BOOK SYMPOSIUM

Pluralism, social action and the causal space of human behavior

Helen Longino: Studying human behavior: How scientists investigate aggression and sexuality. Chicago: The University of Chicago Press, 2013, 256pp, \$25 PB

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Helen Longino's *Studying Human Behavior* is an overdue effort at a nonpartisan evaluation of the many scientific disciplines that study the nature and nurture of human behavior, arguing for the acceptance of the strengths and weaknesses of all approaches (as opposed to the vitriolic defense of one and lambast of others). After years of conflict, Longino makes the pluralist case for peaceful coexistence. Her analysis of the approaches raises the following question: how are we to understand the pluralistic relationship among the peacefully coexisting approaches? Longino is ironically rather unpluralistic about her pluralism, forcing a choice between integrative pluralism and her preferred ineliminative pluralism. I hope to show that the analysis of approaches she offers actually accommodates a pluralism that is both integrative and ineliminative.

Approaches to studying human behavior

Philosophy of biology took shape as a discipline in the 1970s. This disciplinary formation overlapped with two episodes in the nature/nurture debate—the IQ controversy and the sociobiology controversy. These episodes polarized the scientists involved. Champions of the nature side of the debate emphasized the

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role of genes and genetic differences in the development of and differences in human traits like IQ and altruism. Advocates for the nurture side of the debate criticized the methods, assumptions, and generalizations used to make inferences about what genes do. Philosophers of biology, drawn to these heated scientific controversies with important sociopolitical implications, often followed in a dichotomized fashion. They jumped into the fray by picking one of the sides, trumpeting that side's virtues, and deriding the other side's vices. Reading the extremes in this literature can be as disorienting as flipping back and forth on cable news between a progressive station and a conservative station (if you are in the US, try MSNBC and FoxNews on any given evening).

Longino's book is a refreshing attempt to move past this trend. "My aim, unlike that of many recent writers," Longino begins, "is not to enter the nature/nurture debate." (2) Rather than picking a side, Longino instead treats the multitude of approaches that study human behavior on their own merits, noting each's strengths and weaknesses. You will not find any cantankerous barbs designed simply to belittle an opponent in her book, and it is relieving. "We can learn more by trying to get a handle on the complexity of the research landscape than by engaging in partisanship." (15)

The book is united around two genuinely intriguing behaviors-one that drives humans apart (aggression) and one that draws humans together (sexuality). With these case studies as the unifying themes, Longino provides impartial introductions to a handful of the various disciplines (or "approaches") that study such human behaviors, noting their respective problems of focus, methods, assumptions, and limitations. The chapters in Part I are each devoted to one of these approaches (along with a summary chapter 7). Quantitative behavioral genetics (chapter 2) studies human behavior with data from twin/adoption studies and uses tools like the analysis of variance to measure the heritability of traits; quantitative behavioral geneticists identify the causes of variation responsible for variation in behaviors in order to determine how much of the variation in, say, antisocial behavior is due to genetic differences and how much of it is due to environmental differences. Socialenvironmental approaches (chapter 3) study human behavior by focusing on the determinants of behavior in the social environment; in contrast to quantitative behavioral geneticists, scientists who study the social/environmental side do not aim to distinguish genetic contributions from environmental contributions, but rather aim to discriminate among the contributions made from the environmental determinants-whether, for example, exposure to violent media or child abuse is more responsible for antisocial behavior. Molecular behavior genetics (chapter 4) utilizes gene-finding methods (such as linkage, association, and genome-wide association studies) as well as molecular methods that link particular segments of DNA with functional protein products in an attempt to make a gene-behavior link, such as associating a mutation in the gene that codes for monoamine oxidase A with aggression. Neurobiological approaches (chapter 5) identify the neural structures and processes involved in human behavior; these approaches rely on methods ranging from neural imaging to pharmacological interventions in order to make a brain-behavior link, such as linking a defective limbic system with aggression. Finally, a number of integrative approaches (chapter 6), such as developmental

systems theory and a gene \times environment \times neurobiological approach, combine results from several of the aforementioned approaches.

Pluralism: integrative versus ineliminative

What is the relationship between these different approaches that study human behavior? Longino turns to answering this question in Part II (chapters 8, 9, and 10). One possible answer would be that one of the approaches is right and the others wrong. This was the answer that many nature/nurture disputants chose (scientists and philosophers alike). Longino points out, though, that this answer (a type of "monism") does not hold up to the survey of approaches she supplies in Part I. It is true that the different approaches to studying human behavior can appear competitive-for sources of funding, for attention from the media, for fulfilling the promises to "win the race" of understanding human behavior. But this competition, Longino warns, belies the fact that the approaches actually overlap surprisingly little. For example, although all of the approaches attempt to answer the question, "what causes human behavior?" in actuality the different approaches focus on very different portions of what Longino calls the "causal space" of human behavior-genetic causes, environmental causes, neurobiological causes (chapter 8). Likewise, although all of the approaches attempt to answer the question, "what causes human behavior?" in actuality "behavior" means different things in those different approaches—a tendency in a population, an episode in the history of an individual, a disposition to respond to situations in one way or another (chapter 9). Finally, although all of the approaches attempt to answer the question, "what causes human behavior?" in actuality the approaches communicate quite poorly with one another when it comes to research uptake (chapter 10).

