Chapter 2
Knowledge for What? Monist, Pluralist, Pragmatist Approaches to the Sciences of Behavior[1]

Helen Longino

> *With the greatest of hubris, quantitative behavior genetics strives to traverse the molecular and psychological levels in one grand inferential leap.*
>
> (Wahlsten & Gottlieb, 1997)

> *Complex developmental processes, …, are not amenable to any microanalysis we currently know how to conduct. … [T]hus mechanistic science is unlikely to yield useful information about complex behavioral problems, ….*
>
> (Scarr, 1995)

2.1

I have been conducting a comparative epistemological and social analysis of research approaches in the sciences of human behavior. In this study, which involves analysis of research reports in journals and at seminars and conferences, meta-analyses, polemical exchanges among the researchers, and public media representations of the research and its implications, I have looked primarily at what might be dubbed, after Ernst Mayr's distinction, proximate forms of explanation. That is, I

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have excluded evolutionary approaches to behavior.[2] Among these proximate forms of explanation, I have investigated both single factor approaches – genetic, neurobiological, social-environment – and integrative approaches – what is known as developmental systems theory as well as a more limited approach dubbed the GxExN approach. In this essay I update arguments I have elsewhere offered for adopting a pluralist stance towards this multiplicity of approaches, but further argue that pluralism alone leaves us without a way of making use of the knowledge generated by the different approaches. Pluralism must be supplemented by a form of pragmatism that attends to what kinds of question a given approach can answer together with what kinds of question our practical experience makes salient.

2.2

Behavior genetics divides into quantitative behavior genetics (also referred to as classical behavior genetics) and molecular behavior genetics, the former drawing on methods of population genetics, the latter drawing on molecular biology. Both are interested in identifying genetic contributions to behavior. Quantitative behavior genetics attempts to correlate variation in the expression of some trait in a population with genetic variation in that population. It is interested in the question: how much of a given behavior of interest B is heritable, which translates into the question: how much of the difference in expression of B among individuals in a population is correlated with genetic difference in that population? The methods involve finding behavioral correlations and variation in correlations among biologically related individuals, and trying to separate genetic from environmental influence by studying adoptees and twins separated at birth or shortly thereafter. For example, a twin study examining a broad range of behaviors examined concordance in measures of antisocial behavior in 331 twin pairs raised together and 71 reared apart. Behaviors were identified through a self-report questionnaire (the MMPI) and included two sets of questions measuring antisocial or aggressive behavior. The concordance in answers among the twins reared apart supported a heritability estimate of .8.[3] Quantitative behavior geneticists extend their methods with a variety of techniques, including longitudinal analyses that address the question about the stability or mutability of genetic influence on a given behavior over time.

One of the values claimed for quantitative behavior genetics is that when some genetic influence is suggested by family concordances or correlations, the behavior becomes a candidate for analysis by molecular genetics whose aim is to find associations between phenotypic traits and sets of specific genes or gene regions.

[2]Proximate and ultimate (or evolutionary) explanations are answers to different kinds of question (ontogenetic and phylogenetic, respectively) and so not susceptible to the kind of comparative analysis I am conducting.

[3]Tellegen, et al. (1988). Twin study heritability results included in a meta-analysis performed by Mason and Frick (1994) range from 0 to .84.

The questions asked by molecular geneticists concern whether genetic markers, which are multi-allelic gene regions whose frequency can be observed relatively readily, can be associated with the incidence of B in a given pedigree or family lineage. The finding of markers associable with phenotypic traits suggests that a gene in the vicinity of the marker is causally influencing the incidence of the trait. In the early 1990s, 14 male volunteers of a Dutch family, all of whom experienced episodes of aggressive behavior, were found also to share allelic variation on a region of the X chromosome coding for the enzyme monoamine oxidase (or MAOA).[4] This enzyme is involved in the metabolic cycle of serotonin. The Brunner study stimulated much concern over possible genetic intervention and genetic discrimination. This has subsided and studies of the roles of irregularities of MAOA related genes and of other genes related to aspects of serotonin metabolism have proceeded apace. The investigative technologies available for studying the genome are advancing rapidly, and such techniques as Genome-Wide Association Study raise the hope that more gene regions can be identified.

