



# Lumpy Heads and Violent Genes: Moving Beyond Simple-Minded Explanations for Complex-Minded Folk

Isabella Sarto-Jackson: *The Making and Breaking of Minds: How Social Interactions Shape the Human Mind*; Vernon Press, Wilmington, 2022, 292 pp., \$63 hbk, ISBN 978-1-62273-331-6

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Whether motivated by political expediency or scientific zeal, simplistic explanations for the functioning and dysfunctioning of the human mind have abounded for centuries. The bizarre 18th- and 19th-century field of phrenology, for instance, sought to infer human mental ability and character from the bumps on an individual's skull. State-of-the-art science in its day, phrenology was a major contributor to scientific racism, with white, male scientists quick to interpret other peoples' skull protuberances as indicative of a natural inferiority. Criminal tendencies and mental disability too were viewed as products of pathological patterns of brain growth and development that bulged through the cranium. Long after phrenology had fallen into disrepute, an extreme localizationism, in which functions were tightly tied to particular brain regions, remained mainstream.

Such pseudoscience is easy to dismiss as a historical anomaly, the product of a bygone era before science came of age. Yet, as Sarto-Jackson documents in her excellent and scholarly monograph, scientists in the 19th century replaced overinterpreting skull shape with overinterpreting brain size. Scientific luminaries, including Samuel Morton and Paul Broca, fallaciously concluded that the inferiority of persons of color, criminals, or the disabled could be explained by a deficiency of brain tissue. And when that twaddle was rejected by the scientific community, in the 20th century psychologists turned to IQ, once again erroneously concluding that assumed intellectual differences between favored

and unfavored types could be solely accounted for by discrepancies in this metric.

What these explanations have in common (apart, that is, from an unsettling tendency for scientists to generate findings that confirm their prejudices) is an overly simple linear notion of biological causation. Because of “intrinsic” and “ineradicable” biological factors, the brains and behavior of classes of individual are perceived to be differentially programmed to possess varying qualities and operate in particular ways. Disturbingly, Sarto-Jackson reveals how 21st-century science and society remain suckers for simplistic explanation. As recently as 2009, a Tennessee man called Bradley Waldroup, who brutally killed and mutilated his ex-wife, had his sentence reduced from murder to manslaughter after the defense counsel argued that he possessed a mutation in a gene (MAO-A) reputed to predispose its carriers to violence. Armed with the relevant scientific facts, this explanation can be seen to be no less ridiculous than one based on skull shape. Almost all human characters, including behavioral characteristics such as aggression, are known to be affected by hundreds, often thousands, of genes (Boyle et al. 2017). Any one of these genes, even the most influential, will explain a tiny fraction of the variation in the trait of interest. Even summed together into polygenic risk scores, the genes combined rarely account for more than 5–10% of the variation in the focal character: no wonder biologists are concerned about “missing heritability” (Manolio et al. 2009). When it comes to the MAO-A gene, allelic variants connected with aggression are far from rare—around a third of the population carry them, almost all of whom exhibit no tendency to violence. And numerous other factors—ranging from early-life experiences, to epigenetic inheritance, to social learning, to situational contexts—are known to contribute to the complex etiology of aggression, just as they do for virtually every aspect of human behavior and cognition.

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Sarto-Jackson's admirable book powerfully demonstrates that the causes of brain functionality, and the explanations for dysfunction neural processing and behavior, are *always* rich, multifaceted, and convoluted, with genes *always* just one of many causal factors involved. Sarto-Jackson is particularly keen to stress how, in interaction with other causal elements, the social environment shapes cognitive development, both for good and bad. In the last few years, the idea that social interactions have an immense impact on infant brain development has gained momentum in the fields of brain research and cognitive neuroscience. Sarto-Jackson explains how social interactions early in life—particularly adverse experiences, such as abuse or trauma—impact brain development and affect adult behavior.

An important contribution of the work stems from its interdisciplinarity, which lends the book an innovative quality. Sarto-Jackson integrates findings from developmental psychology, cognitive neuroscience, medicine, cultural evolution, ecology, and evolutionary biology into a coherent and compelling account. A central hypothesis—and for me the critical insight—is that, “individuals are not just passive entities harried by external forces, but change their environment, sometimes to a very significant extent” to “themselves influence their own development” (p. 19). The point is not just that environmental factors, ranging from violent parents to impoverished conditions, influence cognitive development. More than that, both children and adults actively choose and construct “niches” suited to their current state of mind and conditioned expectations, in the process themselves contributing to the etiology of developmental disorder. Here Sarto-Jackson draws on “niche construction theory” (Odling-Smee et al. 2003), which stresses the active role of the organism in evolution. Humans have constructed “ecological, cultural, social, technological, and even virtual spaces” (p. 20) that strongly influence their development.