With monism dispatched, Longino turns to the alternative—pluralism. But what kind of pluralism? Integrative pluralism is one option. Integrative pluralism, developed by Sandra Mitchell, allows for a plurality of approaches to exist, but they are not isolated; the answers and results from one approach can co-inform the questions and investigations of another (Mitchell 2003). I, like Mitchell, have explicated how integrative pluralism works in science, and Longino groups Mitchell and I together as representatives of this "moderate pluralism" (144–148). We are moderate pluralists, according to Longino, because the integrative pluralism we advocate is but a temporary way-station to be eliminated on the way to an integrated monism. The problem with this version of pluralism is that it "presupposes a commensurability that may not obtain in all cases and does not obtain in the case of behavior." (147) This is a point that Longino drives home over and over throughout the book-a grand integration of all the approaches is epistemologically and ontologically impossible because of the differences between them. So Longino is a pluralist, but her pluralism is not of the temporary, integrative sort; it is ineliminative: "We are left, then, with a plurality of approaches generating accounts of the etiology of individual behavioral dispositions that are not reducible to some fundamental level of causation, not integratable into a single comprehensive account, and not empirically commensurable in a way that would permit elimination of rivals in favor or one." (135)

Pluralism: integrative and ineliminative

Longino offers us an ironically monist vision of pluralism; you are either a moderate, integrative pluralist or you are a strong, ineliminative pluralist. It would be wiser, I will argue, to be pluralists about our pluralism. Let me explain by way of briefly reviewing the history of research on the causes of depression. I discuss this example in detail in my forthcoming *Beyond Versus: The Struggle to Understand the Interaction of Nature and Nurture*, but an abbreviated version will convey the point (Tabery 2014).

Why do some people develop clinical depression, while others do not? Throughout the 1980s, a variety of epidemiological research suggested that both genes and the environment contributed to the risk of developing depression. On the environmental side, research by George Brown identified stressful life events (such as divorce and serious illness) as contributors to depression (Brown 1987). On the genetic side, a number of twin and adoption studies from quantitative behavioral geneticists implicated a genetic component in depression and associated traits, such as neuroticism (Meltzer and Arora 1988; Loehlin 1989). These epidemiological studies suggested that stressful life events and something genetic acted as causes of variation responsible for variation in depression. But these studies did not elucidate how those causes of variation made their difference. How does stress "get in the brain," as it were? And how do genes actually contribute to this process?

Answering the "how" questions involved the story of serotonin. Serotonin was discovered in the 1940s and linked to brain function in the 1950s. Neuropharmacologists subsequently learned that drugs acting on serotonin levels could treat a range of diseases and disorders; many of these drugs, it became clear, affected serotonin levels by acting on the serotonin transporter. The serotonin transporter is a protein that resides on the membranes of neuronal synapses; during neurotransmission, when serotonin floods into the synapse, the serotonin transporter reabsorbs serotonin back into the presynaptic neuron, thus terminating serotonergic neurotransmission. By 1990, serotonin and reduced serotonin transporter activity in particular were linked to increased risk of depression (Meltzer 1990).

With quantitative behavioral genetic research suggesting that something genetic was contributing to depression risk and neurobiological research suggesting that somehow the serotonin transporter was involved, the next question became: what is the genetic basis of the serotonin transporter, such that differences in it account for variation in depression risk? Klaus-Peter Lesch spent much of the early-to mid-1990s answering precisely this question. He and his colleagues first identified and localized the gene responsible for producing the serotonin transporter; they then tracked down the source of variation in that gene that would account for variation in the serotonin transporter product—a polymorphism in the promoter region, which can be relatively short ("s") or long ("1") (so the individual possibilities are s/s, s/l, or l/l). This physical difference in length translated into both molecular and behavioral differences; at the molecular level, cells with the l/l-form were twice as efficient at serotonin uptake as cells with either the s/l- or s/s-form, and individuals with either the s/l or s/s forms scored significantly higher on measures of neuroticism than individuals with the l/l-form (Lesch et al. 1996).

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Lesch's research on the serotonin transporter gene was designed to first identify and then link up a genetic difference with a behavioral difference. The serotonin transporter gene directly affected the efficiency of serotonergic neurotransmission, and that efficiency in turn resulted in behavioral differences. But just how that molecular level linked up to the behavioral level remained unclear. Several neurobiological studies at the turn of the twenty-first century led by Daniel Weinberger made this connection. Weinberger's team attended to both brain function and brain morphology. Focusing on the amygdala (a brain region involved in emotional learning and memory), they utilized neuroimaging to assess how differences in the serotonin transporter gene translated into differences in brain activity; they found that individuals with either the s/s- or s/l- forms experienced much greater amygdala activity than individuals with the l/l-form when exposed to fearful stimuli (Hariri et al. 2002). Individuals with the short form also had smaller amygdala (and cingulate) volume than individuals with the long form, as well as poorer amygdala-cingulate connectivity (Pezawas et al. 2005).