Neurophysiology and neuroanatomy are interested in identifying the role neural structures and processes play in behavior.[5] One intensely studied aspect of neurophysiology has been the serotonergic system: the set of processes involved in the diffusion and reuptake of the neurotransmitter, serotonin. Variation in serotonin concentrations, in number and distribution of serotonin receptors, and in serotonin reuptake has been associated with a number of psychological/behavioral phenomena from depression to suicidality to aggression. As is often the case with physiological research, after initial findings of a relationship of some substance or process to a higher level trait, these lines of investigation initially created more puzzles than they solved. Research in the 1990s sought to elaborate the mechanisms of involvement and separate out possible physiological confounders. Was the culprit decreased serotonin production or diminished uptake of serotonin? To address this question, one study of ten subjects and five controls by Emil Coccaro and colleagues investigated the possible involvement of serotonin receptors in the causal pathway.[6] Researchers administered a serotonin antagonist that would block the serotonin receptors to the subjects but not to the controls. They then administered an agent, buspirone, that physiologically mimics serotonin. Receptor sensitivity was assessed by measuring prolactin levels before and after administration of buspirone. Prolactin is released when serotonin or one of its agonists bind to serotonin receptors. Lower levels of prolactin have been associated with higher levels of aggression/irritability. Prolactin levels in subjects whose receptors were blocked were lower in relation to individual baselines than in controls. This experiment implicates receptor function rather than serotonin production in serotonin's behavioral effects.

[4]Brunner, et al. (1993). Five members of the family exhibited extreme levels of violence, while nine others exhibited more moderate, but still higher levels of violence.

[5]I deliberately use the broad locution, "play a role in", and avoid causal locutions such as "produce" as there are very different kinds of causal relation that can be investigated. And in the case of neurophysiology, there is a very live question as to whether what is investigated is causation or constitution.

[6]Coccaro, Gabriel, and Siever (1990).

Other kinds of question addressed in this research approach include whether the neural processes associated with a behavior are distributed or local,[7] with what other neural and organic processes the processes associated with the behavior interact, and so on. Neurobiological research also includes the use of the various neuroimaging techniques, as well as autopsy, to identify neural and brain structures involves in various behaviors.

Social/environmental approaches seek to understand the role that environmental and other exogenous factors play in a given behavior. They may investigate the role gross or macro-level social variables (social class, ethnic, racial, and cultural identity, urban/suburban, immigrant/native, etc.) play in the expression/frequency of a behavior of interest. They may investigate the role micro-level variables such as family, school, peers, media exposure, play in the expression of the behavior. Does one of these predominate in its expression? Other research questions include: Do micro- and macro- level variables interact in the expression of the behavior? If so, how? How do differences within a family influence the expression of B by its members? They may employ large databases such as are made available from courts and other governmental institutions, or may conduct more fine-grained laboratory observation of behavior.

In one study, Cathy Widom and colleagues employ the first strategy in efforts to link adolescent and adult violent and antisocial behavior to abuse in childhood. In one of their studies, they compared the records of 416 adults with histories of physical and sexual abuse in childhood with those of a control group of 283 adults with no documented history of abuse.[8] The rate of antisocial personality diagnosis in the group with histories of abuse was 13.5% as compared with 7.1% in the control group. The researchers conclude from this (and other studies in similar vein) that experience of abuse as a child is a significant causal factor in adult violence, and that special prevention efforts directed towards victims of abuse could reduce later criminal behavior.

