Review of *Studying Human Behavior*

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Helen Longino, ***Studying Human Behavior: How Scientists Investigate Aggression and Sexuality***. Chicago: University of Chicago Press (2013), 256 pp., $75.00 (cloth).

In "The Pluralist Stance," Stephen Kellert, Helen Longino, and C. Kenneth Waters (2006) contrast *monism* about the sciences with *pluralism*. The core claim of monism as they define it is that the sciences are aiming to give a single complete, comprehensive account of the natural world; this requires that there be methods that could yield such an account. Pluralism, however, is simply the view that whether such a single account could exist for any field is an empirical matter, and the existence of methods contributing to more than one model is not necessarily problematic. More strongly pluralist views of the sciences are, however, possible, and in *Studying Human Behavior* Helen Longino takes just such a view of a series of approaches used to study the (developmental) causes of human behavior, in particular aggression and homosexuality: she argues that these approaches could not contribute to such a single complete account and that it is not clear that such an account is even desirable. The approaches in question are quantitative behavioral genetics (QBG), molecular behavioral genetics (MBG), social environmental approaches (SEA), neurobiological approaches, and a variety of integrative approaches such as genes × environment × neurology (G × E × N), multifactorial path analysis, and developmental systems theory: I will refer to these as the behavioral biological approaches (BBAs). The first part of *Studying Human Behavior* (chaps. 2–7) is a very interesting, well-researched discussion of major work, research questions, methods, and relationships between the BBAs.

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Longino's main argument for taking her strongly pluralist stance toward the BBAs is given in the second part of the book. Her reason is that the results of these various approaches are incommensurable: this means there is no way to combine the results of the various BBAs into a single complete account of the causes of behavior. She identifies two types of incommensurability. First, the way that each approach "parses the causal space" is different (15, 126, 203). The different causes of behavior (e.g., genes, epigenetic mechanisms, various environmental causes) get divided up by the different fields in different, overlapping "chunks." For example, SEA would tend to parse the shared uterine environment of twins into the "genetic" (i.e., ignored) portion of the causal space; QBG might treat it as part of the "environment" (127–29, 134). But this means one cannot combine results from QBG and SEA into a single model. Confirming such a combined model, according to Longino (203–4), would require the scientist to check the model applied in particular cases. This in turn would require that one could measure the effects of varying one parameter while holding the rest steady. But if these parameters overlap, then one cannot vary one parameter independently of the others.

Second, Longino argues (primarily in chap. 9) that just as there is no common way to parse causes among the BBAs, there is no common definition of the behaviors these causes are supposed to generate. Each of the BBAs defines behavior (and particular behaviors) in different ways relevant to the concerns and commitments of the project; each operationalizes those definitions in ways relevant to their particular measurement methods—for example, surveys, databases, and so on (5, 8, 151–77, 207–8). If BBAs do not agree on how to categorize the behavior they study, then the results they produce about the causes of "aggression" or "homosexuality" do not even refer to the same kind of behavior.

The consequence of all this incommensurability is that the BBAs generate (or are in the process of generating) multiple independent models. For example, there are models at both the population and the individual levels (117–21, 135–36). There are nonoverlapping partial accounts of what is happening in populations (117–21). There are narrow descriptions of individual-level mechanisms limited to single disorders or variants (116–17, 135). Longino argues at some length that these various partial models are not just acceptable but may be desirable in some circumstances (146–48). Indeed, she argues in chapter 10 that models generated by the BBAs are used in multiple different ways by different scientific and academic communities: this suggests that they have a variety of different functions.

So has Longino made her case? I am not totally convinced, despite my own pluralist sympathies. While she might be right that the BBAs are offering more than one account of behavior (e.g., there might be accounts at different levels of explanation such as populations vs. individuals), I want to take issue with the view that the results of the BBAs cannot be combined

into any complete account (i.e., that all of these models are partial models). The main problem with Longino's argument is that she is not clear on what type of single complete account she is rejecting or what "combining" the results might come to. While Longino is not required to accept the claim that such an account could exist, her argument that it could not exist does require that she be precise about what she is rejecting. In the discussion on 203–5, for example, this seems to be an additive model, one that accounts for different effects of various causal factors on behavioral variation (204). However, this sort of account would only really be appropriate at the population level (behavioral variation is a population-level property). Many of the BBAs are not offering population-level explanations (at least, directly—but the most obvious exception is QBG) and instead are trying to identify the entities, events, and processes involved in the development of the behavior of individuals.[1] Moreover, the "complete account" the BBAs are contributing to at the individual level is probably best understood as a description of the developmental mechanisms generating particular behaviors. A mechanistic account does not describe how causal factors additively contribute to variation but instead describes the entities and activities (e.g., in our case, coding genes, regulatory sequences, neural activation patterns, transcription, experiences of abuse) that generate a phenomenon of interest (in this case, a behavior; Machamer, Lindley, and Craver 2000). Combining the results of the BBAs into a mechanistic account would require showing how the putative causes each identifies are connected into a system that generates the proposed behavior.

