

## Towards a New Sociology of Genetics and Human Identity

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Tim Owen\*

### Abstract

The intention here is to contribute towards metatheoretical development as part of the post-Postmodern 'return to' sociological theory and method associated with Sibeon [2004, 2007], Layder [2004, 2007], Mouzelis [1991, 1993, 1995, 2007], Archer [1995, 1998] and Owen [2006a, 2006b, 2007a, 2007b, 2009, 2012], in tandem with an attempt to build bridges between the social and biological sciences in the form of an ontologically-flexible, *Genetic-Social* framework with which to study issues pertaining to genetics and identity. This involves marrying aspects of anti-reductionist sociological theory with selected insights from evolutionary psychology and behavioural genetics in a similar fashion to Owen's [2012] recent attempt to do so in relation to crime and criminal behaviour. Selected meta-constructs from the framework are applied to selected examples from the literature on genetics and identity in order to demonstrate the explanatory potential. The term, *Genetic-Social* is favoured here in order to distance the conceptual toolkit's approach from that of hardline Sociobiology.

### Introduction

The framework outlined here arises out of a critique of the following illegitimate forms of theoretical reasoning, and is intended as a way that sociological theory might move beyond what appear to be major obstacles towards a new sociology of genetics and identity. These obstacles are, the nihilistic relativism of the Postmodern/Post-Structuralist cultural turn; the oversocialised gaze and harshly environmentalist conceptions of the person; genetic fatalism or the equation of genetic predisposition with inevitability [Owen, 2009, 2012] and biophobia [Freese et al, 2003] that appear to dominate mainstream social science. The starting point is to modify Sibeon's original anti-reductionist framework to include a new focus upon the biological variable [the evidence for a partial genetic basis for human behaviour in relation to sexuality, language, reactions to stress etcetera], genetic fatalism, the oversocialised gaze and asocial psychobiography.

1. *Reductionism*. This term is included in Sibeon's (1999, 2004) original anti-reductionist framework. Reductionist theories are ones which attempt to reduce 'the complexities of social life to a single, unifying principle of explanation or analytical prime mover (Hindess, 1986a, 1988) such as 'the interests of capitalism', 'patriarchy', 'rational choice', 'the risk society', 'trust', 'the information society', 'globalisation', or whatever' (Sibeon, 2004: 2).
2. *Essentialism*. This term is included in Sibeon's original framework. *Essentialism* is, 'a form of theorising that in aprioristic fashion presupposes a unity or homogeneity of social phenomena' (Sibeon, *ibid*: 4). This can include social institutions, or taxonomic collectivities such as 'white men'.

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\* University of Central Lancashire, UK, tim.owen1@o2.co.uk

3. *Reification*. This term too is included in Sibeon's original framework. *Reification* is the 'illicit attribution of agency to entities that are not actors or agents' (ibid). In Sibeon's (ibid) view, and echoed in the 'new', modified framework, an *actor* is an entity that, 'in principle has the means of formulating, taking and acting upon decisions'. Sibeon's original, non-reified definition draws upon Harre's (1981) concept of agency and Hindess's (1988: 45) 'minimal concept of actor', which specify that for an entity to be regarded as an actor, it must be capable of formulating and acting upon decisions. As Sibeon (ibid) suggests, on the basis of such a non-reified definition, there are two types of actors, namely individual human actors and Hindess's concept of 'social actors' (1986: 115). 'Social actors' include organisations such as government departments like the Home Office, organised pressure groups, and committees such as the Cabinet and micro groups such as individual households (Sibeon, ibid: 5). These 'social actors' have been termed 'supra-individuals' by Harre (1981: 141).

4. *Functional Teleology*. Again, this term forms part of Sibeon's original framework. It may be defined as, 'an invalid form of analysis involving attempts to explain the causes of social phenomena in terms of their effects, where 'effects' refers to outcomes or consequences viewed as performances of 'functions' (Sibeon, ibid: 6). Sibeon's definition draws upon the work of Betts (1986: 51), and the argument is that, if there is no evidence of intentional planning by actors 'somewhere, sometime' (Sibeon, ibid), then it is a teleological fallacy to engage in explication of the causes of phenomena *in terms of their effects*.

5. *The Oversocialised Gaze*. This term forms part of the 'new' modified framework alongside the previous four illicit forms of theoretical reasoning. The 'new' term refers to harshly 'environmentalist' accounts which are characterised by a strong antipathy towards genetic, or partially genetic explanations. The term has been applied to the work of Gagnon and Simon (1973) by Owen (2006a, 2006b, 2007a, 2009, 2012), in order to criticise the symbolic interactionists' theory that there is no 'natural', sexual drive in human biological make-up'. Contrary evidence can be found in the work of Hamer and Copeland (1999: 163), who have clearly shown how genes influence our sexual desire, how often we have sexual congress and 'help make us receptive to the social interactions and signs of mutual attraction that we feel instinctively and now call love'.