Although Lesch did associate differences in the serotonin transporter gene with differences in behavior, it should be emphasized that the difference at the genetic level accounted for a very small portion of the total variation in the behavioral level-3 to 4 %. The serotonin transporter gene was a very poor predictor when taken by itself. Around the same time that Weinberger was examining the neurobiological implications of differences in the serotonin transporter gene, Terrie Moffitt and Avshalom Caspi thought to combine the serotonin transporter gene with the earlier environmental research on depression. On the genetic side, Moffitt and Caspi divided up research participants based on which form of the serotonin transporter gene they carried, and on the environmental side they divided up research participants based on the number of stressful life events they had encountered in life. The combined genetic and environmental variables made for a much stronger predictor of depression than either of the variables alone. Individuals with the s/s-form were much more susceptible to developing depression when exposed to stressful life events compared with individuals with the l/l-form; indeed, individuals with the s/s-form who were exposed to four or more stressful life events had approximately a 40 % probability of experiencing an episode of major depression, and if they were exposed to childhood maltreatment that risk jumped up to 60 % (Caspi et al. 2003). Moffitt and Caspi have more recently teamed up with neurobiologist Ahmad Hariri (a former member of Weinberger's lab) to offer a picture of how all this research points to a "unifying mechanism"; individuals with the s-form of the serotonin transporter gene are at increased risk of negative affectivity, but that negative affectivity is only converted to a psychopathological trait like depression when it is exposed to environmental stressors-"just as glass is always characterized by the trait of brittleness, but shatters only when a stone is thrown." (Caspi et al. 2010, 515)

Moffitt and Caspi's proposed unifying mechanism is now at an interesting crossroad. Their initial study was replicated several dozen times, with many of those replications positive and many negative. This mixed-bag result led several different teams of researchers to perform meta-analyses on the replications; but rather than solve the problem once and for all, they only perpetuated it by presenting their own

mixed-bag of meta-analyses; two came back negative and one positive (a fourth is in the works). I explain why these meta-analyses of the purportedly same phenomenon reached opposite conclusions in Beyond Versus, but for our purposes here let us ignore the recent episode concerning the dueling meta-analyses and focus on the broader history of research on the causes of depression. Research from quantitative behavioral genetics, social-environmental approaches, molecular behavior genetics, and neurobiological approaches all contributed to the current picture of the nature and nurture of depression. The process was both integrative and ineliminative. It was integrative in that the scientists from these various approaches participated in a co-informational process wherein the answers and results from one approach could shed light on the questions and investigations of another. The quantitative behavioral genetic approach and the social-environmental approach both pointed to something genetic and something environmental as playing a role in the development of and differences in depression. The molecular genetic approach followed up on the quantitative behavioral genetic result to look for a gene that could link up differences at the genetic/molecular level with differences at the behavioral level. When a candidate was found (the serotonin transporter gene), neurobiologists attempted to track differences in that gene up through differences in brain function and morphology. Finally, Moffitt and Caspi combined that genetic thread of the research up with the environmental research on stressful life events to offer a unifying mechanism accounting for how individuals with different forms of the serotonin transporter gene responded differently to stressful life events based on different neurobiological responses to stress. The integrative pluralism I have described above, however, was not an eliminable waystation on the way to monism. The pluralism was also ineliminative. The quantitative behavioral genetic approach did not reduce to the neurobiological nor did the social-environmental become obsolete after the molecular behavioral genetic approach came along. They all contributed to the product.

Longino actually discusses Moffitt and Caspi's research at length as an example of one of the "integrative approaches" (discussed in chapter 6); however, she warns that their gene \times environment \times neurobiological approach is confined in its application to only psychiatric disorders (95, 99, 112, 116). Two things to say here: First, it is true that Moffitt and Caspi's research is integrative, but the history of research on depression discussed above revealed that the approaches were integrating before Moffitt and Caspi entered the story. The neurobiological approach built off the molecular behavioral genetic approach, which itself built off the quantitative behavioral genetic approach... all before Moffitt and Caspi came along to integrate research from the social-environmental approach. Second, their integrative approach is not confined to psychiatric disorders. It is true that they have focused primarily on psychiatric disorders (antisocial behavior, depression, and schizophrenia), but they have also studied IQ (Caspi et al. 2007). Moreover, their integrative strategy has been extended to a range of non-psychiatric behaviors, such as memory and externalizing behavior. Psychiatric disorders do present a particularly neat category because researchers can divide up populations based on those with a diagnosis and those without, but this neat categorization usually hides a spectral phenomenon that psychiatrists simply divide at some point. All a trait needs in order for it to be studied with Moffitt and Caspi's approach is to display a similarly spectral quality; so, for example, memory can be divided up between those with better memory and those with worse memory, and then researchers can look for whether or not differences at the genetic level translate into differences at the brain-system level and are impacted by differences in environmental exposure to lead to differences in memory.

Pluralism about pluralism

Helen Longino has done the philosophy of science a service by providing a nonpartisan assessment of the many scientific approaches that study the nature and nurture of human behavior. She convincingly shows that we need not pick winners and losers among the approaches, but rather can think constructively about the relationship between those approaches. I hope to have shown that Longino's point applies equally well to the philosophy of pluralism. We need not pick winners and losers when it comes to integrative pluralism and ineliminative pluralism; we can think constructively about the relationship between those philosophies.

