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Epigenetics Prematurely Born(e): Social Work and the Malleable Gene

Sue White and David Wastell

Department of Sociological Studies, University of Sheffield

Professor Emeritus, University of Nottingham Business School.

Correspondence to:
Sue White
Professor of Social Work
Department of Sociological Studies
Elmfield
Northumberland Rd
Sheffield
S10 2TN

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Abstract

Biological sciences are currently in the cultural ascent, promising to provide a theory of everything in the natural and social worlds. Beginning with the decade of the brain in the USA in the 1990s, neuroscience was first onto the stage, but developments in genomics, known as epigenetics have profound implications for society and culture, and the responses of the State to intimate family life and personal choices. Epigenetics provides an explanation of the mechanisms underpinning the interaction of the environment and the DNA blueprint, and thus invites an interest in the impact of adverse conditions, such as deprivation, or normatively deficient parenting. The implications of this biology of social disadvantage for social work are far reaching. Epigenetics is part of an increasingly political biology with the potential to affect the moral direction of social work. This paper reviews the state of the field and its immediate implications for the profession.

Key Words: Epigenetics, biology, early intervention, foetal programming, child protection, surveillance

Introduction

> *[F]or decades we've all been told: you are what you eat. You are what you drink. You are how much, or how little, you exercise…. And yet a quiet scientific revolution is changing that thinking. For it seems you might also be what your mother ate. How much your father drank. And what your grandma smoked. Likewise your own children, too, may be shaped by whether you spend your evenings jogging, worrying about work, or sat on the sofa eating Wotsits (Epigenetics: How to alter your genes , Chris Bell, The Daily Telegraph, 16 Oct 2013*

> *In practice, ideas and discoveries presented by biomedical science are often particularly compelling. Providers of services not only want good evidence to justify difficult decisions they have to make; they are also susceptible as anyone to persuasive nature of hard scientific 'facts' (Early Intervention Foundation, Feb 2016)*

These epigraphs are emblems of our time. Techno-sciences increasingly promise to provide a theory of everything and recent developments in genetics, known as epigenetics, have implications for society and culture, and the responses of the State to intimate family life and personal choices. Here, we examine the actual and potential applications of current understandings of epigenetics in social policy and social work. We interrogate the claims made within the research literature and examine their presuppositions.

We begin by briefly mapping the contours of our case to make clear what we are arguing and, crucially, what we are not. Across a raft of government policy in the UK and increasingly elsewhere, for example in the projects of the World Bank, there is a pre-occupation with the very beginning of human life (for example, All Party Parliamentary Group for Conception to Age 2 – The First 1001 Days, 2015). Discoveries at the molecular level create new aspirations for the Developmental Origins of Health and Disease (DOHaD) paradigm. In 2015 International DOHaD Society launched its manifesto. Economics, efficiency and productivity feature prominently in the narrative.

> *[A]n unhealthy lifestyle in prospective parents … perpetuates cycles of poor health, reduced productivity and shorter life expectancy, trapping populations in a trough of low human capital from which they cannot easily escape. (International DOHaD Society 2015: 1)*

The foci of proposed interventions are individual lifestyle 'choices' such as the consumption of unhealthy food, failure to take adequate exercise and the use of cigarettes and alcohol, which sit alongside concerns about environmental exposure to toxins or microbes. Psychological 'stress' is also being implicated more and more in the epigenetic discourse. Characteristic remedies run as follows:

> *Support optimal timing of pregnancy, healthy weight, good macro- and micronutrient status, physical activity, sleep and other behaviours in women and their partners before, during and after pregnancy …. Support breastfeeding, healthy complementary feeding, regular physical activity, a healthy lifestyle and parenting skills, to exploit critical windows of opportunity for the optimal physical and mental development of children … (International DOHaD Society 2015: 1)*

The benignancy of the aspiration to purge the next generation of debilitating or deadly disease is indisputable. Yet, the goal of epigenetic optimisation is freighted with moral and ethical implications. There is a clear focus on reproduction and maternal behaviours, paralleled by a pervasive lack of explicit reference to the alleviation of poverty and social disadvantage. Through such developmentalist reasoning, poverty can be recast as a biological phenomenon.

