had these schemes proved effective, intrinsically they provide no clear-cut answers regarding the importance of the first three years. Duration, timing and programme content are inextricably confounded in their implementation; it is impossible to know which was critical.

Bruer’s polemical deconstruction of the myth provoked a reaction from its votaries. While conceding that his critique ‘won the plaudits of influential critics ... mak[ing] many valid points’, Stien and Kendall (2004: 2009) seek to dismiss his argument on grounds of motive, as springing from ‘the desire to please parents and appease guilt feelings’. More authoritatively, Rutter (2002: 13) has taken a different view. In his presidential address to the Society for Research on Child Development, he inveighs against the ‘evangelism’ behind claims that ‘early years determine brain development’. He goes on, explicitly invoking Bruer: ‘As several commentators have pointed out, the claims are misleading and fallacious ... the assumption that later experiences necessarily have only minor effects is clearly wrong’ (Rutter, 2002: 13).

We conclude this section with a necessarily brief and selective overview of ‘post-Bruer’ developments on the effectiveness of early years intervention, first in the US and then in the UK. These programmes range from educationally focused interventions, dominant in the US, to more family-based, social interventions in the UK, such as

Sure Start. These programmes share an orientation to early years as a critical period. In 2000, an impressively rigorous and nuanced report of the National Research Council and Institute of Medicine was published, entitled *Neurons to neighbourhoods* (Shonkoff and Phillips, 2000). It stated: 'Taken together, the follow-up literature provides abundant evidence of intervention-control group differences in academic achievement during middle childhood' (2000: 351) but 'for children at risk because of low socioeconomic status, *the short term benefits of higher IQ scores typically fade out during the middle childhood years*' (2000: 378, emphasis added). Although striking a generally positive tone, the report acknowledged the deficiencies of the knowledge base, including the publication bias against null results and the inattention to 'the much larger number of measured outcomes that demonstrate no program-control differences' (2000: 351). It also noted the failure to find consistent or distinctive benefits associated with a particular type of intervention, and lamented the limited data regarding cost-benefit trade-offs. There was also a mark-worthy acknowledgement that life may not be quite as simple as the early interventionists like to think: 'the premature initiation of services may lead in some circumstances to inappropriate labeling or the removal of children from typical experiences, thereby reducing the possibility of self-righting corrections or compensatory growth spurts' (2000: 364).

Moving forward, we consider the results of more recent evaluations. In January 2010, the final report of the 'Head Start Impact Study' (one of the longest-running programmes to address systemic poverty in the US) was published. While there was evidence of 'a positive impact on children's preschool experiences' (US Department of Health and Human Services, 2010: xvi), the picture that emerged was again one of transient benefits: 'However, the benefits of access to Head Start at age four are largely absent by 1st grade for the program population as a whole. For 3-year-olds, there are few sustained benefits, although access to the program may lead to improved parent-child relationships through 1st grade (US Department of Health and Human Services, 2010: xxxviii).

Back on this side of the pond, how does the evidence stand? A recent evaluation of the benefits of early years education concluded that 'large sample results indicate that on average attending early-years education had no impact on any of our outcome measures' (Hopkin et al, 2010: 47). The results showed some gains for disadvantaged groups, but the effects did not achieve statistical significance. And what of Sure Start? The most recent evaluation (Meadows, 2011) found that local programmes cost £4,860 per child but delivered quantifiable economic benefits less than 10% of this figure; so, no 'gold bars' here. The report speaks of the 'potential to generate economic benefits in the future' (2011: 1), but potential is hardly actual. Crucially, *families* do appear to have gained, but to find these benefits we need to look, not at the intracranial spaces of the infant brain, but at the child within the relational ecology of the family. Evaluation of family support activities within Sure Start revealed here-and-now benefits in 'coping' and 'caring' (eg, Tunstall et al, 2005; Featherstone et al; 2007; Frost and Parton, 2009), but such results provide neither evidence that irreversible brain damage has been prevented, nor evidence that the first three years are critical in terms of neural development, nor do they purport to do so.