The initiative is a welcome one. In a field historically plagued with simplistic explanations, amassing a compendium of scientific data that reveals the true multiplicity of causal factors underlying human minds is a major virtue. Here, as much as anywhere, the famed nature-nurture dichotomy is a severe obfuscation: cues to transcribe genes commonly emanate from the environment; social and other environmental experiences shape gene expression; learning, including the social learning that supports culture, is also reliant on gene expression; by choosing and modifying development environments, human behavior feeds back to fashion cognition; and so on, and so on. Causation comprises iterated cycles of reciprocal interactions between organism and environment.

In Chap. 1, Sarto-Jackson takes a historical perspective to expose deterministic views of human nature, historically manifest as “preformation” (development is an

“unwrapping” of a tiny fully formed human) and more recently as the genetic program metaphor. Historically, these views have been used to justify the neglect of social interventions to address inequalities and eradicate poverty. Early IQ research falls into this tradition. When tested, immigrants to the US from several countries were found to have below-average IQ, and alarmed psychologists concluded that lower caliber “stock” were invading. The flawed premise was that IQ test performance measured “innate” intelligence and was little affected by culture, schooling, or practice. How wrong that was is illustrated by the fact that, two generations later, the grandchildren of those same immigrants were performing above average on IQ tests. Likewise, the Flynn effect (Flynn 1987), which refers to the steady increase in IQ scores over time in countries around the world, documents how IQ-test performance is not, as it is purported to be, a “pure” measure of “innate” intelligence, but is strongly influenced by social factors.

Such findings are representative of cognition and brain development more generally, for which genes are just one of many causal factors. Despite widespread assumptions to the contrary, there is no “blueprint” telling an organism how to wire a brain; a rich developmental dialogue between genome and environment are involved. Myopia is a case in point. Living inside, reading, and engaging in much close work, leads eyeballs to grow in ways that would be checked in the presence of natural light, because the latter stimulates the release of dopamine, a neurotransmitter that blocks eye elongation. It is real-life vision that guides the growth of the eye. Environmental feedback, arising through human action, impinges on our biological endowments. This dynamic interplay of genes and environment, mediated by an active human agent, leaves the nature-nurture distinction problematic.

The same dynamical interplay shapes brain development and behavior, with this sensitivity to feedback leaving humans vulnerable to stress and disease, a point to which Sarto-Jackson turns in Chap. 2. Here, she critiques the historically dominant view that the brain is hardwired with specific functions localized to specific brain regions. Sarto-Jackson documents the emergence of a counter-perspective emphasizing flexibility and neuroplasticity. Now mainstream, this new thinking recognizes the ability of the brain to reorganize itself and adjust within the lifespan to environmental experiences. For instance, she describes a child who was forced to have half of her brain removed after contracting a disease and not only recovered walking and moving but also went on to complete a college degree. That brain plasticity is only surprising because of the legacy of fields such as phrenology, which led scientists to anticipate extreme localization of function. Extensive research now reveals that patients with damage to regions traditionally

associated with particular abilities often recovered those functions, with that neural plasticity continuing into adulthood (although adult brains are certainly less malleable than younger ones). Such findings, combined with the observation that different patients with the same brain-damaged regions often have quite different symptoms, undermine conventional, strict localizationism.

This malleability derives from a key, but poorly appreciated, mechanism of vertebrate development: “exploratory mechanisms” (Gerhart and Kirschner 1997). The brain is not built simply through following genetic instructions, but rather through a selectional process arising in development, in which a vast abundance of neurons and neuronal connections are generated early in childhood, and are subsequently pruned according to experience. Brains develop on a “use-it-or-lose-it principle.” Genes may specify the gross structure and circuitry of the brain, but the details depend very much on experience. The resulting neuroplasticity not only underlies learning and memory formation, but also enables humans to compensate for brain lesions or defects. The vertebrate adaptive immune system and vascular system work on similar selectionist principles. While such mechanisms are taxonomically widespread, they are particularly important to our own species because we possess an unusual developmental niche. Humans are the most altricial of primates, probably because we must be born before our brains get too large and cause birthing problems, yet we have an extended childhood. As a consequence, we are receptive to inputs from the external environment, particularly the social environment, for an unusually long period of time, which contributes importantly to our enhanced cognition.