Epigenetics follows the path of a popularised version of neuroscience, which we and others have critiqued in detail (*inter alia* Wastell and White, 2012; White and Wastell, 2013; Gilles et al 2016; Macvarish, 2016). Powerful advocacy organizations have been working with public relations agencies for some time to produce 'a core story of development' (Shonkoff and Bales, 2011:17) to influence policy makers. This simplified neuroscience has been invoked to justify the funding of targeted early intervention initiatives, and has gained traction in the child protection system (White and Wastell, 2016).

Such campaigning has taken place well away from the activities of the laboratory scientists themselves, working on animals (mainly rodents) to attempt to unlock biological processes at the molecular level. This level might be very helpful in understanding the mechanisms giving rise to diseases such as cancer, and furthering knowledge of the impacts of toxins or infectious agents. We are not mounting a critique here of such work: much of it is fascinating and holds great promise. Things become rather more morally murky when policy-makers draw on such research to advocate behavioural changes, on the precautionary principle that a variety of ordinary 'choices' might be damaging the epigenome for subsequent generations. Under such a gaze, women potentially become 'eternally pre-pregnant' (Meloni, 2016: 217; Waggoner and Uller, 2015). As an example, the UK Houses of Parliament briefing on epigenetics and health stresses the need to equip practitioners with the competencies and skills needed to support behaviour change:

> *"Evidence from epigenetics research is [that] epigenetic changes are potentially modifiable through lifestyle and diet. Advice to pregnant women on behaviour change to avoid exposure to potentially harmful factors during early embryonic development is likely to be particularly important (Houses of Parliament 2013: 4)".*

Social workers are likely agents to carry such measures into effect, potentially creating policy and practice shifts away from social, material disadvantage, focusing instead on changing individual behaviour.

What is Epigenetics?

Whereas genetics has conventionally focused on the DNA sequence (the genotype), the newly flourishing field of epigenetics examines additional mechanisms for modifying gene expression in behaviours, traits, physical features, health status and so on (the phenotype). It provides a mechanism whereby the environment can influence an otherwise immutable DNA blueprint, and this naturally invites interest in the impact of adverse conditions, such as deprivation or normatively deficient parenting. The implications of this new "biology of social adversity" (Boyce *et al.*, 2012) for social policy are potentially profound. 'Hard' heredity, in which inherited characteristics were seen fixed for life, inspired the eugenics movement of the late nineteenth and early twentieth century. Subsequently, the 'barbarous utopia' of the Nazis (Meloni, 2016: 28) severed biology from the acceptable face of politics and social reform. But epigenetics now shows signs of rendering biology politically acceptable again, with implications for the moral settlement in which social work takes place.

In the scientific literature, a number of epigenetic mechanisms have been identified, integral to the functional biology of multi-cellular organisms, such as those which maintain cell specialisation. Here we focus on the two 'celebrities' of the literature: DNA Methylation and histone modification, both of which can change gene expression, in the popular argot, by 'switching genes on or off'. Both work on the gene transcription sites, where RNA is produced for protein synthesis, with methylation attenuating expression, and histone modification either augmenting or diminishing it. These epigenetic inscriptions have been shown to respond to varying environmental conditions. We may presume such changes to be adaptive, but they can be seen as the source of dysfunctional consequences in the longer term. Our focus is on humans, but as noted, the vast majority of the primary science is on animals; the crux of the argument flowing from this work is as follows:

> *Classic studies in rodent animal models and humans have demonstrated a close relationship between elevated stress in early life and the appearance of behavioural disorders in later life (Cunliffe, 2015 p. 59).*

It will be instructive now to examine the influential studies in the field.