The brain seduction

Developmental neuroscience is an intoxicating ingredient in contemporary UK policy. Here, we dwell on this enchantment, aiming to break its spell. In a recent paper on the persuasive power of brain images (Ramani, 2009), several studies are described; that by McCabe and Castel (2007) is particularly notable. Undergraduates were asked to evaluate some fictitious and highly implausible news articles on brain research. The evidence presented included no image, a brain image or a bar graph depicting the critical results. When asked to rate the credibility of the reports, those including brain images were rated consistently higher than those without. Brain images ‘shout science’ (Poerksen, 1995); they are ‘a fast acting solvent of critical faculties’ (Tallis, 2011: 280). This is especially so for those produced by functional MRI scans, which display a topography set out in colour schemes like real-world maps, thereby greatly adding to their verisimilitude. That such images are the result of very complex processing and dependent on a technology that is unimaginably sophisticated, and yet crude in terms of what it tells us about the brain, is quite invisible to the enchanted viewer. The images are more real than reality itself, hyper-real indeed (Baudrillard, 1994).

So potent is the spell cast by the images, the limitations of the experiments are dissolved away. But the crudeness is often risible and is lampooned by Tallis, who gives several examples, including a study on ‘the neural basis of unconditional love’:

Care assistants were invited to look at pictures of people with intellectual difficulties first neutrally then with a feeling of unconditional love. By subtracting the brain activity seen in the first situation from that seen in the second, the authors pinned down the neural network housing unconditional love. (Tallis, 2011: 74)

The paper reels off the following brain regions as making up this network: ‘the middle insula, superior parietal lobe, right periaqueductal gray, right globus pallidus (medial), right caudate nucleus (dorsal head)...’ (Beauregard, 2009: 93). The list of Latinate names suggests real knowledge, but this is kitsch science (Lugg, 1999). The underlying logic of ‘cognitive subtraction’ (Legrenzi and Umiltà, 2011) depends on the simplistic assumption that there is some discrete psychological quality (of unconditional love, as opposed to other kinds of love) that can be independently isolated by subtracting one mental state from another. Equally untenably, it relies on the a priori assumption that brain states can be decomposed similarly into atomic elements, which also combine additively and correspond one to one with psychological states.

Brain scans embody the idea that different parts of the brain do different things. Naturally, we believe this must be so, after all this is a characteristic of all the complex technologies devised by man. The idea has a long history, going back to Broca who in 1861 described a patient with a cerebral lesion who could only say ‘tan’. The post-mortem located the lesion in the left frontal lobe and since then it has been a ‘known fact’ (Legrenzi and Umiltà, 2011: 3) that this region is responsible for speech production. Broca thus established the principles on which modern neuroscience relies, that the brain can be divided into different areas with different functions, which

are independent of each other and can be isolated. But how valid is this foundational notion? Let us take the neuroscience of violence as our example, as it is particularly relevant to contemporary social policy. Two brain areas are constantly invoked: the frontal lobes and the amygdala. But as Pustilnik (2009) notes, research studies have shown the frontal lobes to be involved in every conceivable cognitive process: general intelligence, problem solving, executive control, attention, decision making, semantic memory, perceptual analysis and self-awareness. Regarding the amygdala, Pustilnik observes that '[n]umerous respected brain researchers question the localisation of fear to the amygdala ... because the amygdala can be activated by many events that have no relation to fear' (2009: 221). If the subject is shown a picture of scrambled eggs in an experiment to identify playing cards: 'Your amygdala will light up. This is not because the subject is afraid of scrambled eggs but because the picture is unexpected' (2009: 221).