In Chap. 3 Sarto-Jackson describes how scientists drew on a machine analogy for the human body to infer that there is a “normal” way for a human to behave. Yet views of behavioral normality have changed strikingly over time (as recently as 1973 homosexuality was classified as a disease in DSM II<sup>1</sup>). Explanations for abnormal behavior that were scientifically respectable in the 19th century can appear ridiculous today. For instance, in 1851, Louisiana doctor Samuel Cartwright defined the medical disorder of “drapetomania” as “an irrational tendency of black slaves to flee captivity,” treatment for which was a combination of whipping and toe amputation! Other so-called diseases of the day included “dromomania,” an impulse to wander, and “inborn vagabondage” or “squalidness.” Pathologizing absconding and treating it as a congenital disorder was socially convenient as both policy makers and parents were absolved of responsibility.

This issue of the pathologization of abnormal behavior remains a matter of dispute. Sarto-Jackson discusses

contemporary intellectual conflicts between “naturalists” who attribute disease to abnormal biological functioning, such as genetic mutation, and “normativists” who regard it as physiological or psychological states that society doesn’t want or value. The former might explain, say, ADHD as resulting from dysregulation of neurotransmitters, while the latter critique its medicalization as designed to benefit society by removing disruptive children rather than to help the children themselves.

The issue is germane to the “neurodiversity” movement, which celebrates diversity in human cognitive functioning. Individuals diagnosed with autism, ADHD, dyslexia, dyspraxia, and many other “conditions” should perhaps be viewed as different rather than pathological. Such individuals are often able to function effectively, and even excel, by picking or constructing social worlds that suit their way of thinking and interacting. For instance, many autistic people have found the internet to have improved their lives, because it offers them ways to communicate that are both socially acceptable and less individually challenging. Here again, a linear causation model has, since the 1970s, been increasingly critiqued, and replaced with a conception of reciprocal causation stressing feedback loops between the organism and the environment. Society too can construct social environments designed to benefit particular individuals. Depending on the cultural niche in which children (who naturally vary in their attention, hyperactivity, and impulsivity) live, their “symptoms may be enhanced, suppressed or redirected into different behavioral patterns” (p. 83).

After discussing the aforementioned Waldroup trial, in Chap. 4 Sarto-Jackson raises other factors causally relevant to brain development, including epigenetics. Chemical attachments to DNA can lead to modification of gene expression, including by affecting gene methylation, histone functioning, or noncoding RNAs. This applies to humans too, for whom 40% of DNA is methylated, and where changes to methylation patterns are linked to many diseases. Environmental experiences, such as famine or exposure to pollutants, can switch genes on and off by changing these epigenetic marks, with these effects often carrying over generations. This allows the experiences of a mother or grandmother—including negative experiences such as trauma—to affect offspring physiology, behavior, and cognition. One consequence of the malleability of human brains is that they are vulnerable to negative impacts from disease or social experiences.

Sarto-Jackson then turns to discussing memory formation. In Chaps. 5 and 6 she describes how extreme localizationists held that individual neurons stored information about a given object or event (i.e., “grandmother cells”). In reality, a neuron that is activated when viewing a photo of a particular person also gets activated when reading that

<sup>1</sup> The Diagnostic and Statistical Manual of Mental Disorders-II.

person's name or seeing a person closely related to them. That is because memories do not remain isolated pieces of knowledge but get embedded into a larger framework of knowledge, an ever-growing network of interconnected content. Neuronal circuits are no longer regarded as fixed, but constantly reorganize themselves throughout an individual's life, with emotion now recognized as playing a key role in labelling and regulating memory content. Episodic memory is less like a tape of all that transpired and more a newsreel of the emotionally salient edited highlights.

This idea that emotions and memory are linked is a relatively new development. Recent neuroscience reveals that emotions play a significant role in memory formation, consolidation, and recall. We remember important moments of our lives well due to going through a state of emotional arousal, with emotionally charged memories becoming more strongly integrated in neuronal networks. This is what causes traumatized people to experience flashbacks as if the traumatic event is happening again, including experiencing the same emotions. Fearful memories may become pathological and develop into anxiety disorders and PTSD. Emotional tags govern neuroplasticity and ensure certain personally relevant events or facts are better remembered.