Tales from the Rat Lab

The seminal studies of US researcher Micheal Meaney (inter alia, 1985; 2001; Weaver et al 2004) on the maternal behaviour of laboratory rats has been especially influential in the domain of policy and practice. Meaney's work showed that variations in the degree of maternal nurturance ("licking and grooming") affected methylation patterns in their hippocampi. These epigenetic alterations could be reversed by cross-fostering with more attentive mothers. Meaney and his team have undertaken multiple complex experiments involving various manipulations of the rats: handling the pups, stressing the pups, stressing the mothers and so forth, typically assessing the effects post-mortem on methylation levels in the brain. When remedies were applied, they are characteristically pharmaceutical.

We have discussed the limitations of these experiments elsewhere (Wastell and White, forthcoming). There are important caveats on what may be extrapolated, but these constraints are seldom acknowledged by those anxious to draw conclusions for policy and practice. Here we focus on the moral direction of travel, by examining a further animal study, by Roth et al. (2009). This experiment had an explicit focus on, so-called, dysfunctional parenting behaviour by rat mothers. Mothers were subjected to severe stress immediately after giving birth. They were placed in an unfamiliar environment with scant nesting material. When their rearing behaviour was compared to unstressed mothers given adequate nesting materials, it was labelled by the experimenters as "abusive" (Roth et al., p. 3):

> *pups were frequently stepped on, dropped during transport, dragged, actively rejected, and roughly handled. Additionally, pups were often neglected (p. 4)*

What is more, 'maltreated' female pups went on, as mothers, to display "significant amounts of abusive behavior towards their offspring" (p. 6), and also showed a tendency for low posture nursing positions. These tendencies correlated with methylation levels which are described as aberrant. A drug (zebularine, a methylation inhibitor) was infused into the prefrontal cortex of "abused" pups and was found to reduce methylation levels to 'normal'. The authors' conclusion is noteworthy:

> *[E]pigenetic mechanisms continue to be linked with neuronal plasticity and psychiatric illnesses… This raises the intriguing speculation that such interventions as … treatment with DNA demethylases or histone deacetylase inhibitors, might prove useful as therapeutic strategies for reversing persisting effects of early-life adversity (p. 8).*

Surely a more appropriate treatment, following directly from the ecology of the experiment, would be the provision of adequate nesting resources: are we really to conclude that the best remedy for (inflicted) poor housing is the pharmacological treatment of one's offspring?

Epigenomic Surveillance and the privations of the mother

Let us now examine the state of knowledge in human studies. We begin with the iconic research on Dutch Hunger Winter. The long-term effects of the winter of 1944/45 on the health of foetuses exposed to the drastic food rationing in the final months of pregnancy (imposed by the German army on the people of western Netherlands) are well documented. Babies were born small, metabolic effects and ill-heath persisted throughout life (Carey, 2011). Cunliffe (2015) summarises some key results as follows.

> *People who had been exposed to the food blockade as foetuses had increased likelihoods of developing diabetes, mood disorders, and obesity as adults. Moreover, fetal exposure to famine was associated with altered patterns of DNA methylation near genes likely to be involved in fetal growth and development (p. 62).*

Here we examine some key aspects of the research, much initiated by Lumey (inter alia, Lumey, 1992; Lumey et al, 2011). First, we note that the babies' head circumference was not affected by the famine, so we may conclude the brains were likely in good shape. Focusing on the finding of decreased birthweights, Lumey (1992) reports data on deliveries before, during and immediately after the famine. There are striking reductions in birthweight, declining from 3261 grams on average for babies born prior to the blockade, to a low point of 3059 grams for babies experiencing the famine in the first and second trimesters of gestation. Birthweights increased for subsequent cohorts. From these results, it would seem that malnutrition during early pregnancy was the most damaging. But the situation is not that simple. Lumey also reports data for areas of the country free from the blockade; a trend of declining birthweight was found for these areas too, indeed the trend seems, if anything, to be stronger (Wastell and White, forthcoming).