Pustilnik is right to be sceptical. The alacrity with which neuroscientists have employed brain imaging has led to a torrent of claims, which play easily with a credulous public. Tallis (2011: 75) mocks such claims: 'They seem like brochures from the Grand Academy of Lagado in Gulliver's Travels ... as manifestations of neo-phrenology'. The term *neophrenology* is an apt one; it was coined by the neuroscientist William Uttal. Uttal is a longstanding critic of attempts to use technologies to understand the brain, which perforce give only a crude, macro-level map of its activity. Functional brain imaging is like trying to understand how a complex organisation works by measuring the electricity usage in different rooms; this would tell us something about how activity is distributed and its variation over time – it would say nothing about the actual nature of the work done. Uttal (2011) identifies two insuperable difficulties for the neuroscience project of explaining psychological processes in terms of brain activity. First, that psychological processes are not modular, that is, they cannot be decomposed into constituent elements (ie violence does not exist on its own as an independent psychological component); nor indeed can brain activity: 'Brain activity associated with mental activity is broadly distributed on and in the brain. The idea of phrenological localisation must be rejected and replaced with a theory of broadly distributed neural systems accounting for our mental activity (2011: 18). So, despite all its 'sound and fury', the reductionist project of the neophrenologist is doomed; there are no discrete modules, at either level, mind or brain. Uttal (2011) also uses violence to illustrate his argument about the distributed nature of brain activity, drawing on a review of 17 brain imaging studies of aggression. Thirty-three different brain regions were identified in this work, only one of which was mentioned more than twice. To the question, which brain regions subserve violence, it is impossible to disagree with Uttal's answer: 'Pretty much the whole brain' (2011: 173).

It should now be clear that neuroscientific knowledge is at an early and provisional stage. As Bruer (1999: 98) avers, after more than a century of research we are still 'closer to the beginning than the end of this quest'. This point was reinforced recently by Belsky and de Haan (2011: 409–10): although the brain 'packs a punch' for policy makers, they conclude that 'the study of parenting and brain development is not even yet in its infancy; it would be more appropriate to conclude that it is still in the embryonic stage'. Neuroscientists may know the limitations of their research, but

such caveats are not what politicians and proselytisers wish to hear; neuroscience has not only blinded the kittens.

Moral judgements, child welfare and biology: old wine in new bottles?

Modernity has already hijacked many moral issues – abortion, death, reproduction, intimate relationships, poverty, oppression, parenting – and translated them into ethical codes that are not designed for moral debate but for public consumption ... once citizens cede their moral responsibility to the state, they accept regulation in place of moral choice. (Smith, 2002: 19)

The idea that moral defects have a medico-biological cause, the ‘medicalisation of morality’, goes back to Victorian times (Rimke and Hunt, 2002). It incorporates the idea that parents pass on such defects, that they are ‘antisocial upshots of a process of degeneration in their descent’ (Rimke and Hunt, 2011: 77). An article in the *British Medical Journal* in 1857 opined that ‘[i]t is a long known fact that drunkards have idiot children in a far larger proportion than sober people ... drunken parents cannot transmit a healthy organization to their descendants’ (Rimke and Hunt, 2011: 78). Such sentiments are not much different from those of contemporary policy, although the language is softened and medicalised by neuroscience. Seen in this light, the present argument for early intervention is part of a longer-term project of moral regulation.

The critical period is the cornerstone of this project. The biological factuality of such periods may be debatable; that some people believe they exist is certainly a fact. Kagan (1998) writes of the three ‘seductive ideas’ that inform the (neuro) developmental belief system. The idea that the first couple of years determine the rest of life is the foundational one. For the ‘infant determinists’, development is seen as ‘analogous to building a house’, and indeed the house metaphor is ubiquitous in their writings (Allen, 2011a: 14; Fox et al, 2010: 29). Despite all contrary evidence, such determinism exerts a powerful lure. As counter-evidence, Kagan (1998) gives the example of war orphans who, after several years of adoption, had achieved an intellectual profile similar to the average child. The widely cited study of English–Romanian adoptees reported its most recent follow-up in 2010 (Rutter et al, 2010). Again, the evidence is of remarkable plasticity and resilience, *especially of the brain*. Despite severe initial impairment resulting from extreme deprivation in orphanages, children showed a pattern of substantial cognitive recovery, still continuing in their mid-teens; and those with the worst deficits showed the greatest catch-up. Less well known are the results for the study’s various control groups. Children coming from a ‘severely deprived background but who had not experienced institutional care’ (2010: 212) did not differ from English adoptees. The deficits thus reflect some unique feature of the desperate conditions in the orphanages; they are not the inevitable sequelae of neglect in general. Even more saliently, orphans who experienced institutional deprivation for the first six months only showed no appreciable ill-effects. Evangelists

