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**C is for complexity: Why genetics doesn't outweigh teaching**  
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“There is no escape from the conclusion that nature prevails enormously over nurture” wrote Francis Galton almost 150 years ago. Reading the recent ‘Genetics outweighs teaching’ polemics (Guardian, 18th Oct 2013), inspired by a few pages in Gove’s former policy advisor Dominic Cummings’ ‘thoughts on education and political priorities’, and, more significantly, the book (G is for Genes: The Impact of Genetics on Education and Achievement) by Kathryn Asbury and behavioural geneticist, Robert Plomin, would suggest that we have not moved too far from Galton.

This may sound surprising, not least for the reason that when Galton wrote these lines, genetics was far from being discovered, not to mention DNA, and the many complexities that are known in the world of biosciences as genomics first and postgenomics today (meaning what has occurred after the deciphering of the Human Genome in 2003). Shall we then conclude that the exciting story of genetics over the last one hundred years or so has been just a trivial repetition of what was already known to Galton, intuitively so to speak, before the dawn of the gene? Or, more likely, should we conclude that the many complexities of genetics are invariably distorted into a dichotomous framework that proposes a conflict between what is supposedly inside us (the genes) and what is out there (teaching, education etc.)?

There are many ways to comment on the claim that ‘genetics outweighs teaching’. There could be technical objections on the misused notion of heritability for instance, a term still enormously misunderstood by the public (with some complicity by scientists themselves, alas) and which is especially problematic when applied to intelligence. Or, technical comments on the plausibility, and highly controversial nature, of some core assumptions and methodology of behavioural genetics - the discipline behind many of the findings inspiring Cummings on genetics and education. Or, on the nature of ‘intelligence’ as measurable by abstractions such as IQ or ‘g’. Or, more profoundly, on the never-ending ambition to use genetic findings as a solid basis for public policy recipes, an unfortunate tendency from which Asbury and Plomin alas do not shy away. They conclude their book with a chapter on “Eleven Policy Ideas” grounded in what they define as the ‘genetic basis’: “we are all different” ....therefore we need to “minimize the Core Curriculum and Test Basic Skills” and others similar non sequiturs.

However, rather than focusing on these and many other possible critiques, I think it is much healthier for social scientists and public policy professionals alike to give attention to what is the real fallacy behind the ‘genetics outweigh teaching’ argument: the fact that the kinds of genes, educational or anti-educational, evoked by Cummings or Plomin may simply not exist.

The postulated intelligence genes, or the general cognitive ability (or g) genes (or whatever other personification of the gene as a discrete entity, that regulates our behaviours and talents,

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is posited) are, far from being the results of “modern developments in genetics”, as Cummings suggests, the product of the infancy of genetics. They are the legacy of a time when scientists fetishized the gene (as in eugenics) as a sort of internal agent in the human body, a conductor of the orchestra of our personal-fate. It is unfortunately the case that this gene-hunting mentality has had a dramatic return since the 1990s, especially with the expectations raised by the Human Genome Project. However, it is precisely the completion of the HGP that has made visible that this supposed ‘gene conductor’, fixed since birth in our genome, operating and disposing of our faculties and talents, is simply not there.

A more careful reading of the history of genetics would clearly illustrate how the best researchers had always known that those discrete and ‘preformationist’ genes, as Lenny Moss (2003) calls them, never really existed, apart from as a practical convenience in designing experiments. Why people at different times tend to forget this simple historical lesson and repeat invariably over and over the same intellectual mistakes and the same frustrating ‘looking for causes in genes’ repertoire might be the subject of a whole new discipline! This is what historian of science Robert Proctor has recently called agnotology (from the Greek agnosis, not knowing, see, Proctor and Schiebinger, 2008); that is the science that explores how ignorance is produced or maintained in diverse settings, public policy included.