The most recent follow-up study (Scholte et al. 2012) comments on these parallel trends. The winter of 1944/45 was an unusually harsh one for the whole of the country (backed up by still-birth rates and mortality figures), and food shortages affected some cities in the east. In general, the size of the effects uncovered is small: two statistically significant effects are reported: an increased risk of unemployment associated with trimester 1 exposure to famine (27% compared to 24%), and an increased risk for cardiovascular disease for trimester 2 exposure (13.5% vs. 12.9%). On the face of it, these increased risks are noteworthy. Yet it must be remembered that this is study is based on very large samples; the effect sizes and levels of significance are low. The authors themselves acknowledge "that the magnitude of the estimated long-run effects is small" (p. 13). In terms of our

understanding of health and economic disadvantage, the results of the famine study, if anything, seem to show that hardship of this sort actually appears to have relatively little impact compared to other factors, such as lack of money and housing conditions. This is a message to which social work would do well to attend, for there is much "sound and fury" arguing in another direction entirely. The contemporary gaze is turned inwards, not outwards, being particularly preoccupied with the inside of women's bodies during the reproductive years.

Foetal Programming: The Inhospitable Womb

Studies on 'foetal programming' by adverse 'maternal mood' burgeon; a trawl on Google Scholar, for example, yields 30,000 hits for foetal programming. The focus on maternal mood is a little odd though, as the foetus is relatively protected from maternal stress hormones by placental enzymes. Animal studies have shown that this protective mechanism can be damaged by 'inflammation' (Edwards et al 1993) and chronic, severe stress, but given the range of environments in which human beings have thrived, it would seem such mechanisms generally work well. We must ask ourselves, why this preoccupation with stress in utero. Moreover, where does this lead us, as a society?

The animal studies are set up to provide a proxy for human parenting. The normative assumptions they embody about maternal attentiveness make it clear that understanding laboratory rat parenting, and its response to stress, is not the primary motivation. The rats can be subject to extreme stress, even torture. They are typically killed at the end of the experiment so that the condition of their brains can be assayed. There are, of course, ethically insurmountable difficulties in undertaking such experiments with human subjects! Animals are thus treated quite differently from human beings, yet at the same time, claims are made that they provide models which can reliably inform understandings of human parent-infant interactions. Despite the obvious gulf between laboratory and housing estate, human studies of the effects of early adversity have burgeoned in wake of the rat work. A swathe of policy makes increasingly unequivocal claims about the adverse effects of pre and postnatal natal exposure of the foetus/neonate to maternal stress.

What exactly is the status of the knowledge in this field? The effects of antenatal depression and anxiety, and associated raised levels of cortisol on the developing HPA axis (the hypothalamic–pituitary–adrenal axis) of the foetus is the hypothesis of choice, uniting epigenetic and neuroscientific modes of inquiry (inter alia Charil et al, 2010; Field 2011; Glover et al, 2010; Glover and O'Connor, 2002). The majority of the studies draw on the work of Meaney and colleagues, which we mentioned above. A thorough review paper (Field 2010) summarises the results of a range of studies on the uterine environment of depressed women. These show associations with low birth weight and shorter gestational age, with babies are described as less responsive to stimulation: they cry more and sleep less. The low birth weight is, in turn, linked with hypertension, diabetes and coronary heart disease in adulthood, as well as depression and anxiety. It is all rather alarming, particularly, we might speculate, to pregnant women

Much of the epigenetic research has focused on glucocorticoid (GC) receptors in the forebrain, and the *NR3C1* gene which encodes the GC protein; these receptors are believed to play a key role in the regulation of the "stress response", mediated by the HPA axis. Oberlander et al (2008) found an association between maternal depression and methylation of a small number of sites of the *NR3C1* gene in the human cord blood of both mothers and neonates, and an augmented salivary cortisol response in the children at 3 months. Whereas the headlines of epigenetic narrative speak alarmingly of long-term harm, studies like this demonstrate only short term effects. Moreover, these are studies in humans, not rats in controlled laboratory conditions. Glover et al (2010) conclude that whilst animal studies 'show convincingly' a long term effect on the HPA axis of prenatal stress 'equivalent work in humans is only just starting' (p21). Charil et al, 2010 also cite numerous rodent studies and point to the relative paucity of, and methodological difficulties in, controlling the environment and stressors in human studies. Outside of concentration camps and war zones the extreme experiences of the rats cannot have a human equivalent.