6. *Genetic Fatalism*. This is another 'new' term, now incorporated within the framework employed here. As Owen (2006a, 2006b, 2009) shows, the term refers to a widespread tendency within social science to *equate genetic predisposition with inevitability*. It is a mistake to view the genes involved in human behaviour as immutable. As Ridley (1999: 153) cogently suggests, 'genes need to be switched on, and external events – or free-willed behaviour – can switch on genes'.

In addition to these 'cardinal sins', the framework focuses upon the *metatheoretical* formulations of *agency-structure*, *time-space* and *micro-macro*, in addition to notions of Foucauldian *power*, the 'new' term, *the biological variable*, and favours a *dualism* rather than a *duality of structure*. A *metatheory* is, as Sibeon (2004: 13) suggests, intended to inform and 'hopefully improve the construction of substantive theories and the design of empirical studies'.

7. *Agency-structure*. These are important meta-concepts which refer to significant aspects of social reality. In Sibeon's original framework, and as applied here in the 'new' modified framework, the conception of *agency* is a non-reified one, in which actors or agents are defined as entities that are, in principle, capable of formulating and acting upon decisions. *Structure* refers to the 'social conditions' (Hindess, 1986: 120-1), or the circumstances in which actors operate, including 'the resources that actors may draw upon' (Sibeon, ibid: 54). *Structure* then, may refer to discourses, institutions, social practices and individual/social actors.

8. *Micro-macro*. Another meta-concept which ‘refers to differences in the units of and scale of analyses concerned with the investigation of varying extensions of time-space’ (Sibeon, *ibid*). *Micro* and *macro* should be viewed as distinct and autonomous levels of social process. Apparently, Sibeon was influenced by Layder’s (1994, 1997) argument to the ends that events in social life, at one level, do not determine events at another level, although ‘there may be contingently produced and contingently sustained empirical connections between levels.’ (Sibeon, *ibid*: 55).

9. *Time-space*. This meta-concept refers to, ‘significant but neglected dimensions of the social’ (Sibeon, *ibid*: 166). As Sibeon has made clear, the term reflects concerns with temporality and spatiality. Classical social theorists (Durkheim, for example) have tended to regard time, as ‘social time’, distinct from ‘a natural essence’ (Sibeon, *ibid*). However, ‘the question of how differing time-frames – including those associated with the macro-social order and those with the micro-social – interweave is a complex matter’ that relates to debates pertaining to *dualism* versus *duality* (Sibeon, *ibid*).

10. *Power*. Like Layder (2004), Sibeon acknowledges the *multiple* nature of power. For Sibeon (*ibid*: 145), his (meta) theoretical precepts lead to a view that, ‘power exists in more than one form; in particular, there are objective structural (including systemic) forms of power, and agentic power’, a term used to refer to ‘the partly systemic and partly relational and potentially variable capacity of agents to shape events in a preferred direction’. This modified notion of Foucauldian power [Sibeon, 2004; Owen, 2009, 2012] recognises the ‘dialectical relationship between agentic and systemic forms of power, and the concept that, *contra* Foucault, aspects of power can be ‘stored’ in positions/roles and as social systems/networks’ [Owen, 2012: 93].

11. *Dualism*. Sibeon’s original framework favours *dualism* rather than notions of duality of structure. In doing so, he is also in the company of Layder (1994, 1997, 2004). As Sibeon has suggested (1997a: 72), like Giddensian *structuration* theory, Foucault’s work has a tendency to ‘compact agency and structure together instead of treating them as a dualism’. Owen (2006a:186) has employed *dualism* in favour of *duality*, and makes the point that, ‘This Foucauldian tendency (1972, 1980a, 1982) to compact *agency* and *structure* together, to collapse distinctions between the two, results in what Archer (1995) calls ‘central conflation’.