#### What Genes Can and Cannot Do

As historian of science Evelyn Fox Keller (2011) has noted, what has become increasingly evident in recent years, especially after the completion of the Human Genome Project, is that only a very small fraction of the genome (slightly more than 1% in fact) is “devoted to protein-coding sequences,” whereas most of it is employed in regulation. That is, employed in responding to environmental signals. This is a very broad notion that includes the environment around the DNA, but extends to embrace the entire organism and, in the case of human beings, its social and cultural dynamics. As philosophers of biology have noticed, the more genetic research has gone forward, the more genomes are seen to “respond in a flexible manner to signals from a massive regulatory architecture that is, increasingly, the real focus of research in ‘genetics’” (Griffiths and Stotz, 2013; see also Barnes and Dupré, 2008, Dupré, 2012).

In sum, if there has been anything like a development in modern genetics, it has gone in the opposite direction to Galton’s fantasy of partitioning nature and nurture into two discrete and opposable entities, of which the first, nature, leads the orchestra. Unfortunatley, this fantasy survives, in spite of all the lip service paid to interaction, in programmes like behavioural genetics, whose core assumption is exactly the possibility of partitioning and measuring the separate influence of genes and experience in a framework that would keep genes very much at the centre of developmental processes.

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Against this die-hard way of thinking, neuroscientist Michael Meaney (2001a) has written extensively, over the last decade, explaining that, “there are no genetic factors that can be studied independently of the environment, and there are no environmental factors that function independently of the genome. ... At no point in life is the operation of the genome independent of the context in which it functions.” Moreover, what we also know today, Meaney continues, is that “environmental events occurring at a later stage of development ... can alter a developmental trajectory,” thereby undermining linear regression studies of nature and nurture.

The new kind of non-linear and reversible gene effects that Meaney has in mind are best exemplified in the experiments he carried out at McGill University, where variations in maternal behaviour in rats were linked to changes in gene expression in the offspring. The intense licking activity of an ‘affective mother’ rat had a positive effect on brain development in pups, an effect mediated by epigenetic changes in the regulation of stress hormones and perpetuated for up to three generations. In contrast, reduced maternal grooming left neglected pups in a state of permanent stress. More interestingly, these mutations might be reversed by cross-fostering the pups to more ‘caring mothers’ (Weaver et al., 2004; Meaney, 2001b).

Meaney’s experiments show how transient environmental factors can leave a mark on the genome, but also how this mark can be reversed by experience. We are, therefore, according to this new postgenomic account, plastic beings in a double sense. We can receive ‘scars’ from the environment, but these scars can also be healed. It should be evident how difficult it remains to partition genes from experience and how this seriously undermines notions of a natural genetic lottery, or a natural endowment, upon which the environment only acts in trivial ways.

Meaney’s experiments, and those of others, including Waterland and Jirtle’s work on the switching of the agouti gene in mice - through a methyl-rich maternal diet in gestation that makes genetically identical offspring look phenotypically different (2003, 2004) - are the basis of the recent surge of interest in what is known these days as molecular epigenetics. This is a discipline that is significantly blurring the boundaries of what is inside (genetics?) and what is outside (teaching?) our body, making Galton’s dichotomous view increasingly untenable. In a nutshell, epigenetics studies molecular mechanisms of gene regulation by which social-environmental information is channeled into genetic information. By making visible these molecular mechanisms, epigenetics reveals that gene expression is dependent on social and environmental factors, which, through mechanisms like methylation, can switch genes on and off.

In more sophisticated research genes have always been considered contextual-dependent entities (the DNA as such is an inert molecule that needs its cellular context for many things included nutrition), but such a degree of dependence on environmental factors was unexpected. This is why epigenetics has been defined as the biological proof of ‘how the social

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gets under the skin' (Hyman, 2009) or as biological evidence of the 'neural mechanisms of nurturance' (Lonstein, 2003).

In terms of evolutionary dynamics, epigenetic mutations are best seen as a case of developmental plasticity, the way evolution solved the problem of making a "fixed genome" respond in a prompt and plastic way to the solicitations from a changing and dynamic environment (Gluckman et al., 2007). Studies on phenotypic plasticity have been increasingly influential, in evolutionary thinking, in the last decade (Pigliucci, 2001; West-Eberhard, 2003) showing how one single genotype can generate many different phenotypes and how organisms are flexible to change in different environments.