Alex Preda

Over the last 20 years or so, a distinct set of disciplines have emerged, known as the behavioral sciences, situated partly within the domain of the life sciences and partly within that of the social sciences (I will call them here life-behavioral disciplines). Building on technological advances, but also on advances in quantitative statistical techniques, research programs located within the life-behavioral disciplines have set out to "decipher" the biological bases (understood as determinants) of human behavior. No small role in this enterprise has been played by advances in new technologies of genetic sequencing and analysis or in brain imaging, among others. Over a relatively short period of time, these technologies have become established within the institutional structures of scientific research and appeared to make good on the (old) promise of delivering a better, precise understanding of the determinants of human behavior. A better and precise understanding would consist of course in unlocking the biological factors (seen either as genetic factors or as neuro-chemical processes) which cause particular forms of social behavior. Unsurprisingly, sexuality and aggression are prime candidates for deciphering and, as Helen Longino points out, political agendas and debates from the 1990s and the early 2000s have contributed to putting them firmly at the center of new research programs. (They were very much present in previous ones as well, albeit not in this novel formulation.)

Expectedly, strong counter-reactions from the (more traditional) social sciences have appeared without much delay. One is reminded here of the heated controversies from the early 1980s around sociobiology or of the more recent controversies within cultural anthropology around claims of sexual and reproductive behavior being genetically determined in small isolated communities (or "primitive tribes"). Are we re-experiencing now the old nurture/culture debate, only on a new plane and anchored in a new set of almost exclusively quantitative techniques? Or

are we re-experiencing this debate not only on a purely intellectual plane but also in relationship to the funding of research programs, to establishing long-term research agendas and to the institutionalization of a new research paradigm? The stakes and especially the policy stakes are high (both within science policy and social policies), and it is unsurprising that we hear the research findings mentioned in this book being invoked again and again in public debates about policies ranging from rights to education, to correctional reforms. We hear less about the impact on research funding and science policies, but this does not mean that there is not any—quite the contrary.

Meanwhile, there are spillover effects outside policy domains as well. If the biological determinants of human behavior have finally been found, we can well imagine businesses will be interested in improving productivity and efficiency through some form of intervention in neurochemical processes for instance. This sounds way too far fetched, you say? A few years ago I attended a conference in my own field, financial behavior, where a presenter laid out exactly such a plan for making traders win more.

In this re-staging of an old debate, the concept of (human) behavior has become murkier and murkier, together with some other relevant concepts such as environment or causation, as Longino rightly points out. I cannot stop noticing here that only a few decades ago many in the social sciences (including here anthropology and sociology) shunned the notion of behavior, preferring to talk instead about social action. There is little by way of this alternative concept in Longino's book, since the notion of behavior has risen to preeminence again.

What happened to the notion of social action? Has it been lost in translation? There is a long tradition in the social sciences, not least in sociology and social anthropology, of thinking of sexuality and aggression, among others, in terms of social action and of culture, respectively, as way of emphasizing both the variability and the complexity of these phenomena, and their dependence on social norms. In this perspective, sexuality and aggression are not only contextual social phenomena; they are socially organized, and our understanding of what constitutes instances of deviance, for instance, depends on social norms. This time-honored line of thinking has led to focusing the research on the social mechanisms, which support the organization and reproduction of what we would call patterns of sexuality or of aggression, to the effect sometimes, that such patterns appear as rational responses to environmental resources and constraints. Let me give just an example here: the relatively recent research conducted by Sudhir Venkatesh (2008) and Stephen Leavitt and Steven Dubner (2009), respectively, suggests that deviant activities (including here crime and aggression) are highly organized, entrepreneurial, dependent on local resources and constraints, and geared toward achieving particular efficiencies. Any policies attempting to tackle them are doomed to fail if they do not take these aspects into account.

Yet the view that the concept of social action should be taken seriously seems to be locked in an asymmetric dispute with that of behavior: asymmetric because the latter can mobilize a different and larger array of material technologies (and hence of institutional structures) in its support. If one were to put it in the concepts introduced by Bruno Latour, the latter concept mobilizes a more complex network of human and material devices in its support. Has the concept of social action been indeed lost in this translation involving genetic analyses and MRI scans?

Studying Human Behavior, which outstandingly presents and discusses four approaches to aggression and sexuality-out of which three are located within the life sciences-would seem to indicate so. Yet, I could not keep but thinking of the rich traditions of sociology and of cultural anthropology in investigating these issues, and perhaps, just to keep the balance, they could have been brought into the picture as well, especially since anthropology's analytical toolkit differs from the regression modeling favored by all the approaches discussed here. Perhaps, the actual conceptual issues at stake could be better highlighted by putting side to side and comparing how the life-behavioral disciplines and the social scientific disciplines approach the issues both from the viewpoint of their basic notions and from that of their analytical toolkit. All too often, concepts, the methodological toolkits used in supporting them, and the data obtained with said toolkits stand in symbiosis. A particular theoretical notion is achieved because of the constraints posed by using a particular technique for generating data (and the associated material setups), which in turn require specific analytical techniques reinforcing the said notion. There is codependency among these aspects of empirical research, a co-dependency which can be better highlighted by widening the array of approaches being contrasted here.

This makes me think that putting side by side these life-behavioral disciplines with cultural anthropology and with ethnographic sociology (which use different procedures for obtaining their data) would have highlighted better both the collinearity of concepts, techniques and data, as well as the boundaries of each of these approaches. One aspect which immediately comes to mind is the difference in the techniques used for generating data, and hence in the ways in which the latter support different concepts: while the life-behavioral sciences use mostly surveys for obtaining data about sexuality, aggression or other social forms of activity, social anthropology and ethnographic sociology rely much more on direct, in situ observations over longer periods of time. The data obtained is naturalistic, rich in detail, but also less prone to be used in regression based models-and hence less prone to be integrated in a causal account together with biological data. This makes me think that while social anthropology and ethnographic sociology are relying more on a traditional scientific method-direct observation-and obtain thus richer and more complex data, this latter do not lend themselves to reduction and integration. Hence, to borrow again from the terminology of Bruno Latour, these data are less prone to be mobilized as part of a heterogeneous network of material technologies, institutions, and human actors.

