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Genetics, neuroscience and the narrative on eating disorders: Where science and storytelling meet

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By Emma Louise Pudge

The narrative around eating disorders is shifting. Researchers and activists have advanced a more complex understanding of eating disorders by presenting cutting-edge scientific and clinical research (increasingly incorporating insights from genetics and neuroscience) insisting on the importance of listening to and representing the experiences of marginalized groups. The concept of complexity, which can be defined as the state of having many different parts and being difficult to understand or find an answer to, has a long history within eating disorders research. Yet the cultural narrative surrounding eating disorders has often been overly simplified, contributing to stigma and reinforcing widely-held misconceptions about who is affected by eating disorders, what causes them, and the recovery process.

Within the medical and scientific community, eating disorders are commonly considered amongst the most complex psychiatric disorders. There is broad recognition that eating disorders are biopsychosocial in nature (Munro, Randell & Lawrie, 2017; Strober & Johnson, 2012). The etiology of eating disorders is generally considered multifactorial and thought to include a combination of predisposing genetic, biological and temperamental vulnerabilities that interact with environmental and sociocultural factors to increase risk within specific developmental contexts (Collier & Treasure, 2004). Anorexia nervosa, the rarest eating disorder, yet the condition that has received the most research and media focus, has the highest mortality rate of any psychiatric illness, estimated at 5% per decade (Birmingham et al., 2005), and is associated with a 50-fold increase in the relative risk of death from suicide (Keel et al., 2003). What's more, currently there are no effective psychopharmacological treatments for AN (Preston, O'Neal & Talaga, 2013), and the evidence-base for psychotherapeutic interventions is limited and conflicted (Fairburn, 2005).

Despite intensive research efforts, eating disorders remain something of an enigma. Their complexity presents a significant challenge for researchers, clinicians, caregivers and individuals affected by these

conditions. Yet this complexity has also inspired and justified research agendas and journalistic and narrative endeavors offering a wide variety of perspectives – each expressing, constituting and potentially re-writing the narrative on eating disorders. Whilst the notion of complexity is sometimes invoked to account for the lack of progress in understanding and treating these conditions, it has much to offer as a productive concept to guide how eating disorders should be conceptualized, narrated, researched and treated.

Eating disorders research has been dominated by two main paradigms: “a biopsychiatric approach emphasizing genetics, temperament, neurochemistry, and neural circuitry, and a sociocultural perspective focusing on multi-faceted, multi-channel social messages about gender, body shape, and eating” (Levine & Smolak, 2014, p. 158). Over the past decade, there has been large scale investment in neuroscientific and genetics research on eating disorders (e.g. Kaye et al., 2008; c.f. Chavez & Insel, 2008), reflecting the growing belief that this is the best way to advance our understanding of mental health and illness more broadly. The differences between biopsychiatric and sociocultural approaches may appear unbridgeable in this context, with biology-based explanations seeming to hold greater explanatory power, scientific legitimacy and cultural currency. However, neuroscience and genetics research is currently undergoing a shift towards thinking through complexity in terms of multiple, dynamic interactions between systems that were previously thought to function independently. Experimental research in this area suggests mechanisms through which genetic and environmental, neurobiological and psychological, individual and societal factors are drawn together to mutually affect each other (Bulik, 2005; Mazzeo & Bulik, 2009). Research on epigenetics and neuroplasticity in anorexia nervosa has begun to reveal how gene expression and the structure and function of neural matter is modulated through interactions with the internal and external, social and physical environment (e.g. Campbell et al., 2011; Connan et al., 2003; Hatch et al., 2010). Gradually this research is being extended to include other eating disorder diagnoses.

Notwithstanding the bridging to be found between biopsychiatric and sociocultural approaches, the discourse on eating disorders can be perceived as a battleground where competing narratives struggle for representation and authority. The persistence of binary thinking despite new evidence that suggests simplistic dichotomies ought to be rethought indicates that these persist for discursive and political reasons, rather than empirical reasons. Dr. Cynthia Bulik, Distinguished Professor of Eating Disorders in the department of Psychiatry at UNC School of Medicine, founder of the 20 country Genetic Consortium for Anorexia Nervosa (GCAN) and lead principal investigator of the Anorexia Nervosa Genetics Initiative (ANGI), has argued that pinpointing the genetic basis of anorexia

nervosa “may hold the key to unlocking the etiological mysteries of this debilitating disorder” (Bulik et al., 2005, S7), thereby enabling the development of targeted, more effective treatments. Bulik (2005) espouses the merits of a genetic etiological model whilst arguing that the etiological perspectives of environmental and sociocultural paradigms have only “face validity” and have therefore “greatly undermined efforts both to understand the neurobiology of eating disorders and to develop effective treatments for these often intractable illnesses” (p. 335). Looking forward, Bulik anticipates,

> “rather than relying on nonspecific blanket theories related to a universal exposure such as the presentation of thin ideals in the media, researchers may soon understand the genetic variants that make some individuals more vulnerable than others to environmental insults such as strict dieting prompted by exposure to extreme media ideals.” (2005, p. 335-36).