There has been some painstaking work looking at the effects of maternal stress caused by natural disaster, which is as close as we can get to the rat experiences. As well as the Dutch Hunger Winter, of note is a longitudinal study on a cohort of children born to mothers who had suffered the extreme privations of the 1988 Quebec ice storm which resulted in power outages, leaving residents to face severe cold and shortage of food for up to six weeks. Researchers were able to study the effects of 'objective stress' - the number of days they were without power and damage to their homes - and disambiguate this from the subjective stress reported by the mothers. Objective stress, rather than the mothers' psychological condition, explained most of the impact on the children in IQ, language, BMI and obesity, insulin secretion and their immune system. The variance is attributed to epigenetic changes. The team note:

> *These data provide first evidence in humans supporting the conclusion that PNMS (prenatal maternal stress) results in a lasting, broad, and functionally organized DNA methylation signature in several tissues in offspring. By using a natural disaster model, we can infer that the epigenetic effects found in Project Ice Storm are due to objective levels of hardship experienced by the pregnant woman rather than to her level of sustained distress (Cao-Lei et al, 2014: 1).*

The ice storm studies, like those on the Dutch hunger winter, lack the normative tone of much of the foetal programming narratives. Mothers subject to natural disasters are not to blame, even if their subjective stress levels are also high. Their privations are severe and tangible, that they have enduring effects on the foetus is thus rather unsurprising.

Much of the work on prenatal depression and anxiety lacks such nuance. As Field notes:

> *…none of the studies have explored the antecedent of depressive episodes as, for example, if the prenatal depression was ongoing from childhood or related to recent losses or traumas, pregnancy factors, illnesses, or family history. And, investigators did not ask about depression before pregnancy (p11)*

Then there are the potential influences of antidepressant and other psychotropic medication, and we could go on. People live in complex systems. The studies tend to treat the womb as a sealed biological space in which the foetus is trapped against its will, as a sci-fi pod connected to an inhospitable endocrine bath. It is not connected to the environment, or to even to the woman and her relationships, life, love or loss. Examining the effects of prenatal stress solely at the molecular level seems curiously inhumane. Where does the vast field of research leave us in terms of social interventions? The knowledge that one's unhappiness is hurting one's baby is unlikely to result in a surge of positive hormones, a conundrum which has not entirely escaped attention. Glover et al (2010) note research by Gutteling et al (2005) which showed raised cortisol in young children whose mothers had experienced antenatal anxiety about giving birth to a disabled child. Worrying about their unborn child it seems has 'hurt' their child.

In a further example, Buss et al (2010) focus on the effects of 'pregnancy anxiety' on brain morphology in 6-9 year old children. Pregnancy related anxiety was measured at 19, 25 and 31 weeks using a scale, which assessed worries about pregnancy, health of the baby and fears about the delivery. These were 'worried well' women; they did not have diagnosed mental disorders. From the original sample of 557, 35 women agreed to MRI scans of their children. The children are described as healthy, and no emotional or behavioural difficulties were evident. Notably, cognitive ability was not tested. These were apparently 'normal' children of mothers who had been relatively anxious at various points in their pregnancy. Results showed that anxiety was not correlated with total grey matter volume of the brain, but some differences were found in some areas for women who had reported being anxious in the first trimester of pregnancy. It is markworthy that we are told the regions showing 'deficits', but not the compensating areas of increased volume which were presumably present to conserve overall volume. The authors go on to speculate that that reduced

volume in areas of the prefrontal cortex might lead to delayed cognitive and motor development, and that 'higher concentration of stress hormones' might cause further delays. We should note, however, that these children were apparently normal, so it would appear that there was no real evidence to support these rather negative speculations. Moreover, cognitive performance, it will be recalled, was not actually measured. The authors conclude that women's pregnancy related concerns should be 'a major focus for public health initiatives' (p149). This seems benign enough, but an irony should be noted; studies like these are highly unlikely to alleviate maternal anxiety. Further, one wonders what an initiative apparently focused on the elimination of worrying would actually look like.