Longino's analysis makes clear that there is little, if anything, by way of a shared concept of behavior among the disciplines she discusses; that the concept of environment is no less unclear; that claims of causation can be seriously exaggerated, especially in media presentations of research results; that the institutional development of this group of approaches has not favored interdisciplinary communication. This leaves us with what she calls a conceptual kaleidoscope or pluralism which might not be eliminated any time soon or which, indeed, is ineliminable. The advantage of such a kaleidoscope would be that each piece of research illuminates a different aspect of human behavior, from a different angle, and thus, in time we could hope to achieve a better, yet not (or never, for that matter) fully integrated understanding of behavioral determinants.

If there is something like a conceptual kaleidoscope or pluralism, then should not social anthropology or ethnographic sociology, with their different emphasis, be part of it? True, they shun the notion of behavior and prefer those of social action or culture. Yet, as in any real kaleidoscope, one can see the boundaries, shape and color of a particular piece only in relationship to (and as distinct from) other pieces. Therefore, if we are to have a conceptual kaleidoscope here the notion of social action should be part of it, not least perhaps because the concepts of action and behavior are bounded by each other. This is so because the concept of human behavior is assumed (even by the life-behavioral sciences) to be distinct both in its character and in its implications, and not entirely reducible to a category such as "primate behavior" or "mammal behavior". After all, nobody wants to discuss policies to tackle the aggressive behavior of, say, dolphins. The nature–culture divide is present within the very concept of human behavior. It would be wrong to believe that the concept is on one side of the divide—it is rather the divide which inhabits the concept.

Notions such as intentionality, mutual orientation or communication have often been invoked in arguments that (social) action as a conceptual construct has the advantage of capturing aspects which are difficult to operationalize analytically with the notion of behavior. There have been many arguments for instance that both aggression and sexuality imply interactional formats which are consequential for these very notions and that these formats cannot be fully captured with survey techniques and that the same formats are context bound and have hence certain variability. Such an alternative approach—and the more micro-analytical toolkit it makes use of—leaves room for acknowledging the significance of social forms as consequential for the phenomena at stake. Acknowledging and putting social science research which pays attention to social forms side by side with life science approaches would complete the kaleidoscope in question and would expand the notion of pluralism at stake here.

Another issue worth discussing here is the linkage between conceptual and institutional developments: as Helen Longino makes clear, authors involved into each of these directions of research, and their publics, have little dialog, if any, at least not by way of academic publications. It would have been interesting indeed to investigate more whether the (irreducible) conceptual pluralism is logical necessity, or rather a consequence of the institutional development (including here technologies and analytical toolkits) which has favored the emergence and academic establishment of distinct disciplinary fields? In other words, are we faced with a conceptual pluralism as something unavoidable (on logical grounds), or are we facing here the outcomes of particular institutional developments, which reflect (mostly) the social dynamics of the research landscape? Life-behavioral studies have been made possible by recent technological developments, among others by gene sequences technologies and MRI scans; the spread of such technologies within research communities has not been without institutional consequences, manifest not least in long-term research programs supported by national funding bodies, journals, conferences, and the like. Research results are regularly reported in the media, albeit

more or less accurately. Nevertheless, this reporting is part and parcel of the institutionalization process, especially in an age where "impact outside the academia" plays a role in funding decisions.

I think this question is not an unimportant one: if we opt for considering conceptual pluralism in relationship to the developments in the research landscape and the academia, we might come to regard it less as a logical necessity than as a the necessary outcome of particular institutional dynamics: of the ways in which agendas, programs and laboratories get established, grants acquired, chairs funded and results publicized. Moreover, if we opt for this second alternative, it means that the ways in which we conceptualize human behavior are not disconnected from the institutional processes supporting specific research agendas. By now readers have recognized an argument familiar from science and technology studies, namely that scientific knowledge cannot be disconnected from the groups and institutions involved in establishing particular agendas, and from the relationships among such groups. It is an argument which has been put forward in numerous historical studies of scientific concept formation and which, more and more, has been applied recently to the social sciences as well. It is perhaps time to extend it to disciplines and approaches which straddle the boundaries between the natural and the social sciences. Then we will be able to understand better how the formation and rise to prominence of particular concepts is linked to the uses of particular technologies and techniques, but also to the institutional dynamics of research.

Finally, on a more speculative note: is there still room for a concept of social action as a social scientific concept, or as a scientific concept at all? It is a concept with a very rich tradition, and after finishing this book I have asked myself, what place does it retain in a research landscape where the focus is shifting toward the life-behavioral domains? I remembered a conference I attended a few years ago, where one of the speakers was aiming to explain the decision making of financial traders in terms of their neurophysiological functions. Everything was there: the neurophysiological data, the regression models, the trades. After the talk, during a coffee break, the speaker in question was sharing tales from the field with other participants. It appeared—but it was not actually mentioned in the presentation that during trading subjects did a lot of other things, such as eating, drinking, chatting, and that all these impacted their physiological functions. In the end, as the speaker acknowledged, it was hard to tell how much of the increase in blood pressure was due to transactions and how much was due to a triple chocolate muffin. Small, marginal and apparently inconsequential activities could still then have consequences. In order to understand what these people did, one still had to observe them close up. This has been valid in the past and will continue to be valid.