In this projection, Bulik’s critique of environmental and sociocultural research as contributing (only) “nonspecific blanket theories related to a universal exposure” disregards the diverse interests, methodological sophistication and empirical foundations and contributions of environmental and sociocultural paradigms. The implicit alignment of this research with inaccurate, simplistic explanations of eating disorders results in undue criticism of environmental and sociocultural perspectives (c.f. Levine & Smolak, 2014). Bulik’s efforts to challenge the nature versus nurture dichotomy at a molecular level are important and progressive. However, the point at which science and storytelling, research and rhetoric, meet is a critical juncture deserving of equally serious consideration. What happens in research spheres stretches outward and into the public sphere, impacting how people think and talk about matters such as eating disorders. The resulting shifts in thinking have material consequences, impacting the provision, allocation and direction of resources and funding. Neurobiological and genetic formulations of eating disorders do more than simply communicate changing scientific opinion: there are social, political and economic motivations for, and consequences of, this framing.

The recent expansion of interdisciplinary fields such as critical neuroscience (e.g. Choudhury & Slaby, 2012), feminist neuroscience (e.g. Schmitz & Hoppner, 2014) and critical medical humanities (e.g. Fitzgerald & Callard, 2015; 2016) have formed a bridge between the activities of social science research and empirical neuroscience. Experiments in neuroscience are often highly sophisticated in their formulation, use of cutting-edge technologies and ability to perform linkage analyses across numerous and varied complex traits. However, the transformation of

complex (and often ambiguous) data into elegant, all-explaining theories about the role of brain and genes risks reducing the cognitive, biological, emotional, sensory and somatic aspects of experience into a narrow set of physical processes. This kind of neurobiological reductionism opposes complexity and can be traced through the language used to communicate findings to an intended audience, detected as biases and interests informing the research design, and resulting from specific neuroscientific techniques such as fMRI. Self-reflexive scientific practices that interrogate the validity of neuroscientific findings – which, for example, investigate the impact that particular guiding assumptions, methodological constraints and complexities of data interpretation have on the outcome of research – ultimately lead to more empirically accurate and clinically useful knowledge. For instance, fMRI studies typically favor a 'snapshot' approach over a developmental approach and therefore cannot tell us about the processes that led to the developments identified on these scans. The majority of research investigating brain functioning in eating disorders is based on the 'recovery methodology' which measures and compares brain functioning during the active phase of the illness and after recovery. If alterations in parts of the brain that are known to correspond with eating disorder-related symptoms and traits continue to be observed after recovery, it is assumed that these existed prior to the onset of the disorder and must have played a causal role. However, it's possible that these differences instead represent 'scar effects' resulting from the disorder (such as the long-term effects of malnutrition) or other life experiences that preceded – or even precipitated – its onset. These include the myriad physical, psychological and social stressors which have been shown to affect brain development and function (Favaro, 2013). Based on this interpretation, these abnormalities may not represent 'hardwired' differences in the brains of people who experience eating disorders after all, and it is important that this is recognized to avoid overstating conclusions.

Critical neuroscientific work also resituates and redistributes concerns about reductionism within a much broader intellectual landscape. It acknowledges that reductionism is not necessarily an inherent feature of neuroscience; rather, approaches and explanations that may be considered 'reductionistic' are co-created and maintained in the interactions between multiple institutions, interests and circumstances (c.f. Choudhury & Slaby, 2009; 2012). Metaphors of the brain as 'hard-wired' or 'pre-programmed', comparable to computer software, can be helpful in communicating complex phenomena using structured and familiar concepts, especially in the interactions between neuroscientists, the media and the general public. Metaphor is a key strategic resource for scientists and journalists in the narration and storytelling of scientific findings. However, metaphors are often misinterpreted as objective descriptions of how the brain 'really is' rather than understood as a rhetorical strategy

that involves aligning the brain's biological reality with a fundamentally different object or concept. The methodological weaknesses and processes of distortion involved in producing rather than straightforwardly acquiring images of the brain are also rendered invisible. As with empirical scrutiny, discursive analyses are committed to upholding the highest standards of empirical and experimental neuroscience, and ultimately help to maintain the integrity of science as a whole. By revealing how scientific conclusions are distorted by the imprecise use of scientific terminology, these critiques help to mitigate the hype and unrealistic expectations that can result in disappointment and the misallocation of resources precisely. Crucially, they hold the shared aim of improving the prognosis and lives of people affected by eating disorders. As a result, discursive analyses acknowledge and grapple with the paradox that problematic and reductionistic explanations may on occasion confer strategic benefits by contributing to a politically persuasive narrative that elicits the desired responses from key stakeholders.