Enthusiasm for screening and prevention is no guarantee of its benignancy. And where does this work lead? In a review of the literature on stress in utero, Reynolds et al 2013 note:

> *Lower socioeconomic position is associated with increased risk of morbidity and premature mortality from physical and mental disorders, and confers similar trans-generational consequences on the offspring. The effects on the offspring appear initiated prenatally as lower socioeconomic position also increases risk of prematurity and influences birth size. The programming insult resulting from low socio-economic status is not known and is likely to be multifactorial and operate through exposures including stress, poverty, housing, poor diet and lower education levels (p 1845).*

The authors conclude with the following "novel pathways for early intervention either in the preconception, pregnancy or early postpartum period:"

> *Initial findings suggest that a simple stress management instruction can improve maternal mood and reduce morning cortisol levels during pregnancy... The observation that placental gene expression is altered in relation to maternal stress suggests that we may be able to identify those most at risk for early intervention. This may also be a target for intervention in pregnancy as a very recent study has shown that a specific dietary supplementation with the methyl donor cholinein the third trimester alters the methylation profiles, and hence expression of genes in foetal derived tissues and in genes that regulate foetal glucocorticoid metabolism (p 1847)*

These are interesting policy and practice suggestions. The proposed remedies for maternal stress probably will not cause any harm, but whether they are operating at the best level conceptually or practically is highly debatable. Addressing the source of the maternal stress is of no concern, i.e. alleviating adverse social conditions or poverty, nor is it suggested that it might be useful to talk with women about happiness, relationships, aspirations, fears or hopes. Instead, the stressed women can be taught to manage their stress and have their methyl levels corrected with choline, even if they don't have a home, or enough to eat.

The grip of the foetal programming hypothesis is tenacious. A well-publicised study on the origins of endemic health inequalities afflicting the inhabitants of Glasgow concludes:

> *A link with in utero programming is an attractive explanation, as there is some evidence supporting the effect of a poor childhood environment and an increased risk of cardiovascular disorders. Indeed, in utero epigenetic programming has been linked to the development of obesity, arteriosclerosis and diabetes and may be related to material diet [.p 158, emphasis added]*

Why such an explanation is described as 'attractive' is a good question: attractive to whom? The study itself did not directly examine in utero epigenetic programming, or maternal diet. It examined the influence of a spectrum of socioeconomic and lifestyle variables on a sample of adult individuals.

Notably, the effect of the deprivation level of the participant's residential neighbourhood was assessed by comparing the poorest 5% of neighbourhoods with the most affluent. This yielded a sample of 239 individuals. The methylation status was assessed of white blood cells looking for inflammatory markers. Results showed that reduced methylation was correlated with deprivation and "social class" (defined as manual vs. non manual work); age showed a non-linear trend, falling in middle age and then rising again, suggesting, it would seem, that methylation patterns are far from fixed for life. No other variables (apart from a slight trend for educational level) affected methylation status, notably including gender, income, diet, smoking, physical activity, obesity or alcohol consumption. Further analysis suggests that the biggest influence on methylation content is manual work, reducing it by 27%. The clear result would appear to be that manual work, regardless of whether you live in a rich or poor area, or have a well or a poorly paid job, reduces your methylation levels. That maternal diet and foetal programming formed the actual headline speaks for itself.