12. *The Biological Variable*. This ‘new’ term reflects Owen’s (2006a, 2006b, 2007a, 2007b, 2009,2012) respectful criticism of Sibeon’s original *anti-reductionism* along the lines that it neglects biological or partly biological causality in explanations of social ‘reality’. We should regard the *biological variable* as the evidence from evolutionary psychology and behavioural genetics for a, at least in part, *biological* basis for some human behaviour. We should keep Ridley’s (1999, 2003) notion of ‘*nature via nurture*’ firmly in mind when focusing upon *biological variables* in analysis. This refers to the ‘feedback loop’ which embraces both genes and environment, acknowledging plasticity and mutuality. Genes predetermine the broad structure of the brain of *Homo Sapiens*, but they also absorb formative experiences and react to social cues. In short, nurture depends upon genes, and genes require nurture. In the work of Owen [2009] the evidence for a biological basis for certain human behaviour is examined, and in particular, the evidence that language is innate and specific to our species was considered with reference to the work of Hamer and Copeland (1999), Ridley (2003), Dunbar (1996) and others and the work of Hamer and Copeland (*ibid*), and Bogaert and Fisher (1995) in which evidence that sexual drive is genetically-determined as opposed to being a cultural and historical construction (Gagnon and Simon, 1973; Foucault, 1980b) was also examined. Combined with additional evidence from the work of those such as Harris (1998) which challenges *nurture determinism* with hard evidence from studies in the field of

behavioural genetics upon personality traits, the decision was made to include *the biological variable* – evidence for genetic or partly genetic causality as a concept in the ‘new’ modified metatheoretical framework.

13. *Psychobiography*. This refers to Layder’s [1997] concept of the, ‘largely unique, asocial components of an actor’s disposition, behaviour and self-identity’ [Owen, 2012: 90]. Here a modified notion of psychobiography is adopted, which embraces the mutuality and plasticity of the relations between genetic and environmental influences.

### **Demonstrating the Framework’s Explanatory Potential**

Now, the task is to demonstrate the framework’s explanatory potential by identifying *selected* examples of the illegitimate forms of reasoning listed previously within the literature and approaches to studying issues pertaining to genetics and identity within contemporary social science and to *selected* examples of related bio-social issues such as the ‘bridge building’ between social and biological sciences. This will also involve an application of some of the metatheoretical concepts to the literature and approaches. The argument put forward in this paper, supported by the work of Owen (2006a, 2006b, 2007a, 2007b, 2009, 2012) and Powell and Owen (2005) is that another ‘opening’ may lie in a ‘bridge building’ exercise in which we acknowledge the *mutuality* and *plasticity* of genes and environment. This, of course, involves acknowledging *the biological variable*, or the evidence from evolutionary psychology/behavioural genetics for biological causality. Yet, predisposition, as Ridley (1999, 2003) cogently suggests, need not be viewed through the lens of *genetic fatalism* – genes respond to social cues and are not immutable.

### **Reification**

*Reification*, the ‘cardinal sin’ of illegitimately attributing *agency* to entities which are not actors can be found within the literature of social science pertaining to the study of identity and genetics. The work of Owen (2006b:911) has suggested that the Human Genome Project is an entity that is *not* an actor, and that to argue otherwise would be to engage in *reification*.

Owen (ibid) suggests, we need to keep firmly in mind Hindess’s (1986: 115) formulation of social actors (a crucial component of the framework’s methodology inherited from Sibeon’s original *anti-reductionism*), in which Hindess defines an *actor* as a ‘locus of decision and action where the action is in some sense a consequence of the actor’s decisions’. There are those, however, such as Wilkie (1994: 187) who, in a discussion of the Genome Project and its implications and the impact of new genetic knowledge upon individuals/social institutions engages in *reification* when he claims that institutions ‘have a history and their own inertia and ‘will to live’’. Additionally, the author *reifies* ‘society’ too, when he claims that, ‘society needs to demarcate those things which will be permitted and to list those which, for the moment, ought to be forbidden’ (Wilkie, ibid: 188).

Cazillis (2001) has identified the problem of embryo *reification* in the use of stem cells. The author claims that we are facing ethical problems related to the moral status of the embryo, genetic engineering and ownership rights on living organisms raised by modern human biotechnology and its use. As stem cells are, ‘the most recent consequence of the over-accelerating biological process, it is not surprising that they are triggering most of the heated debates in the life sciences section’ (Cazillis, ibid: 1). Such debates have to steer a path through two main dangers, according to the author in a EUROPA, European Commission Research Centre article. On one hand, there is the possibility of taking hasty decisions (under pressure from international competition), and on the other hand, there lies the possibility of missing the opportunity ‘for therapeutic progress able to relieve suffering and save lives’ (ibid). The author argues that embryonic stem cells pose acute problems. They can only be obtained from embryos aged approximately one week, at the *blastula* stage.