In a study conducted at a finer level of granularity, researchers sought to correlate familial interaction patterns with long-term disruptive behavior in eight and nine year old boys.[9] The boys chosen for the study were identified by teachers who completed Social Behavior Questionnaires on their students. Interactions in 44 families were studied by observing the parents and child in question engaged in joint tasks in the researchers' laboratory. Observers used checklists in rating dyadic interactions between father and child, mother and child, and between the parents. Researchers found that negative behaviors (such as verbal abuse or attacks) and positive behaviors (such as endearments) in the parent-child dyads were not reciprocal, but that negative behavior of one parent toward the boy was correlated with negative behavior on his part toward the other parent. In addition, negative behavior of boys toward their mother was correlated with fathers' negative

[7] A distributed process being one that involves neuronal structures throughout the brain, while local ones are specific to a single region or even a single neuron.

[8] Luntz and Widom (1994).

[9] Lavigueur, Tremblay, and Saucier (1995).

attitudes toward their female spouses. The researchers speculate that coaching the parents in alternative styles of interaction could reduce the chances that their child's disruptive behavior will later develop into more serious anti-social behavior.

Most researchers accept that observable behaviors are outcomes of interactions among all these factors. The points of contention concern not whether any of these factors are real or contribute, in some way, to a given behavior, but 1) which predominate, 2) how to quantify their relative contributions to behavioral outcomes and 3) how to represent the interactions among them. Hence, researchers don't need an argument that one or another factor plays a role, but rather a way of measuring and calculating their respective roles. Competition among / uncertainty about the approaches concerns whether any one has the tools required to calculate values for the factors stressed by the others. The debates, then, are less about ontology than about methodology: given that all the factors identified in the various approaches play some role, which approach is likely to be most informative about the etiology of behavior?

2.3

All approaches must assume that the traits under investigation are well-defined. By this I mean that the traits have clear criteria of identification, of operationalization, and of measurement. This may seem a trivial requirement, but I have elsewhere shown that this assumption is not satisfied in the case of aggression or of sexual orientation, two families of behavior that have received extensive study.[10] Because the research interest consists in understanding relatively enduring traits, the object of investigation is dispositions to behave in certain ways in certain conditions, rather than episodes of behavior. Episodes are taken to be indicative of dispositions.

More to the point for the present analysis, all select from a range of possible types of cause. This range is what I call the potential causal space, or space of potential causes, and it can be displayed in a grid, as in Fig. 2.1.

Genotype 1 [allele pairs]	Genotype 2 [whole genome]	Intrauterine environment	Physiology [hormone secretory patterns; neurotransmitter metabolism] Anatomy [brain structure]	Non-shared environment [birth order; differential parental attention; peers]	Shared (intra-family) environment [parental attitudes re discipline; communication styles; abusive/nonabusive]	Socio-Economic Status [parental income; level of education; race/ethnicity]

Fig. 2.1 Undifferentiated causal space

The specificity of assumptions informing and shaping the individual research approaches and the methods of observation and measurement they employ means that this range or space of potential causes, all members of which are implicitly agreed to play some role, is only partially activated in any given research approach.

[10] Longino (2001) and forthcoming.

These assumptions, it should be stressed, are not explicit, but rather assumptions required to confer evidential import on the data.

Assumptions of the behavior genetic approach include the following:

1. The causal contributions of genes to the inculcation of a behavioral disposition are separable from other causal influences on the inculcation of that disposition, that is, given that there is interaction between genetic and non-genetic factors, it is possible to distinguish their respective contributions to the variation in the disposition to exhibit some particular behavior.
2. Conversely, the effects of genes are separable from effects of other factors, that is, it is possible to distinguish at the phenotypic level what, or how much, of a trait is produced by genetic factors and how much by non-genetic.
3. Heritability is an appropriate measure of that genetic contribution, that is, appropriately designed studies of variation in the expression of behaviors in stipulated populations, will reveal the genetic contribution to variation in those behaviors.[11]

Other assumptions, built into the methods of heritability studies (twin and adoption research that attempts to separate similarity of genetic structure from similarity of rearing environment), include:

4. The available causal space can be represented as including genetic and environmental causes (with a noise factor built in to the equation).
5. The environment is distinguishable into shared and non-shared environment, thus accounting for variation accounted for neither by genetic factors or by shared environmental factors.