Author's Response: Helen Longino

My thanks to both James Tabery and Alexandru Preda for their thoughtful responses to *Studying Human Behavior*. That their thoughts go in somewhat opposed directions is fortunate, as this gives me a chance both to elaborate on specific ideas in the book and to go somewhat beyond those to more general considerations about sciences of human behavior.

First to Tabery, who charges me with being a monist about pluralism. Touché? Not quite. I do allow that there are different kinds of pluralism; I also think there may be areas of science where complete and comprehensive accounts of a phenomenon may be given and areas where the kind of pluralism Mitchell and Tabery advocate (convergent pluralism, we might call it, integrative pluralism as they call it) is possible. What kind of pluralism (or monism) is appropriate for a given area of research is really a bottom-up empirical question: what kind of pluralism/monism do the relations among a set of approaches permit? So, with respect to integrative versus ineliminable pluralism about the sciences of behavior, the question Tabery's response provokes is: does the GxE or GxExN approach championed by Avshalom Caspi, Terrie Moffit, and their collaborators support integrative pluralism? I take integrative pluralism to be the view that, for some abstract category of phenomena, for example, division of labor in social insects, there may be different approaches to the study of that phenomenon, but that, for specific concretely realized subcategories, e.g., division of labor in honey bees (or a single species of honeybee) a single account that either reduces to one of the multiple approaches or constitutes an integration of approaches is possible. One can go further, and stipulate that understanding a single concrete phenomenon requires a single such account, but, assuming that ought implies can, I will leave this normative claim aside and focus on whether the GxE/GxExN approach qualifies as a single comprehensive approach to at least some well-defined behavioral phenomena, or as a single approach among others.

Tabery usefully reminds us that Caspi, Moffitt, et al., are not the originators of efforts to integrate research focused on different aspects of the complex phenomena of behavior. When exploring the prehistory of their model, it is important to remember that there is more exploration of these themes than even Tabery reports. In addition, there is lots of debate (well, certainly difference of opinion) among geneticists about the most useful way to go about understanding the role of genes in any high-level phenotype (Tabery's 2014 does discuss some of these). Some prefer genome-wide association studies (Risch and Merikangas 1996; Risch 2000) others see promise in epigenetic studies (McGowan et al. 2009; van Ijzendoorn et al. 2010) and others are committed, with Caspi, Moffit, and their collaborators, to exploring the effects of single gene variants.

What is the nature of my skepticism about GxE or GxExN? As I say in the book, the most well-worked-out models coming from the approach are models of disorders like depression or schizophrenia. But even here, the goal of a single explanation is elusive. Instead what we are offered are sketches of models of possible mechanisms or subsystems associable with a subset of cases of depression or schizophrenia, namely that subset for which an environmental stressor (or set of stressors) has been identified.

Let me stress that I have no doubt that genetically influenced biochemistry affects brain function which in turn affects various cognitively mediated performances/ actions/behaviors. That is not the question here (but note that the causal verbs, "influence" and "affect," are not deterministic, indicating either stochastic or interactive processes.). The question is whether the methods available to or utilized by the GxE approach are such as to offer a complete and comprehensive account of a given behavioral (or, as in the case of depression, temperamental/affective) phenomenon. To explain my skeptical response to this question, it is necessary to outline the method.

As described in Moffitt et al. (2005), the method starts with a measurable condition. Let us say "condition" instead of disorder. The next step explains why one might think the method restricted to disorders, as it specifies identifying an environmental pathogen that has a measurable effect on the expression or non-expression of the condition. The step after that is determining whether a gene variant moderates the effect of the pathogen on expression of the condition. If it does, we have evidence of an interaction. The effort in both these steps is to identify environmental or genetic factors that could plausibly be thought to have a causal role in expression of the condition, so to go beyond mere association. What has intrigued many other researchers and commentators is that this method can show that gene variants that, when tested alone, show no appreciable difference in expression of a condition, but when tested on populations differentiated with respect to exposure to specified environmental pathogens, do make a difference.

Because Tabery emphasizes the variety of conditions to which GxE has been applied, I will spell out the method without using the normatively laden language ("disorder," "environmental pathogen") more frequently used. The steps of the method can be diagrammed as follows. First partition the population into those with the condition C and those without C, and further partition both C and \sim C cells into those exposed to a given environmental factor EP, and those not so exposed. (Table 1). The next step is to further partition the four cells thus achieved by genotype. For simplicity's sake we assume that G1 and G2 are the only variant forms of G (Table 2).