The embryo at the *blastula* stage is approximately nine days old. At the centre of this cell mass there lies a cavity filled with liquid, and the few cells it contains – embryonic stem cells – are the ‘cause of all the excitement among biologists’ (ibid). Research on embryos is extremely controversial irrespective of the stage of the embryo’s development. As Cazillis (ibid) shows, a report by British stem cell experts, in this case from the UK Department of Health in 1990, stressed that a significant body of opinion believes that it is morally and ethically unacceptable to use an embryo for any form of research whatsoever. This is for the reason that some feel the embryo should be recognised as having a full human status immediately it is conceived. At the other end of the spectrum, the Department of Health researchers found that others claim that the embryo neither requires nor merits any kind of ‘special status’. Others, the report shows, accept the ‘special status’ of the embryo as a ‘potential’ human being, but maintain that the respect due to the embryo increases as it develops and grows, and that this ‘respect’ has to be weighed against any potential benefits of research.

As Cazillis (ibid) points out, some countries do finance research upon human embryos, others refuse to engage in it, and some ban the practice outright. In the United Kingdom, where public financing accepts research along existing lines, but not the creation of new ones, the private sector is subject to the rules laid down by each Member State (ibid). As the author suggests, in cases where such research is permitted, there is the question of where the embryos actually come from. ‘Researchers who have developed existing stem cell lines (the exact number is not known, but it undoubtedly runs into dozens) have used surplus embryos’ (ibid). These were created then conserved by the process of freezing with the intention to use them in *in vitro* fertilisation, but then they ceased to be part of a parental project either because the couple had parted company or the *IVF* was successful. They were thus destined to be destroyed. As Cazillis (ibid: 2) suggests, ‘It is not known how many embryos of this kind are available, and if a major research activity were to develop, there are some who inevitably envisage creating *in vitro* embryos specially for scientific purposes’ (ibid). As Cazillis makes clear, this would pose great ethical implications and would be seen as ‘one more step in the direction of embryo reification’. Cazillis does not appear to view embryos (which some view as ‘potential’ human beings) as social actors, hence the use of the phrase *reification*. Strictly speaking, if an embryo is unable to formulate and act upon a decision, under the definition employed here it is clearly *reification* to regard the entity as an actor.

Another controversial area of human biotechnology is the transfer of somatic nuclei, ‘sometimes known as ‘therapeutic cloning’’ (Cazillis, ibid). This involves transferring the nucleus of a ‘normal’ cell to a previously enucleated egg, with the aim of ‘creating’ an embryo carrying a patient’s genes in order to extract stem cells which are compatible with his/her immune system. In this case too, Cazillis (ibid) points out that embryo *reification* may apply. What is more, the door may be opened to, ‘reproductive cloning or, in other words, the universally condemned practice of creating an embryo carrying the genes of a single individual and then bringing it to its full development’ (Cazillis, ibid). Present research is taking place within a very varied, changing legal framework. As the author suggests, questions are being raised about the ‘ownership’ of cell lines and the tissues they are able to ‘create’ and eventually transplant (the cell banks will soon be a social reality) and ‘on the patentability of the products and techniques which originate during such research’ (ibid: 3).

Newman and Brody (1988] refer to an area of concern pertaining to the moral/ethical domain which is part of ‘the cultural context of doctor-patient relationships’. It refers to, ‘what both doctors and patients regard as good, proper, right, correct or desirable; it applies equally to the converse – what they regard as bad, improper, wrong, incorrect or undesirable’ (ibid). Such perceptions are arguably determined by the overall cultural, including religious and legal, context of medical practice (ibid). As the authors go on to suggest, ‘in most of the Western world they favour, at least as ideals, the principles of autonomy (or self-determination)’, and ‘justice (as in equality of access to resources and competing claims to scarce resources), and beneficence in medical care (the value that the patient, the person in

need, not the family, the doctor or the state) comes first' (ibid). Newman and Brody (ibid: 2) are of the opinion that, 'taken together they express the Kantian view of respect for persons', in this case, the patient. However, such ideals are inevitably compromised. Thus, certain religious groups view the principle of patient autonomy with considerable alarm. As Newman and Brody suggest, 'in the past this was expressed mainly by limiting access to contraceptive technology on grounds that coitus was intended only to produce children, and that such technology would foster promiscuity' (ibid). Today, the pro-life movement express similar alarms, whilst working towards the prohibition of abortion and towards granting foetuses the status of persons, thus interfering with the clinical judgement of physicians, the experience of patients and research to improve both, in the view of Newman and Brody. Here, we can apply the metaconcept of *reification* again, as the reference to the 'pro life' desire to award special status to foetuses may be interpreted as embryo *reification* as was identified in the work of Cazillis (2001). It seems difficult to regard a foetus as an *actor* in the sense employed in the framework, that is to say, an entity that is, in principle, capable of formulating and acting upon decisions.