These assumptions mean that the causal space open to investigation by the methods of classical behavior genetics takes the form of Fig. 2.2:

Diagonal lines = active space (although in principle could include features of shared (intra-family) environment, in practice these are not taken into account or are subsumed under the SES categories)

Solid black = inactive space (empty: either randomly distributed or effect of genotype)

Fig. 2.2 The causal space for behavior genetics

[11] There is a certain amount of equivocation in the representation of conclusions from heritability studies, a slide from thinking about the genetic contribution to *difference in a population* in the expression of a trait to expression of a trait *simpliciter*.

Molecular geneticists first identify a population both sharing a trait and likely (by pedigree, or familial, analysis) to share genetic configurations. They then, using additional hints provided by the pedigree analysis, seek evidence of shared allelic variation. Assumptions of this approach include:

1. The base rate of the trait in the general population is both determinable and high or low enough to establish significance of the allelic variation correlated with trait variation in the sample.
2. The sample size in any particular study is sufficient for the detection of relevant allelic variation.
3. The causal space of interest is the variety of possible alleles and/or the whole genome.

This yields the following selection (Fig. 2.3) from the grid:

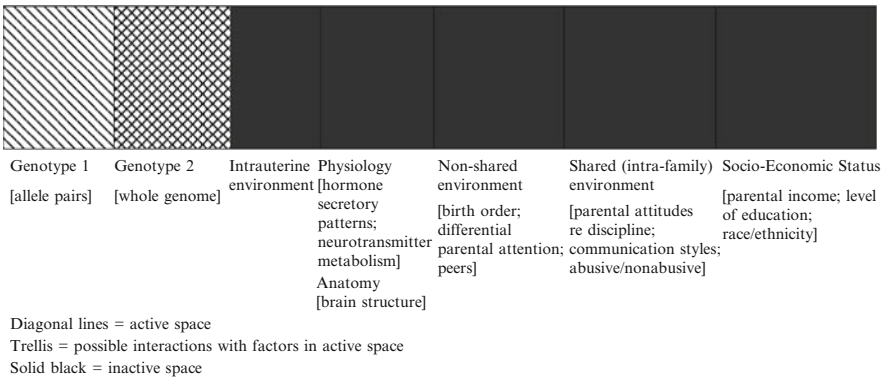

Fig. 2.3 The causal space for molecular behavior genetics

Social-environment researchers are interested in identifying the factors in individuals' environments that incline them towards one behavioral pattern rather than another. Their assumptions include:

1. Social and familial factors are causally independent of the subjects whose behavior is the object of study and for whom they constitute an environment.
2. Subjects are sufficiently endogenously uniform or genetic variation among subjects is randomly distributed and averages out in the population enabling variation in behavior to be correlated with variation in environment.
3. The causal space of interest is the variety of environmental factors that can impinge on behavior and the development of dispositions.

This assumption yields this quite different selection (Fig. 2.4) from the grid:

Fig. 2.4 The causal space for social/environmental approaches

Neurobiological approaches also require assumptions related to the investigative methodologies they have at their disposal. These yield Fig. 2.5 and include:

1. Brain areas showing greater glucose metabolism during a particular thought process are causally (or constitutively) involved in that thought process.
2. Anatomical correlates of behaviors are functionally related to the behaviors with which they are correlated.
3. The development of these anatomical correlates preceded rather than followed the relevant behaviors.
4. The causal space of interest is structures and processes in the brain and nervous system.