of early interventionists are in danger of being hoist by their own petard; if the damaged brain were to become the criterion for action, would intervention ever be justified?

Infant determinism is a powerful, totalising worldview. To experience its thrall, let us look at a recent review article, which constitutes something of a manifesto for the cause (Fox et al, 2010). The usual research on critical periods is invoked, and although the result we quoted regarding the occlusion of both eyes is mentioned, the obvious conclusion is not drawn. When plasticity is finally acknowledged, it is tellingly described (in a study of pups) as follows: 'Placement of deprived pups into an "enriched care" environment resulted in aptitude similar to high-care pups; however, hippocampal volume did not change, suggesting that plastic mechanisms form typical behaviour *despite lasting structural deficits* (2010: 36, emphasis added). Those who espouse the fixity of the brain show noteworthy fixity themselves, seemingly trapped in the incorrigible grip of their belief system. The pups are still irreparably damaged, even though they are not; their brains show it! In the final paragraphs there are signs of epiphany: 'Those working in the field of intervention' are exhorted to 'take stock of what is now known about neural plasticity' (2010: 36), but the paper still signs off with its core mantra intact: 'For the millions of children who begin their lives in adverse circumstances, we should *act with alacrity* to improve the lives of these children before neural circuits become well-established' (2010: 36).

No one would disagree that deprivation and violence are social ills that call for remedy. Nor do we argue that the prenatal environment and early life of the child are unimportant. Undoubtedly, adverse childhood experience can play a role in the subsequent aetiology of psychopathology, and the brain is not invulnerable to lasting physical damage. But the latter threats come from 'extreme deprivation, inadequate nutrition and neurotoxic exposures' (Shonkoff and Phillips, 2000: 198), such as iron deficiency leading to hypo-myelination. They do not arise as a necessary and direct consequence of chaotic family life, inattentive parenting or unconventional lifestyles. The developing infant brain is not a uniquely fragile object, a medical emergency waiting to happen. The danger of such medicalisation is its crushing of the debate we need to have as a humane society about where and how the state should tread, and its limits.

The mythological version of the infant brain is fast becoming part of the policy and practice of child welfare, easily invoked to profound rhetorical and material effect. We have attempted here to challenge this ascendancy and to demystify the pseudo-scientific 'expertise' of neuromania (Poerksen, 1995). It is the now-or-never part of the argument that is so threatening to real debate and progress, focusing on parental culpability without meaningful help: less practical aid, more parenting programmes (Furedi, 2001). It mandates the removal of children from families on seemingly incontrovertible, precautionary grounds, as illustrated in the following quote of Martin Narey (chief executive, Barnado's): 'More babies should be removed from their mothers at birth before irreparable harm is inflicted. There is an argument to be made ... that even intervening at this early stage is too late' (Walsh, 2010: 12).

Medicalisation transforms child welfare from helping-families-in-the-community to standardised pseudo-medical interventions, targeted packages of support based on

a drug metaphor (Stiles and Shapiro, 1989). The language of *Neurons to neighborhoods* shows this starkly:

Weighing the difference between costs and benefits in the determination of appropriate program ‘dosages’ is a critical policy challenge. Moreover, it is most important to recognize that the only way to provide definitive answers to questions about the relative impacts of the timing, intensity, and duration of service delivery is to conduct randomized experimental studies on specific populations. (Shonkoff and Phillips, 2000: 364)