In a thorough sociological analysis of epigenetics and its implications, Landecker and Panofsky conclude:

> *With its pronounced focus on exposures during critical periods of early development, it is entangled with the culturally tender and often fraught areas of how humans care for, feed, and pollute one another and their young. The citation peaks of the scientific literature in the area of epigenetic gene regulation look like the scientific topography of modern parenting angst…. these narratives of maternal responsibility have profound cultural ramifications (Landecker and Panofsky, A. 2013: 532)*

The Spectre of Epi-eugenics

Epigenetics breaks us out of the straitjacket of genetic determinism, we are molecularly free. But the plasticity has an ugly side:

> *This ugly side of epigenetics arises out of the heart of what makes epigenetics promising: that it focuses on plasticity, rather than determinism… makes it open to intervention and improvement, even 'optimization' … [but] The notion of optimization renders epigenetic changes as disorderly, as damage not adaptation(Mansfield and Guthman, 2014 p 3 and p11)*

Gestation becomes the playground for epigenetic manipulations. Women are held to be responsible for optimizing good biological influences, making the right choices and consuming the various remedies and therapies on offer to 'optimise' their uterine environments: practise stress management in the mornings, consume the choline supplements in the evening, and all will be well. We thus come perilously close to losing any concept of a normal continuum: all difference is potentially suboptimal and perfectibility comes within grasp. This is a paradox because epigenetics is all about difference, and inevitable variation in response to the outside world. But by equating difference with (incipient) disease it creates a particularly insidious form of eugenic thinking.

In the USA, this is racialized (Mansfield and Guthman, 2014). This side of the Atlantic, such thinking is almost certainly going to be refracted through social class. 'Optimisation' of early life environments, and the conflation of 'suboptimal' with 'marginal maltreatment', might make the case for benignly intended public health measures and parental education approaches (Barlow and Calam 2011). But they also expose particular sections of the population to increased scrutiny in the name of prevention. Arguments have also come forward in favour of broad ranging public health or environmental interventions.

> *[E]ffective mitigation of environmental health risks is unlikely to be achieved by sex, or life-stage-specific behavior change, but will require action that recognizes the much greater breadth of these risks across the life course (Cunliffe 2015:67).*

Although there are epidemiological studies linking fathers' lifestyle (smoking, diet) to disease risk in the male line (Pembrey, et al., 2006), nevertheless, the mood music in the policy world suggests it is mothers who will bear the brunt of the current epigenetic line of reasoning. Glover et al (2010) note there is 'starting to be some evidence that the nature of the stress response can be modified by sensitive early mothering' p21). Given that the birth mother is the source of the suboptimal stress response, where might this sensitive mothering be found?

Strong hints of the likely policy trajectory flowing from epigenetics comes from a recent review, co-authored by Meaney (Zhang et al., 2013). Drawing together animal and human studies, it reaffirms that "epigenetic mechanisms serve to mediate the association between early childhood and gene expression", thus explaining "in part at least, individual differences in vulnerability/resistance for specific forms of psychopathology" (p. 119). The opening comments reveal the moral direction of travel:

> *Parental factors also serve to mediate the effects of adversity derived from extra-familial sources on neurodevelopment … the effects of poverty on emotional and cognitive development are mediated by parental factors to the extent that if such factors are controlled, there is no discernible effect of poverty on child development ... Treatment outcomes associated with early intervention programs are routinely correlated with changes in parental behaviour. (pp.111-112)*

From high licking and grooming, supermum rats to sensitive human mothering is but a small step for some, although it may seem a giant leap to more sceptical minds. But biology seems to be the way to go. This has led to a search for reliable markers in peripheral tissues as proxies for changes in the central nervous system, which is rather inaccessible in humans unlike animal models. A current favourite is cheek (buccal) cells. The relatively non-invasive nature of such tests opens up the population to epigenetic screening. A recent protocol for the evaluation of an early intervention project by the 'Warwick Consortium' (2014) includes: "a number of biometric measures (e.g. hair samples to assess cortisol levels at 2 years; buccal cheek swabs to assess epigenetic changes at 3 years; accelerometers to assess activity at 7 year" (page 4). The utility of such proxy measures is at yet unclear, with a recent review noting

> *…epigenetic adaptations may be reflected, at least in part, in similar changes in peripheral tissues. Whether such changes may share some relationships, and through which molecular mechanisms, remains fully unknown (Lutz and Turecki, 2014: 151-152)*

The uncritical enthusiasm for the use of biomarkers, however questionable their accuracy, to make normative judgements about social adversity and perfectible development is as disturbing as it is revealing about the way we could be moving in the times ahead.