This way of thinking brings to light another inconsistency of behavioural genetics, the claim that there is a 'gene x environment' interaction. As the late S. J. Gould would have said, this can be accepted only as a very poor way of talking, because, in reality, there are not genes interacting with the environment, but merely organisms interacting (and changing) their own environment or niche. Indeed, epigenetics may also produce an important rethinking of classic twin studies from which much of the knowledge of behavioural genetics derives, and may do so by focusing on divergences rather than the similarities between identical twins (Spector, 2012).

Last, but not least, epigenetics and the environmental regulation of gene expression is by no means the only disruption to outdated views of the gene still circulating in society. For example novel findings regarding, for instance, the phenomenon of mosaicism - the fact that it is possible to find a mosaic of different genomes in different cells and tissues of the same individual, rather than the same genome everywhere (Charney, 2013; see Parry and Dupré, 2010).

I do not mention these postgenomic novelties here to claim that we are entering a golden age of biosocial investigations or to suggest that, in the age of epigenetics, social scientists should abandon critical vigilance with regard to genetics, quite the opposite. New epigenetic studies, for instance, have been applied to crucial social and political issues like race and social inequalities, and some of the findings may appear problematic to social scientists. The fact that different social categories (from race to class), and environmental factors (from maternal care, to food and toxins) are being increasingly conceptualised in molecular terms (Landecker, 2011; Niewohner, 2012) and that nurture is increasingly becoming thought as a form of exposure to genetic expression (as exemplified by the recent emergence of the notion of "exposome": Wild, 2012) is far from being trouble-free.

My point is that there is a specific profile of problems in the postgenomic age, a whole array of new issues and ethical and social demands that may however, be very poorly reflected in the image of the dominant gene emerging from Cummings or Plomin and now re-entering public debate.

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How science can and cannot be translated into the public sphere

Let me return to Cummings' document and Plomin and Ashbury's book to argue that these two interventions into public debate are of a different league and should not be confused. The first contains a few, very questionable, notions of genetics. The latter is an interesting scientific book, albeit based on some unquestioned core assumptions that remain at the basis of behavioural genetics, which is itself under scientific challenge. However, what makes the two texts continuous is their parallel attempt to jump from scientific to some general public policy conclusions.

In the light of the many complexities I have highlighted, and the impossibility of, not only a real partitioning of gene and experience but, more radically, in relation to the elusiveness of the notion of the gene itself, this is quite frankly an unscientific exercise. While understandable from Cummings who is not a scientist, it is less of an excuse for Ashbury and Plomin. It does not matter that their conclusions can be seen as more progressive than many earlier applications of genetics to social policy. It is not a matter of progress or reaction, of using genetics to help, (as Ashbury and Plomin argue with the best of intentions) or to condemn people. What matters is that behavioural geneticists or, for that matter, epigeneticists, who jump quickly to social policy conclusions, are over-selling their discipline. They seek to transform a rather chaotic, intricate and often controversial field into a secure basis for grounding social policy agendas. And, more problematically in my view, in doing so scientists forget (agnology, again) the past of these dangerous (but apparently very appealing) attempts to make a social policy out of genetics.

Luckily, a thorough immersion in what historians and philosophers of biology have to say about how genes work will easily dispel many of these recurring illusions and illustrate the wide gap between the fantasy gene still present in many social policy debates and the actual elusive 'gene' of genetics. Here is where social scientists need to stand up today, becoming more aware and less fearful of the many complexities around the gene that make its simplistic mobilization in policy findings a futile, or worse, irresponsible exercise. They need to engage with the debate and bring about a proper deflation of the agency and role of the gene (Meloni, 2013). This, in turn, would bring with it a deflation of the allure of genetics as a simplistic resolution of social policy issues. With genes seen as complex and open to the environment like those proposed in postgenomics and epigenetics, who needs to be biophobic anymore?

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