The first partition tells us that, among those with C, 70 % were also exposed to environmental factor EP. The second partition shows us that within that 70 % G1 are represented two and one half times more than G2. This is what suggests not just

N = 1,000	Condition C N = 100	Condition $\sim C$ N = 900
EP	70	100
$\sim EP$	30	800

Table	2	Second	partition

Table 1 First partition

N = 1,000	Condition C N = 100	Condition C N = 100		Condition $\sim C$ N = 900	
EP	50	20	50	50	
$\sim EP$	15	15	400	400	
	G1	G2	G1	G2	

statistical interaction, but material interaction. But, of course these partitionings only indicate the end points of a process, not the process itself. The N that Caspi and Moffitt add is sometimes specifiable, although neither the pathway from G to N nor the pathway from EP to N, is understood. But, notice how in the focus on 50 versus 20 in the upper left quadrant, we are pointing to one possible mechanism involved in some cases, but still know nothing about the rest of the cohort. What about those exposed to EP who do not develop condition C, or those not exposed to EP who do develop condition C? Unless these questions are answered, the discovery of a GxE interaction does not constitute a comprehensive model of C, as opposed to one possible pathway to C.

My suspicion about the GxE or GxExN approach is that what it really can do, when it is applicable, is illuminate a subset of cases of a condition for which an environmental trigger can be identified and a differential representation of some gene variant found among those both exposed to the trigger and manifesting the condition. My larger suspicion is that absent specific unusual outcome conditions or unusual-specific environmental triggers, "normal" behavior and affect are the outcome of multiple complex systems with multiple redundancies functioning in a coordinated way. A single gene variant can, if protective redundancies are not in place, disrupt that functioning leading, say, to a reduction in serotonin reuptake, which may increase the vulnerability of some individuals to environmental stresses. It is also possible that gene variants could disrupt or alter "normal" functioning in a way that may enhance certain capacities, leading to exceptional performance by some individuals given exposure to certain environmental facilitators. But this is hardly a comprehensive account of whatever condition is under investigation. I have tried to steer away from the disorder and pathogen language in order to make this methodological point clear. I am not denying that mechanisms may be identifiable nor that this approach may be used to identify treatments or effective interventions for some individuals. I am claiming that neither the mechanisms the approach points to, nor the approach itself, constitute a complete and comprehensive account of a given behavioral phenomenon. The price of integration is partiality. Plurality is not eliminated.

Now to Preda. Alexandru Preda's response led me back to Max Weber, who introduced the concept of "social action" as the focal object of sociology (Weber 1950). Social action, sensu Weber, is an action in which the individual takes account of the behavior and anticipated responses of others. It has also been defined as an action to which the individual assigns meaning. The importance of social action in Weberian sociology is that social structure is the outcome of social actions undertaken by individuals in (a) society. This grounding of social phenomena in individual actions contrasts with the program of that other great founder of sociology, Emile Durkheim, for whom individual assignment of meaning or self-understanding was irrelevant to understanding social phenomena (Durkheim 1965). Such understanding was to be grounded in "social facts," which are facts about social structure. I do not want to enter into *this* debate, but it is useful to be reminded of the origin and theoretical role of "social action."

In what way is "social action" an alternative or better concept than "behavior" or even an additional facet to the kaleidoscope? Two aspects of "social action" are

woven into Preda's discussion: meaning and complexity. Preda's account of the study of financial traders nicely illustrates the role of context and reminds us of the complexity of human behavior in its natural context. In the laboratories of experimental behavioral economics the multiple factors possibly bearing on the subjects' behavior are as far as possible eliminated in order to focus on the unsullied associations of behavior and neurophysiological state/process. The study of the behavior requires reducing the complexity of the phenomenon by eliminating the contextual factors that might alter it. The trading study reported by Preda captured data that apparently reflected multiple aspects of the situation and not just the relation between recorded behavior and recorded neurophysiological data.

Although there have been some calls in psychology to incorporate significance into the definition of 'behavior,' they have not been much taken up (Bergner 2011) and the effort remains the description and explanation of behavior in purely external, "objective" fashion. In the scientific approaches surveyed in Studying Human Behavior, human behavior is treated as a natural phenomenon, continuous with non-human behavior. Indeed, the aim in classification and description is precisely to devise rubrics and indices that permit decontextualized identification and classification.¹ Significance, by contrast, is acquired relative to the details of a context. My twirling can be part of a dance performance, a game, or even a religious practice. Which it is depends on features of the context in which the twirling is performed. The significance I assign and my anticipation of others' responses, e.g., of my partner in the duet catching me or of others competing with me in endurance or of joining me in praise of the deity depends on both the context and my understanding of the context. Not only must features of the context be included to distinguish dance-performance twirling from game twirling from ritual twirling but also the agent's intention toward that context.

Understanding intentions involves interpretation, however, not measurement. The disciplines of ethnography and cultural anthropology are preeminently interpretive disciplines (save perhaps when commandeered by functionalism). They are efforts to understand the practices of a society in terms of the society's (or its members') self-understanding, not in terms of categories imposed by or of interest to the observer. Social action so conceived constitutes a different explanandum than the behavioral operationalizations of behavior genetics, neurophysiology or social-environment oriented developmental psychology. The pluralistic kaleidoscope it completes is not the explanatory one proposed in *Studying Human Behavior* but one reflecting different conceptions of what is to be explained.