Conclusion

Meloni notes that freedom from the determinacy of our genetic inheritance might help make the case for more resources to 'fix' or prevent damage to the epigenome of disadvantaged groups. But he argues it may also have less desirable sequelae:

> *This all sounds desirable, but how likely is it in a society where class, race, and gender inequalities remain so vast? What is our society going to make of the notion that… the socially disadvantaged are also (epi)genetically damaged? … And what will oppressed groups do with this flurry of epigenetic studies concerning their own condition? (Meloni 2016: 221)*

The efflorescence of technobiology brings with it complex moral issues and internal contradictions. On the one hand, it can be seen as a massive forward shift in the project of human progress, but

even within medicine, where epigenetically based drug treatments have real promise and are beginning to appear, the science is as yet unsettled. In fixing one thing, we may easily finish up breaking another. These developments may be resisted by those very disadvantaged groups who have come under the epigenetic gaze; common sense, in its true meaning, may yet prevail. The following comment from a Glaswegian citizen, following publication in the press of the results of the above mentioned study is an illustration:

> *I am just flabbergasted by this latest research – I am 81 years old and was born into what I would describe as extreme poverty … but with caring parents who were not into accepting "charity" but gave me and my siblings the best they could in spite of a lot of unemployment. I have led a useful life, was pretty intelligent at school, and held responsible jobs, have married successfully, had children … and feel I was anything but deprived or damaged. Just grateful that these statistics weren't available in my past! (Citied in Meloni, 2016: 221)*

What implications are likely to ensue for social work, we may ask, of a moral imperative that requires each generation at least to maintain (if not improve) the quality of the human genome and epigenome, and pass it *on in no worse condition*? How does it change a social worker's understanding of parenting if they see a sensitive child as an epigenetically compromised individual, damaged *in utero* by a 'neurotic', low nurturance mother, herself biologically broken as a result of the carousing of a feckless grandfather? And why, we may ask, are such pessimistic hypotheses apparently so appealing?

In 2015, the neurobiologist Adam Perkins wrote *The Welfare Trait: How State Benefits Affect Personality*. The book's central argument is encapsulated below:

> *'childhood disadvantage has been shown in randomised controlled experiments… to promote the formation of an aggressive, antisocial and rule breaking personality profile that impairs occupational and social adjustment during adulthood…A welfare state that increases the number of children born into disadvantaged households therefore risks increasing the number of citizens who develop an aggressive, antisocial and rule-breaking personality profile….(p2-3)*

Perkins' road is paved with good intentions: preserving the welfare state for the worthy by ridding it of the burden of the work-shy. His reasoning is biological, reductively attributing the perverse incentives of welfare regimes to a biologically and neurologically programmed personality type: the 'employment resistant personality'. The claims, evoking animal models, are bold:

> *Selective breeding for personality causes significant genetically influenced changes in personality within as few as five generations. (Perkins, 2016: 111).*

A moral project thus manifests itself. The consequences of the ascendant moral and scientific settlements favour policy responses that are individualised and increasingly medicalized. It would currently seem that epigenetic forms of reasoning suggest greater opportunities for big pharma and possibly fewer for social housing projects and food cooperatives, which have little currency at the molecular level and often struggle to demonstrate the sort of 'outcomes' economically-minded politicians prefer. It is easier in the short term to show the effects of a pill on a biomarker than of access to decent food and human company on community wellbeing. That talk of poverty has become so unfashionable in contemporary political discourse, underscores the moral conditions for the policy translation of epigenetic reasoning. Poor people are not poor because of inequality, disadvantage or plain bad luck. They are biologically altered for the worse, and may be fixed with a pill. Epigenetics is surely prematurely borne; handle it with care, and for the time being, wear gloves.

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