In what follows, we apply the meta-concept of *essentialism* to the study of genetics and identity and related literature which attempts to redefine the relationship between 'the social' and 'nature'. Our task here, to recap, is to demonstrate the conceptual usefulness of the 'new' modified framework.

### Essentialism

*Essentialism* as employed in Sibeon's (2004: 4) system refers to, 'a form of theorising that in aprioristic fashion presupposes a unity or homogeneity of social phenomena, such as the law or some other social institution' or taxonomic collectivities such as 'women' or 'white people' etc. The term *essentialism* is used here to refer to the illegitimate attribution of homogeneity to social phenomena on a priori grounds. This is *distinct* from 'the uses of this term which are to do with the question of whether phenomena - social categories such as 'women' for example - have real essences or are socially - constructed' (ibid].

Sibeon (1996: 34) makes the point that although *essentialism* is clearly related to *reductionism* it has a tendency to be more specifically linked, 'to the reductionist notion that taxonomic collectivities - such as "women" - are a relatively homogeneous category comprised of individuals (individual women, in this instance) who have more or less common (and "objective") interests' that are 'given' to them because of their structural position in the taxonomic collectivity'. The author suggests that *essentialism* can be found in abundance in contemporary feminisms, for example (ibid). As he goes on to show, *essentialist* feminist theories of which Elliott and Mandell's (1995) work is cited as an example, mistakenly suppose that, "'women" as represented in the theory is a social category that is empirically "real"', in other words a category given by biology/society rather than the 'product' of social construction or a feminist theoretical construct (ibid). With these points in mind, it is therefore possible to identify the 'cardinal sins' of *essentialism* within certain feminist critiques of human biotechnology. Hanmer (1993) refers to how the Western 'birth control movement' was influenced by Galtonian eugenic ideas and their political expression, in addition to how the movement may be perceived as a grass-roots movement to enable women to space their children in order to protect their health. In doing so, she arguably engages in *essentialism* and *reification* when she refers to 'this double edge of control by women and of women by the state remains an unresolved issue today' (ibid: 231). Hanmer refers to women as if they are a homogeneous category whilst attributing *agency* to 'the state', which is an entity most definitely *not* an actor according to the definition employed by Sibeon, and in the 'new' framework outlined here. She also draws attention to a critique of human biotechnology common to feminisms, in which the notion of choice for women is challenged by 'arguing that the pressures on women to have children, but only when it is socially acceptable to do so' (ibid: 232) are so great that it is pointless to discuss issues of 'choice'. Again, the emphasis is upon women as an *essentialist* monolithic block. 'Women are seen as

having no option about whether or not to have babies', and 'motherhood is socially compulsory, even in countries with coercive population control policies' are phrases (ibid) which further indicate Hanmer's *essentialist* tendencies. Hanmer identifies another 'line of attack' which criticises human reproductive technologies from feminist standpoints, for being, 'about something very different from what is claimed', in other words, 'not about helping women to have children, but rather being an aspect of genetic engineering closely associated with new developments in biotechnology' (Kollek, 1990, Shiva, 1988). This includes the attempt to map the entire human gene pool, beginning with the Human Genome Project, to 'improve' human embryos through genetic testing and the addition/subtraction of genetic material (Kaufmann, 1998; and Leuzinger and Rambert 1998). Again, the emphasis in Hanmer's work is upon the implications for 'women' as a category which is, in an *essentialist* way, empirically "real" and homogeneous. Hanmer goes on to refer to feminist literature such as that of Bridenthal, Grossman and Kaplan (1984); Degener (1990); Ewing (1988); Kaupen-Haus (1988); Schleiermacher (1990); and Zimmerman (1990), which suggests that 'modifying and eliminating people before birth gives a new twist to eugenics' (ibid: 234). However, her claim that 'it can also be seen to be about men as a class taking control of women's reproductive activities' is surely a case of *essentialism*. Here, Hanmer appears to refer to both 'women' and 'men' in *essentialist* terms as homogenous social phenomena.

In what follows, we apply the meta-concept of *Genetic Fatalism* to the study of genetics and identity, in order to demonstrate the explanatory potential of this 'new' term. Sibeon's original anti-reductionist framework does not include this concept, which as Owen (2006a, 2006b, 2007a, 2007b, 2009, 2012), Owen and Powell [2006] and Powell and Owen (2005) define as the tendency within social science to associate genetic predisposition/determinism with *inevitability*.