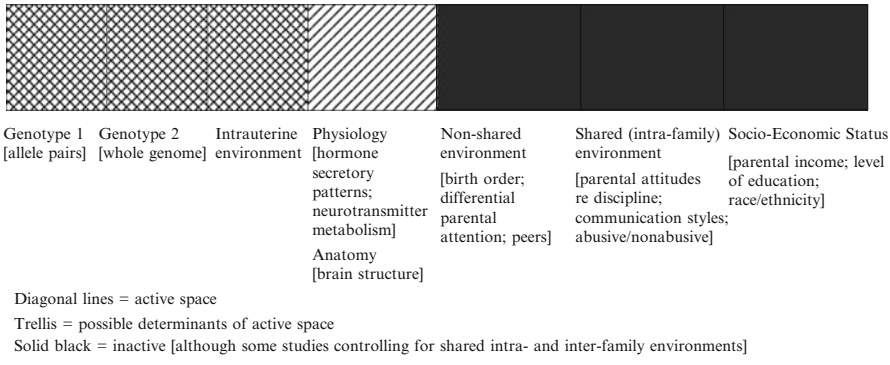

Fig. 2.5 The causal space for physiological and anatomical approaches

As the grids illustrate, each approach effectively situates itself in a different causal universe, making comparative assessment impossible. Two kinds of measurement are in play. One is measurement of the phenomenon to be accounted for (the "explanandum" or the "dependent variable"): a particular behavior pattern/disposition or variation in a behavior pattern. Here the approaches use similar measurement techniques. The other kind of measurement is of the factors an approach investigates as accounting for the phenomenon to be explained, the "explanans" or "independent variable". Measurements of the same factor being treated as explanans or independent variable conducted under one assumption concerning the structure of the causal space need not be consistent across approaches. From a god's eye point of view we may see the whole space, but if immersed in research, factors that are unmeasurable within a given approach may exert an influence that the measurement strategies either fail to pick up or attribute to different categories. Uterine factors, for example, will get classified as environmental factors under a genetic approach that focuses on biological relatedness or genetic similarity. Under an environmental approach that measures relevant differences in the social environment they do not appear at all, fading into the undifferentiated biological background. And both genetic and environmental approaches can say of the other that it fails to pick up causal relations identifiable by the one. Given that all acknowledge the interactivity of multiple causal factors in the inculcation of behavioral dispositions, a more comprehensive approach looks more promising. But here we encounter different difficulties.

Developmental systems theory (DST) is the name given to a bold set of claims about organismic development, including the development of behavioral dispositions. It has set itself up as a challenger to orthodox evolutionary theory as well as to developmental genetics.[12] The unit of evolution and of development is the developmental system, a set of complexly interacting factors whose effects coincide in the individual organism, but are not wholly contained within its skin. These include, for example, the environment of rearing and aspects of the system of nurturance of newborns and infants typical of any given species. The developmental system is not just the individual organism but the organism in its environment. The questions typical of this approach include: how does a given behavior B come to be expressed in individuals? what developmental trajectories (that is, sequence of changes in the developmental system) can be identified that culminate in B? is the disposition to B canalized? if so, how? at what levels of organismic integration and organization do the causal/developmental processes relevant to B occur (at the genetic level? at the cellular level? organic? environmental? some combination of these?)? how do complexity of organization and specialization of function develop in the individual organism? given that different types of causal factor are not separable, how can intra-level and inter-level interactions be studied?

[12]Primary expositors of Developmental Systems Theory have been Susan Oyama and the late Gilbert Gottlieb. See Oyama (1985); Wahlsten and Gottlieb (1997); Gottlieb (2001).

The assumptions of this approach include:

1. The interactivity of causes means that separation of causes is never possible.
2. The only interesting biological question is a developmental question.
3. Methods to support the central claim about the parity and interaction of causal factors will be found.
4. The unit of analysis must be the developmental system.

Given assumption 1), the causal space of DST includes all the types of factor, i.e. the entire grid, and assumption 4), what changes is not really a single property or propensity of an organism but the entire system configuration. In contrast with the preceding approaches, the entire set of interacting factors, both cause and effect, belong to the same universe and are distinguished as one stage of the system from another stage of the system represented in Fig. 2.6. A more complete representation would show how each type of factor can affect each other type of factor and affect how each other type affects higher level states of the organism.