Medicalisation of social care is not limited to damaged brains. It is manifest in the language of the ‘randomised experiment’. Again, there are profound implications in terms of policy and practice. We are not opposed to controlled experiments in social care; for ‘targeted packages’, which can be delivered like shots of a drug, they may be valid. Our concern is that the privileging of such interventions inevitably tends to downgrade or rule out approaches that cannot be so readily formulised and tested. The provision of social care to families often involves multiple interventions, with a cast of potential change agents. The drug metaphor assumes that interventions remain constant regardless of who performs them and of their relationship with the family, an assumption that Stiles and Shapiro (1989: 527) deem absurd (see also Cartwright and Munro, 2010). Specified indicators and the preoccupation with programme fidelity limit the range of possible improvisations, and artificially foreshorten the duration of professional involvement. Moreover, families who do not change in the ways specified in the indicators are deemed to have failed.

Inevitably, the shift from help to ‘screen-and-intervene’ (Rose, 2010) engendered by medicalised discourse pushes more children into the care system, with rates in the UK having doubled in the three years² since the death of (Baby) Peter Connelly. The need to avoid permanently damaged brains is used increasingly to justify such decisions. Asked to comment in a BBC interview (Radio 4, *Today* programme, 9 February 2012) on the recent unprecedented increase of removals, the President of the Association of Directors of Children’s Services observed that this was, in part, due to ‘a better understanding of the corrosive and damaging impact of neglect on children’s development ... it is about understanding the effect of neglectful parenting due to drug and alcohol problems and the physical damage to brain development it can do with very young children’.

But removal is not a risk-free, brain-boosting antidote to disadvantage and dysfunction. Bruer (1999: 173–4) invites us to consider the downside of the infatuation with the early years and why the thought-style must be cast off: ‘Overemphasizing the importance of the first three years ... amounts to thinking about and attacking problems from an artificially limited perspective and a limited armamentarium of possible interventions.’ We need practical help for families rather than moral panic about damaged brains. Questions about what is expectable of parents, what are acceptable levels of care for children and what the state can meaningfully offer, are moral ones and need informed, open debate. The playing of the neuroscientific trump card is stifling this, and that has been our case.

Neuroscientists and clinicians concern themselves with understanding the workings of the brain, the aetiology of neurodevelopmental disorders and eventually their work may produce new treatments, but currently the knowledge is not 'policy ready'. The research literature on the effects of stress and trauma on the brain is vast, for instance, often yielding contradictory results. In a review of studies relating to parenting, Belsky and de Haan (2011) conclude that much more work is needed to draw generalisable claims about cause and effect, or to add anything practically to what could be gleaned by conventional observation and treatment.³ Our position, then, is much the same as that of the distinguished neuroscientist Steven Rose (2011: 69, emphasis added), who asseverated in a recent Royal Society policy paper:

I would argue that any genuine increase in knowledge of brain processes ... can only enrich our understanding of ourselves. Nor can such increased knowledge replace or diminish the insights into what it is to be human that come from philosophy, the social sciences or the humanities — *therefore, there should only be benefits, providing one can pick one's way through the 'over-hyping' of apparent neuroscientific claims...*

Notes

¹ The Journal ran briefly from 2000 to 2003. Its contents seem somewhat idiosyncratic, for example the last issue contained an article entitled 'When did Mozart become a Mozart?'. Doubtless such pieces make a stimulating read, but this is not mainstream neuroscience. Perry's article is the journal's most downloaded piece.

² The increase from April to June 2008 to the same quarter in 2011 was 106% (Cafcass, 2011).

³ It is pertinent to ask what neurophysiological measures add unless accompanied by behavioural or emotional indicators of trauma or damage, and if these exist, what is neuroscience really contributing? In time, it may produce detailed understandings of aetiology and inform effective new treatments, but treatments that currently claim neuroscientific credentials (eg, Perry, 2009) draw on *theoretical* models for the repair of traumatised brains. They may work, but rely on behavioural or functional improvements to show it and use established therapeutic practices, rather than neurological interventions.

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