In 2009, the Academy for Eating Disorders (AED) published a position paper with the title, Eating Disorders are Serious Mental Illnesses. The authors assert that "anorexia nervosa and bulimia nervosa, along with their variants, are biologically based, serious mental illnesses (BBMI)" (Klump et al., 2009, p. 97) according to a definition of a BBMI as "a condition that current medical science affirms is caused by a neurological disorder of the brain" (p. 98). This, they argue, can be supported with "a reasonable degree of medical or scientific certainty" (ibid.). However, the data used to support this argument pertaining to heritability, alterations in brain structure and function, and emotional and cognitive deficits is not unambiguous, and the neuroscientists and genetics researchers they cite are often more circumspect in their original conclusions (e.g. Trace et al., 2013). In an article outlining the National Institute of Mental Health's (NIMH) perspective on eating disorders and its research funding priorities, the former NIMH director acknowledges, "although eating disorders are presumably brain disorders, very little is known about their pathophysiology" (Chavez & Insel, 2008, p. 8). Technological advances in neuroimaging and gene mapping hold promise in elucidating contributing biological factors and for developing precision treatments. However, so far, the preliminary insights regarding genetic, neuroanatomical, biochemical and neurohormonal correlatesof eating disorders do not meet the strict scientific standards of causality.

From the shared perspective of those committed to eating disorder advocacy, however, the sociopolitical consequences of assigning biology a specifically causal role is more salient than the semantics or even scientific accuracy. The authors of the AED position paper clearly state that classifying eating disorders as brain-based mental illnesses is intended to ensure that eating disorders "receive health care coverage

and research funding that is equal to that of medical disorders as well as psychiatric conditions categorized as serious forms of mental illness" (Klump et al., 2009, p. 100). A close examination of the figures relating to research funding reveals why this goal requires urgent and aggressive pursuit (MQ, 2015). In the UK, the average amount spent on research per adult affected by mental illness is £9.75 – less than 0.1% of the average £1,571 spent on research per adult affected by cancer (p. 3). In the case of eating disorders, this figure drops to just £0.15 – less than 0.01% of research spending per adult affected by cancer (p. 12). In other words, we are spending at least one hundred times less on potentially life-saving research per adult affected by an eating disorder versus cancer. The UK spends on average £115 million on mental health research, yet only £0.5 million on eating disorders specifically (ibid.). The total economic and social cost of mental health problems in England is estimated to be £105 billion, and £15 billion is thought to be attributable to eating disorders alone (p. 3). Eating disorders therefore contribute to nearly 15% of the economic burden, yet research on eating disorders receives just 0.4% of UK mental health research expenditure. Already, research spending per adult affected by eating disorders equates to just 1.5% of average research spending per adult affected by mental illness (p. 12). There is significant disparity between different conditions, with eating disorders receiving just 0.24% of research spending per adult affected by psychosis (including bipolar and schizophrenia) (p. 9). Given that psychosis is typically presented as a condition with a particularly strong biological and genetic underpinning, discussing eating disorders in this way may help to build the case for equal research funding.

A similar picture is painted in the US where a funding report by the US National Institute of Mental Health demonstrated that across all psychiatric conditions, eating disorders are met with the greatest discrepancy when research funding is evaluated against the burden of illness they represent (Murray et al. 2017). As in the UK, the discrepancy is highest between eating disorders and other psychiatric conditions more widely recognized to have a biological and genetic basis. In 2015, federal funding for eating disorder research equated to approximately $0.73 per affected individual – just 0.8% of the $86.97 (over 100x less)spent per individual affected by schizophrenia (Insel, n.d.). Moreover, specific research has been conducted analyzing National Institutes of Health (NIH) funding trends over the past decade according to whether clinical trials on anorexia nervosa tested exclusively biological models, versus biopsychosocial formulations that considered psychological and social aspects also (Harris & Steele, 2014). The findings demonstrate that brain studies have been the most generously supported studies to date and spending continues to increase, whereas studies taking a biopsychosocial perspective start as the lowest funded studies, and funding has decreased since 2006, almost to the point of non-existence by 2012 (p. 91).