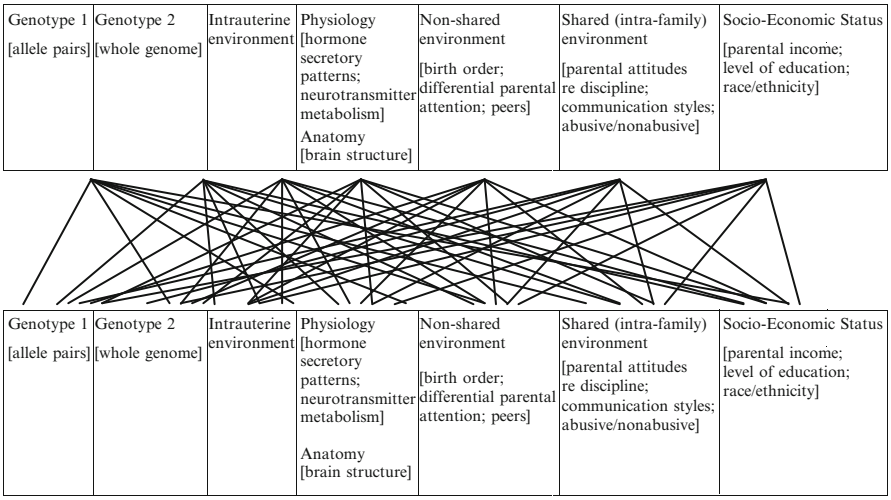

Fig. 2.6 A partial representation of the causal relations posited by Developmental Systems Theory

There is probably some sense in which this, or something like it, is the correct picture. Organisms are complex objects, and organisms in environments, even more complex. But this is not a parsing of the causal space that lends itself to empirical investigation. Furthermore, in order to be evaluated empirically in relation to any of the single factor approaches, the values of all factors and the strength of their interactions and mutual modifications would have to simultaneously measured. Even if one could construct computer simulations showing how a hypothetical system might work, an empirical determination exceeds the capabilities of present measuring systems. Thus, even if this is a correct picture, we are not entitled to claim so on the basis of empirical evidence. Empirical research does demonstrate the inadequacy of

any single approach, but this is not the same as demonstrating the adequacy of this particular representation of the causal relations.

Recently a more restricted integrationist approach has garnered a great deal of attention. The team of Avshalom Caspi and Terri Moffitt and their collaborators have worked out a specific model representing the interaction of genes and environmental factors in the etiology of specific behavioral and psychological disorders.[13] Their model posits a neural substrate for any given disorder that is acted on by both genes and environmental stimuli. The research questions of this approach include: For some specific psychiatric disorder D_P, what is the neurological substrate N of psychiatric disorder D_P? What is the specific disorder D_N of N underlying D_P? How do G and E interact in affecting N to induce D_N? The empirical information on which the model is based consists of both behavior/psychiatric genetic research showing some correlation between D_P (e.g. depression) and some allelic configuration and environmental research showing some correlation between D_P and exposure to some environmental stressor (the death of a spouse). What Caspi, Moffitt, and collaborators have done is to find that individuals characterized by overlaps (both the allele and the environmental stressor) show a much higher incidence of the particular disorder or problematic behavior, than individuals characterized by one factor alone. This, and the assumption of neural involvement, leads them to posit the following model (Fig. 2.7):