### **Genetic Fatalism**

We have examined evidence from the work of Ridley (1999, 2003) against this 'cardinal sin' of illegitimate theoretical reasoning, which suggests a great degree of *plasticity* and *mutuality* between genes and environment. As Ridley notes, a favourite slogan of those involved in the counter-attack against the ideas of E.O. Wilson was, 'Not in our genes!' Perhaps at the time it was 'a plausible hypothesis to assert that genetic influences on behaviour were slight or non-existent' (Ridley, ibid). However, the work of Rose, Kamin and Lewontin (1984) may be 'no longer tenable' in the light of studies in behavioural genetics which offer cogent evidence that, 'genes do influence behaviour' (Ridley, ibid). It will be recalled that the work of Owen [2009] examines the convincing evidence from authors in the fields of Evolutionary Psychology and Behavioural Sciences, such as Hamer and Copeland (1999), Enard et al (2002), Lai (2001), Dunbar (1996), Pinker (1994), Harris (1998), Bogaert and Fisher (1995), Curry (2003) and Cosmides and Tooby (1997) amongst others for an, at least in part, genetic basis for human behaviour.

It is to the concept of the *oversocialised gaze* that we must now turn our attention. Again, we examine the explanatory potential of the meta-concept in relation to the study of human identity and genetics.

### **The Oversocialised Gaze**

*The Oversocialised Gaze* refers to accounts which are harshly environmentalist and antipathetic towards genetic or partially genetic explanations of human behaviour. The accounts may even reject genetic variables altogether, as Giddens (1993: 57) appears to do when suggesting that, 'human beings have no *instincts* in the sense of complex patterns of unlearned behaviour'. There is cogent evidence for *instinctive*, unlearned patterns of human behaviour in the work of authors such as Ridley (1999, 2003), Hamer and Copeland (1999), Pinker (1994) and Dunbar (1996) especially regarding the idea that, 'grammar is innate' (Ridley, 1999: 104).

There is a tendency of writers of the 'embodied school' or 'sociologists of the body' to engage in *oversocialised* approaches. Tim Newton's (2003: 29) criticisms of Sahlin's (1972) and Pollock's (1988) approaches (in the area of 'building bridges' between the social and life sciences) centre around the latter authors' respective portrayals of stress as the product of 'natural' instincts. In Newton's view, 'such arguments present a *crude dualism*' which reduces complex social 'problems' to 'outmoded' conceptions of 'the biological body' (ibid). He then goes on to argue for a 'putative non-reductionistic 'biological sociology''. It is difficult to envisage a credible 'biological sociology' in the age of the Human Genome Project which denies human instincts altogether. It is not appropriate here to rehearse all the evidence provided in the work of Owen [2009] for the *instinctive* component of human language ability, sexuality etc. However, it could be argued that Newton (ibid) appears to be unaware of the fairly recent evidence provided by Ridley (1999) for an account of stress in human beings which posits the '*nature via nurture*' approach to causality. Newton's arguably *oversocialised* concept of stress emphasises the influence of complex social factors whilst rejecting any reference to 'natural' instinctive, genetically-based behaviour. Ridley (ibid) arguably shows how it is indeed possible to include both genetic *and* environment variables in an explanation of stress in human beings.

As Ridley (ibid: 149) acknowledges, stress in human beings is 'caused' by 'the outside world', and 'short-term stressors cause an immediate increase in epinephrine and norepinephrine', which are the hormones that make the human heart beat faster, the feet go cold etc. These hormones act to prepare the human body for 'fight or flight' in emergencies. Stressors that last for longer activate a different pathway that results in a much slower, but more persistent increase in cortisol. Cortisol is 'used in virtually every system in the body' and is a 'hormone that literally integrates the body and the mind' by altering the configuration of the brain' (ibid). As the author suggests, 'one of cortisol's most surprising effects is that it suppresses the working of the immune system', and those who have shown the symptoms of stress are 'more likely to catch colds and other infections', because one of the effects produced by cortisol is the reduction of white blood cells (ibid: 149-150). Ridley goes on to show how the relationship between genes and environment is one of *mutuality* rather than an *oversocialised* conception which rejects genes and instinct in favour of a solely social explanation of the kind suggested by Newton (2003) in his treatment of the work of Sahlin (1972) and Pollack (1988). Ridley (ibid: 150) explains how the hormone cortisol reduces the 'activity, number and lifetime of lymphocytes', or white blood cells, showing how cortisol does this by switching genes on.