Researching eating disorders from a biopsychosocial perspective is increasingly untenable, not for scientific reasons but due to a lack of political motivation and funding. Meta-analyses are generally considered the strongest form of scientific evidence. They are, in many ways, scientific metanarratives, offering totalizing, definitive accounts of scientific phenomena. If biopsychosocial research does not receive sufficient funding, it will not be possible to replicate studies and generate the volume of data required for meta-analyses. As a result, minor biopsychosocial narratives will be overshadowed by a neurobiological metanarrative deduced from the conclusions of meta-analyses. Framing eating disorders as brain-based mental illnesses may therefore be an effective strategy to stimulate total research spending, and individual researchers may be persuaded by the allure of research grants and greater potential for career advancement. However, an excessively narrow neurobiological approach may in fact widen the gap between achieving the immediate goal of increased investment in research, and the ultimate goal of improving the outcomes and lives of people affected by eating disorders. Given that even this short-term aim of adequate investment has not yet been realized, neurobiological and genetics research on eating disorders remains in its infancy. It will require sustained, large-scale financial investment over time before we will know whether this approach will succeed in delivering the promised transformation in scientific understanding and clinical outcomes. Arguably, the likelihood of receiving this investment is enhanced by propelling biology-based narratives. However, in view of the lack of definitive evidence for neurobiological models of eating disorders, it is crucial that the intellectual and ethical implications of this strategy are carefully weighed against the economic and political motivations.

Chavez and Insel of the NIMH have argued that “considering eating disorders as medical illnesses with pathophysiology that can be elucidated with the modern tools of genomics and neuroscience offers the best hope for finding cures or preventive strategies.” (2008, p. 2). Yet even if further evidence accumulates to support the assumed neurobiological basis of eating disorders, it does not necessarily follow that the future eating disorder treatment and prevention lies in targeting the brain or genome directly. Bulik (2005) has argued that “genetic research has paradoxically opened up the potential for greater specificity in our understanding of the role of environment” (p. 335) and that “it may be possible to model the manner in which genes and environment mutually affect each other” (p. 337). Based on the understanding that genes and environment “coevolve and interact, which is to say that neither influence is fully deterministic” (Strober & Johnson, 2012, p. 163), it is not beyond the realm of possibility that the more appropriate, feasible, ethical and impactful site of intervention may be the sociocultural environment, rather than the human genome.

Stigma-reduction efforts are also shaped by the narrative framing of eating disorders. Proponents of genetic explanations believe these will reduce stigma by relieving personal responsibility, blame and guilt. However, this represents only one side of the story as there are two main types of stigma affecting eating disorders: the stigma of being considered to be mentally ill, and stigma relating to the perception that eating disorders are behavioural choices (Easter, 2012, p. 3). Whilst the genetic presentation of eating disorders has the potential to alleviate the latter type of stigma described, it may have the unintended consequence of exacerbating the former. Bulik (2013) has publicly argued new insights into the genetic basis of eating disorders are helping to 'fight stigma with science'. However, research on how the public interpret genetic explanations of mental illness suggest optimistic predictions of increased compassion and reduced stigma are rarely borne out (Angermeyer et al., 2011). Instead, genetic presentations tend to increase prejudice, fear and social distancing by others, and may be internalized by people affected by eating disorders. When women who had experienced eating disorders were interviewed about how they interpreted genetic presentations and the impact they believed this would have on stigma, half the respondents raised concerns that this framing could lead to fatalism and hopelessness (Easter, 2012, p. 10). Since neuroscientific explanations contribute to notions of "hardwiring", an emphasis on genetics was interpreted as suggesting the eating disorder was an essential and permanent aspect of the person – "part of their DNA" – which limited their ability to envision ever being "free" from having an eating disorder (p. 9).

If we are to accurately represent the full breadth of research and experiences relating to eating disorders, we need to complicate and move beyond dichotomous understandings of brain-based and genetic narratives versus narratives that emphasize sociocultural and environmental factors, as well as individual biography. Science and storytelling are deeply entangled – as are we, in our various roles as researchers, writers and scientific storytellers, with those whose realities we attempt to represent. Genetics and neuroscience research has already undergone a shift towards thinking in terms of dynamic interactions between biology and biography, genes and environment, individual psychology, life experiences, and wider social systems. However, the point at which science and storytelling meet is a critical juncture that has received significantly less attention. The mutual shaping of scientific and popular opinion means that socially responsible reporting of neuroscience on eating disorders must include consideration of the implications of how research is discussed, as well as the specific findings that are disseminated. Recognizing that science and storytelling do not sit in opposition but rather unfold together through complex interactions will allow us to pursue neuroscience and genetics research on eating disorders whilst remaining sensitive to the challenges, limitations and

potential consequences of this approach. This offers a more ethically and empirically sound way to advance our understanding of eating disorders and may lead us toward biopsychosocial models with far-reaching implications for research, treatment and prevention also. Perhaps most importantly of all, by adopting this approach, the narrative on eating disorders remains open to an infinite number of revisions, retellings and reinterpretations.

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