One might think of these conceptions as expressing different orientations or perspectives on the phenomena. The ethnographer or cultural anthropologist takes a second person orientation to the agents and their behavior/actions. As second person, "you," is like the first person, "I," in having motives, intentions, expectations, reasons, and assigning significance to her/his actions. When I engage you, I presume you have an internal constitution of reasons, desires, beliefs, emotions, as I do. A third person orientation, to her or him or them, enables a more

¹ One of my points in *Studying Human Behavior* is the incomplete liberation from context and folk psychology achieved in the definitions of the particular behaviors whose research is the book's subject.

detached, objective relationship, such as a researcher seeks to have toward research subjects. Here, I think, are the ingredients for a kaleidoscopic view not of the *explanans*, but of the *explanandum*: still one thing, but viewed differently and subject to different kinds of explanation: interpretive—oriented to subjectively meaningful action— or empirical—oriented to objectively describable behavior. And among both these broad categories of explanation one will find multiple approaches. Of course, one of the debates stimulated by actual and possible advances in brain research is whether the interpretive-subjective is an epiphenomenal illusion to be displaced by the empirical-objective (see, for example, Churchland 2002). This is not the place to enter into *that* debate, either, but the invocation of social action does call it up.

A few last points. I do not want to be an obscurantist about consciousness, action, or the will. From a scientific point of view, I do not think they have special features not shared with other natural phenomena. Rather, as do some other natural phenomena, they pose challenges of complexity, scale, and perspective. And, I do think the intentional actions of individuals can be studied scientifically/objectively when treated as aggregates in a population. This is what opinion polling is all about. But here we take a third person stance, using stated opinions as data to be integrated with other data to support conclusions about a population not as data/information to help us understand the individual. Finally, the population level or ecological approach adumbrated in *Studying Human Behavior* will encompass all behaviors of a given sort, intentional or not, and ask questions about them *en masse*, questions about their frequency, distribution, and the association of these with other population variables. This, too, involves changing perspective from first or second person to third.

To conclude, Tabery's and Preda's comments, pulling me in opposite directions, together underscore both the complexity of our shared subject matter and its proximity to difficult theoretical and philosophical debates. I am grateful to both for the opportunity to expand and reflect on the analysis offered in *Studying Human Behavior*.

References

Bergner, Raymond M. 2011. What is behavior? And so what? New Ideas in Psychology 29: 147–155.

- Brown, George W. 1987. Social factors and the development and course of depressive disorder in women: a review of a research programme. *British Journal of Social Work* 17: 615–634.
- Caspi, Avshalom, et al. 2003. Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science* 301: 386–389.
- Caspi, Avshalom, et al. 2007. Moderation of breastfeeding effects on the IQ by genetic variation in fatty acid metabolism. *Proceedings of the National Academy of Sciences* 104: 18860–18865.
- Caspi, Avshalom, et al. 2010. Genetic sensitivity to the environment: the case of the serotonin transporter gene and its implications for studying complex diseases and traits. *American Journal of Psychiatry* 167: 509–527.

Churchland, Patricia. 2002. BrainWise: studies in neurophilosophy. Cambridge MA: The MIT Press.

Durkheim, Emile. 1965. *The rules of sociological method*. Trans. W.D. Halls (first published Paris 1895). Glencoe IL: Free Press.

- Hariri, Ahmad R., et al. 2002. Serotonin transporter genetic variation and the response of the human amygdala. Science 297: 400–403.
- Levitt, Steven, and Stephen J. Dubner. 2009. Freakonomics: a rogue economist explores the hidden side of everything. New York: William Morrow.
- Lesch, Klaus-Peter, et al. 1996. Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. *Science* 274: 1527–1531.
- Loehlin, John C. 1989. Partitioning environmental and genetic contributions to behavioral development. *American Psychologist* 44: 1285–1292.
- McGowan, Patrick O., Aya Sasaki, Ana C. D'Alessio, Sergiy Dymov, Benoit Labonté, Moshe Szyf, Gustavo Turecki, and Michael J. Meaney. 2009. Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. *Nature Neuroscience* 12: 342–348.
- Meltzer, Herbert Y. 1990. Role of serotonin in depression. Annals of the New York Academy of the Sciences 600: 486–500.
- Meltzer, H.Y., and R.C. Arora. 1988. Genetic control of serotonin uptake in blood platelets: a twin study. Psychiatry Research 24: 263–269.
- Mitchell, Sandra D. 2003. Biological complexity and integrative pluralism. Cambridge: Cambridge University Press.
- Moffitt, Terrie, Avshalom Caspi, and Michael Rutter. 2005. Strategy for investigating interactions between measured genes and measured environments. Archives of General Psychiatry 62: 473–481.
- Pezawas, Lukas, et al. 2005. 5-HTTLPR polymorphism impacts human cingulate-amygdala interactions: a genetic susceptibility mechanism for depression. *Nature Neuroscience* 8: 828–834.
- Risch, Neil. 2000. Searching for genetic determinants in the new millennium. Nature 405: 847-856.
- Risch, Neil, and Kathleen Merikangas. 1996. The future of genetic studies of complex human diseases. Science 273(13): 1516–1517.
- Tabery, James. 2014. *Beyond versus: the struggle to understand the interaction of nature and nurture.* Cambridge: The MIT Press.
- van Ijzendoorn, Marinus H., Kristin Caspers, Marian J. Bakermans-Kranenburg, Steven R.H. Beach, and Robert Philibert. 2010. Methylation matters: interaction between methylation density and serotonin transporter genotype predicts unresolved loss or trauma. *Biological Psychiatry* 2(68): 405–407.
- Venkatesh, Sudhir. 2008. Gang leader for a day: a rogue sociologist takes to the streets. New York: Penguin.
- Weber, Max. 1950. *The Methodology of the Social Sciences*. In *An anthology of Weber's writings*, ed. E.A. Shils and H.A. French. New York: Macmillan.