As Ridley suggests, 'you cannot produce, regulate and respond to cortisol without hundreds of genes', practically all of which 'work' by 'switching each other on and off'. In white blood cells, 'control is almost certainly involved' in 'switching on' the gene known as *TCF*, on chromosome ten, which enables *TCF* to make its own protein, 'whose job is to suppress the expression of another protein called interleukin 2' which in turn, is a 'chemical that puts white blood cells on alert to be especially vigilant for germs' (ibid). Thus, cortisol suppresses the 'immune alertness of white blood' and renders the person, 'more susceptible to disease' (ibid). The author goes on to pose an interesting question: 'Who's in charge? Who ordered all these switches to be set in the right way in the first place, and who decides when to start to let loose the cortisol?' (ibid). The answer does not lie with the genes solely, even though 'the differentiation of the body into different cell types, each with different genes switched on' is a genetic process 'at root' (ibid). Genes are not the 'cause' of stress, and as Ridley makes clear, 'the death of a loved one, or an impending exam, do not speak directly to the genes' (ibid). They are, in essence, 'information' processed by the human brain. Is the human brain 'in charge'? On one hand, as the author suggests, the hypothalamus of the human brain, 'sends out the signal that tells the pituitary gland to release a hormone that tells the adrenal gland to make and secrete cortisol' (ibid: 151). The hypothalamus takes 'orders' from 'the conscious part of the brain which gets its information from the outside world' (ibid). However, as Ridley suggests, this 'answer' will not suffice 'because the brain is part of the body'.

In answer to what *did* 'set up the system', Ridley suggests that 'natural selection did', and 'somewhere down the cascade of events that is the production, control and reaction to cortisol' stress-prone people 'must have subtly different genes from phlegmatic folk' (ibid). Who or what is ultimately 'in charge'? Ridley's answer is that 'nobody is in charge', rather an 'intricate, cleverly designed and interconnected system' in which nurture depends upon genes, and genes require nurture in an elegant mutuality. This is, arguably, far removed from the *oversocialised* explanation for stress posited by Newton (2003), or that which the author identifies as '*crude dualism*' in the work of Sahlin (1972) and Pollack (1988).

Owen (2006a) has identified the work of Gagnon and Simon (1973) and Foucault (1980) as examples of accounts which rely upon the *oversocialised gaze*. As Owen (2006a:190) suggests, 'Foucault's (1980b) argument that sexuality is a socio-cultural creation, that sexuality as we know it is the production of a particular set of historical circumstances and obtained only within the terms of a discourse' is *oversocialised*. Owen refers to the work of Hamer and Copeland (1999: 163), which shows how genes influence our sexual desire, how often we have sex and, 'help make us receptive to the social interactions and signs of mutual attraction that we feel instinctively'. In the case of Foucault's (1980b) work, his belief that sexuality is 'controlled' through definition and regulation (rather than by the 'impression' suggested by Rich's (1981) feminist analysis of heterosexuality), in particular via the creation of sexual categories such as heterosexuality and so on is open to question. In the case of female sexuality, Foucault (ibid) argues that it is 'controlled' not by the denial or 'silencing' favoured in Walby's (1990) feminist account, but by *constant referral*. Foucault suggests that the history of sexuality is in effect a history of shifting forms of control/regulation. In the case of the last hundred years or so, the shift has been away from the church's moral regulation towards an increased regulation through education, medicine, psychology, law, social work, social policy etc. 'Sex', for Foucault (ibid) is not a biological 'entity' but rather an *idea*, a concept which is always specific to certain cultures and cultural and historical periods. Sexuality for Foucault is produced through *discourses on sexuality*, which shape human sexual values and beliefs. This, as Owen (2006a, 2006b, 2007a, 2009, 2012) suggests, appears to be an *oversocialised* perspective which denies the notion of a biological sexual drive altogether. As Ridley (1999: 149) shows, 'a gene on chromosome 10 called CYP17' directly affects sexuality in human beings. The gene manufactures an enzyme which enables the human body to convert cholesterol into cortisol, testosterone and oestradiol, and without the enzyme, the pathway is blocked and the only hormones that can be made from cholesterol are progesterone and corticosterone. People lacking a 'working copy' of this gene 'cannot make other sex hormones so they fail to go through puberty; if genetically male, they look like girls' (ibid).

Owen (2006a) has identified Gagnon and Simon's (1973) symbolic interactionism as *oversocialised*. Gagnon and Simon suggest a radical form of social constructivism in which there is no sexual drive in the 'natural' sense that it is said all human beings possess to varying extents as part of biological make-up. They argue for a model of human sexual drive purely as social construction. Not only do we *learn* what 'sex' means, and who/what is sexually arousing to us, but we also learn to want sex. Though Gagnon and Simon acknowledge that the body has a repertoire of biological 'gratifications' (ie, the capacity for orgasm), it does not automatically follow that we would want to engage with them. Certain 'gratifications' will be selected as 'sexual' via the learning of 'sexual scripts'. For example, a particular experience would not be repeatedly sought after unless there was the presence of a 'meaningful' script. From Gagnon and Simon's *oversocialised* perspective, which denies a biological component to *sexual drive* (different from the 'gratifications') socialisation is not about learning to control innate sexual desires in order that they are expressed in socially acceptable ways, but rather the learning of 'sexual scripts' of some complexity which serve to specify circumstances which will elicit sexual desire and 'make' the person wish to engage in certain acts with his/her body. Thus, for the authors, sexual drive is, as it is for Foucault (1980b), a 'learnt social goal'. Having examined the explanatory potential of the

*oversocialised gaze* as a meta-concept of the ‘new’ framework, we must next look at the concept of the *biological variable*.