Moreover, through epigenetic inheritance, maternal emotion can also affect offspring development. Exposing a pregnant laboratory animal to high stressors, for instance, will cause lifelong changes in the physiology of her offspring by changing glucocorticoid secretion in the fetus, leading to larger stress responses and slower recovery from stress. If a pregnant rat encounters severe stressors, her offspring will typically grow up to be more anxious. In turn, heightened stress reactivity causes subjects to perceive ambiguous social cues as more negative. Traumatized people, and those exposed to chronic stress, can end up living in an emotionally re-labelled world in which the evaluation of social cues is biased towards the negative.

Research into epigenetic inheritance is also shedding new light on the impact of social neglect in childhood, as Sarto-Jackson outlines in Chap. 7. Experimental work in rodents shows how maternal care patterns can be epigenetically transmitted across the generations. Animals develop their stress response according to the level of environmental stress that their mother encountered. Thus neglectful, abusive, and violent parenting can have both direct and indirect negative effects on child development, with psychosocial stress strongly impacting epigenetic regulation of genes. Traumatic experiences early in life can also affect the child's developing oxytocin system and, in this way, cause adverse effects on attachment, bonding, and anxiety.

In Chap. 8, Sarto-Jackson highlights interactions between genetic predispositions and abusive childhoods. About half of boys with abusive childhoods grow into men who

commit violent crimes. Studies show that the aforementioned MAO-A gene variants purported to influence violent behavior only have detectable effects when perpetrators suffered childhood abuse. For Sarto-Jackson: "an individual's psychopathology is influenced by a crosstalk between his or her genetic makeup and childhood experiences" (p. 215). Physical, sexual, and emotional abuse, as well as stressful life events, cause excessive and prolonged activation of the stress response systems in the body and the brain. Traumatic experiences can reset this stress axis. Childhood abuse interferes with the extinction of conditioned fear. Individuals with PTSD, for instance, often struggle to inhibit their fear memories, even in the presence of safety cues, perhaps leaving them slow to trust others. Tragically, many severely neglected and abused children never form meaningful relationships later in life.

Parents who have been victims of domestic violence are at higher risk of exerting violence against their own children. A key claim by Sarto-Jackson is that childhood experiences shape cognition and provide a template of how social relationships with spouses, friends, and other people are expected to be. Thus, adult relationships reflect early social experiences and the attachment style developed in childhood. Humans sometimes generate social situations ("niches") to match their internal structures rather than trying to modify their internal structures. Alarming, when abused or neglected children become parents they themselves frequently create or pick abusive or neglectful familial environments.

Fortunately, not all abused or traumatized children exhibit destructive behaviors later in life: at least a third grow up into caring, competent, confident adults, with these survivors labelled "resilient." In the final chapter, Sarto-Jackson explores what resilient children have in common, in the process uncovering some positive and encouraging findings. Resilient children tend to have at least one concerned or compassionate relative or teacher able to compensate for the missing parental bonds. Seemingly, it only takes one caring individual to help right past wrongs. Likewise, parents or their surrogates have the ability to mitigate genetic predispositions or social and environmental risk factors through good parenting. For Sarto-Jackson, such caring adults operate by helping to construct a "good niche," just as abused and abusive individuals often construct "bad" ones.

A strength of Sarto-Jackson's book is its niche-construction perspective, which not only helps readers to understand how and why abused individuals can be trapped by circumstances and end up perpetuating abuse across generations, but also reveals how positive interventions can break the vicious cycle. As such, the explanation provides a more hopeful account than the oversimple and now discredited genetic determinist explanations that have dominated the

field. More than that, for Sarto-Jackson, neuroscientific knowledge can contribute to a self-help program that guides people who have suffered or are suffering from abuse, neglect, and trauma. Here the author embraces the “psycho-education” of psychiatrist Carol Anderson (Anderson et al. 1980), which sets out to empower persons directly or indirectly affected by adverse experiences. The implementation strategy is to provide educational information “about the neurophysiological and putative neuropathological effects of negative experiences” and the opportunity “to explore their thoughts and feelings related to the information,” whilst “avoiding the use of pathologizing language” (p. 240). Sarto-Jackson’s marvelously compelling and rigorous book puts the very best psychoeducation into practice, in the process offering inspiration and optimism to thousands who have directly experienced trauma and abuse, or witnessed its impact. The closing sentence captures her ambition: “to pave the way for handing back options for life-affirming actions to the person afflicted, thereby fostering a commitment to long-term involvement and self-efficacy” (p. 241). Far from being victims of the dictates of our genes, escape really is in our hands.

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