However, if the BBAs "complete account" at the individual level is meant to be a mechanistic explanation, this alone will not avoid Longino's problem of how such a model would be constructed—this is because most of the BBAs are unable to precisely identify the components of these mechanisms or the exact causal relations in which those components stand in the developmental system. Instead, at best they suggest what form those causes might take or where they might be found. For example, they show that particular genes might be involved in the development of aggression or that abuse might be implicated in the development of psychopathy, and so on: Longino notes that this kind of hedging language is used by scientists but does not explain why it is used (104).

What is clear, then, is that if any sort of complete account of the development of these behaviors is to be given, the BBAs cannot provide such an account on their own: the process of actually successfully describing these mechanisms will require any work in the BBAs to be combined with work

1. I should note that some of the BBAs (in particular MBG and SEA and derivative projects such as G × E × N) appeal to population-level studies as evidence for putative causes; this does not mean they are offering population-level explanations.

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from other fields. In integrative BBAs such as G × E × N, which are starting to construct such mechanistic explanations, the sources of the additional information are the lower-level sciences such as molecular biology and developmental and cognitive neuroscience (Caspi and Moffitt 2006). These sciences could provide additional causal details that would (1) identify the details of the mechanisms that involve the causes identified by the BBAs (e.g., how an identified gene contributes to the development of particular brain systems) and (2) determine that the causes proposed by the BBAs could therefore do the work they are proposed to.[2]

Longino does seem to be happy with the idea that to a degree such kinds of mechanistic description are possible; she clearly accepts the results of mechanistic integrative projects like G × E × N (93–99, 111–12, 205). However, she argues that the actual attempts to describe mechanisms result in accounts that become narrower; that is, they end up being about single disorders or variants (95, 99, 112). In other words, they are still partial accounts of the overall system. Longino seems to be moving the goalposts here, since the initial interest she had was in work on particular behavioral variants (aggression and homosexuality), and the G × E × N approach seems to be capable of just that. But let us accept for now that the "complete account" any monist or weak pluralist wants is a complete account of the developmental processes that produce all behavior. It is still not clear why these stories about how individual variants develop cannot contribute to a complete account of the "normal" mechanism, since the individual accounts of disorders have to describe (at least part of) the normal mechanism in order to explain the variant case (e.g., variation in monoamine oxidase A levels changing normal levels of serotonin and resulting in a vulnerability to psychopathy). What is more, the descriptions of individual entities, processes, and properties making up these mechanism accounts are part of a common ontology derived from independent lower-level science work. This means that the descriptions of the mechanisms underlying the specific disorders should be thoroughly commensurable with each other and could be integrated.

To be fair to Longino, developmental neuroscience and psychology are still at very early stages of development: it still might turn out to be impossible to give a complete individual-level account of the development of these behaviors. Unfortunately, Longino has chosen to take the strong view that the nature of the current sciences rules this out, and it seems to me that this is simply not true. This does not, however, rule out a weaker pluralism: for

2. It might also allow scientists to bootstrap between giving a definition of the behavior they are looking for and a description of the mechanism that produces it: presumably such a process would start with a rougher definition of behavior, use that to identify an underlying mechanism, and then use the proper output of the mechanism to refine the behavioral description (this might be a way around the problem identified in Longino's second argument for incommensurability).

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example, it does not rule out this "individual-level" account coexisting with a separate "population-level" account and maybe other partial accounts used for simpler, temporary purposes (e.g., MBG contributing to ruling out the possibility that homosexuality is a "lifestyle choice").

REFERENCES

Caspi, A., and T. E. Moffitt. 2006. "Gene-Environment Interactions in Psychiatry: Joining Forces with Neuroscience." *Nature Reviews Neuroscience* 7:583–90.

Kellert, S., H. Longino, and C. K. Waters. 2006. "The Pluralist Stance." In *Scientific Pluralism*, ed. S. Kellert, H. Longino, and C. K. Waters, vii–xxix. Minneapolis: University of Minnesota Press.

Machamer, P., D. Lindley, and C. F. Craver. 2000. "Thinking about Mechanisms." *Philosophy of Science* 67:1–25.