### **The Biological Variable**

The *biological variable* is referred to as a ‘new’ term. The term, or meta-concept, is ‘new’ in the sense that it is *not* employed by Sibeon. We should regard the meta-concept as referring to the evidence for an, at least, in part *biological* basis for some human behavior. Its use arises out of a respectful critique of Sibeon’s original anti-reductionism on the grounds that it neglects biological or part biological causality in explication of social ‘reality’ (Owen, 2006a, 2006b, 2007a, 2007b, 2009, 2012 ; Owen and Powell [2006]; and Powell and Owen, 2005).

It is the contention here that theoretical analysis, particularly that pertaining to the study of genetics and identity and related areas in which links are attempted between ‘the social’ and biology (Benton, 1991, 1994; Williams, 1998, 2003; Bury, 1997; Newton, 2003), it is *essential* to recognise that, ‘genes do influence behaviour’ (Ridley, 199: 306) and that the harshly ‘environmentalist’ accounts of Foucault (1980b) and Gagnon and Simon (1973) in which sexuality, for example, is completely *learned* behaviour are no longer plausible hypotheses in the age of the Human Genome Project. Wilkie’s (1994: 171-72) treatment of the ‘moral consequences of molecular biology’ contains the expression of unease at the possibility that the Project will ‘point up differences between individual humans at a genetic level’. The author suggests that if such genetic knowledge is ‘not handled properly and seen in its proper, biological context’, it may lead to the generation of information which enables ‘new grounds of discrimination’. From the point of view of ‘the Project’s very existence’, we may take an increasingly ‘atomistic’ view of human beings and indeed of life itself’ (ibid). Wilkie’s (ibid) greatest fear is that the advances in human biotechnology may lead to a tendency to ‘define ourselves in genetic terms and neglect the rest’. The author draws attention to the possibility of the risk that, ‘we may all become reductionists reducing our lives to their supposedly fundamental components’, missing the holistic ‘complexity and richness of life in its entirety’. Arguably, Wilkie’s fears are misplaced. There is no reason to suppose that such ‘*geneticization*’ (also referred to by Opitz, 2000 ] should ever come about. The ‘*biological variable*’ should be recognised as playing a part in causality, but equally there is a role for the environment (part of the ‘complexity’ and ‘richness’ referred to by Wilkie) which is ‘massively important’ (Ridley, ibid) in the sense that genes are ‘switched on’ by social cues. Acknowledging a genetic component to causality does not have to entail the *genetic fatalism* engaged in by Wilkie. The three authors of *Not in our Genes*; Steven Rose, Leon Kamin and Richard Lewontin made the point that ‘biological determinists’ believe the credo “you can’t change human nature” to be the beginning and end of the human condition. However, as Ridley (1999: 307) makes clear, ‘this equation-determinism equals fatalism – is so well understood to be a fallacy that it is hard to find the straw men that the three critics indict’.

The evidence for biological variables playing a part in human behavior, such as reactions to stress, language and sexuality has been explored in some considerable depth by Owen [2009] and the source of the insights (largely *evolutionary psychological*) has been examined with reference to the staunch defence of such ideas by Curry (2003); Dawkins [ 1986); Daly and Wilson (1998); Tooby and De Vore (1987); Cosmides and Tooby (1997), Ridley (1999, 2003) etc, and criticisms by those such as Rose and Rose (2000); David (2002); Rose (2000); Rose et al (1984).

### **Conclusion**

The *Genetic-Social* framework outlined and selectively demonstrated here is arguably best equipped to point a possible way forward towards a new sociology of genetics *and identity that entails a reliance upon multifactorial analysis, and an avoidance of reified and essentialist analysis and the anti-foundational relativism of the cultural turn*. Additionally, the

sensitizing device sidesteps the nature-nurture divide, emphasising instead, 'a balanced account of the mutuality between genes and environment' [Owen, 2012: 94]. These elements combined hopefully make the framework a useful conceptual toolkit which incorporates a realist social ontology with a rejection of bio-phobia.

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