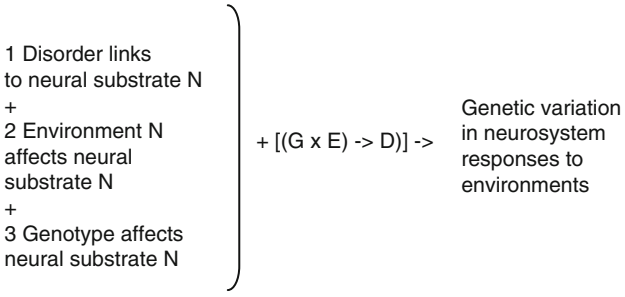

Fig. 2.7 GxExN model, modified from Caspi and Moffitt 2006, p. 585

The hope is that a specific psychiatric disorder can be linked to some specific neurobiological deficit or disorder, and that the neurobiological disorder can be linked to a genetic configuration. The neurobiological contribution will be identified by some kind of triangulation involving genes (identified through heritability and linkage studies) and environments studiable through socio-environment methods.

[13] Caspi, Sugden, and Moffitt (2003); Caspi and Moffitt (2006). About nine months after this talk was given in Hannover, Neil Risch, Kathleen Merikangas and colleagues published a meta-analysis casting doubt on the gene-depression connection that was one of the main empirical supports for the Caspi and Moffitt integrationist approach (Risch, Herrell, Lehner, et al. 2009).

Application of the model assumes that genes moderate the effect of environmental pathogens (their terminology) on disorder (i.e. that the higher frequencies in the overlap of genetic configuration and environmental stressor is accounted for by a genetically influenced sensitivity to environmental stressors), that experimental neuroscience can specify the proximal role of nervous system reactivity in the gene–environment interaction (i.e. will be able to identify the nature of increased sensitivity) and that it is possible to overcome the challenge of small sample sizes (through, for example, idealizations and analogues).

With these assumptions the potential causal space is somewhat reconfigured as in Fig. 2.8:

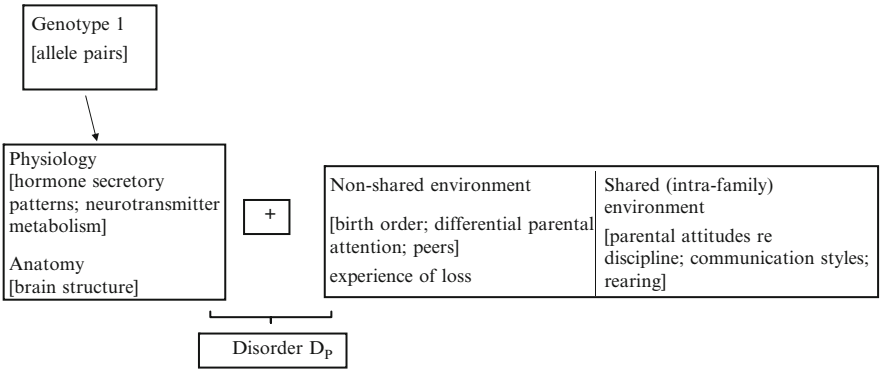

Fig. 2.8 Causal space for GxExN. Only a selection of the potential factors is studied, and they interact in producing the particular disorder

2.4

Analyzing the causal presuppositions and methods of these approaches, then, reveals that each operates in a distinct causal universe. By "distinct causal universe," I don't mean separable, ontological distinct spheres of causality, but conceptually constructed spheres of investigation. Philosophy offers several ways to respond to such a situation. Let us, for ease of consideration, limit ourselves to an epistemological response. Monism, as an epistemological view, holds that there is one, correct, comprehensive account and that it is possible to engage in comparative evaluation of alternatives in order to identify which it is. Inquiry ought to be directed to finding that one correct account.[14] Pluralism holds that given any given set of alternative accounts of a phenomenon, while some may well be false or deficient, it is nevertheless possible that there are multiple correct accounts, that none should be expected to be

[14]For more discussion of monism and pluralism see Longino (2002.pp. 93-95, 175-202) and Kellert, Longino, and Waters (2006).

comprehensive, and that it is possible to engage in intra-approach comparative evaluation, but not in inter-approach comparative evaluation (among approaches that meet some minimal empirical requirement).[15] Pragmatism suggests that alternative approaches should be judged in relation to practical goals of action with respect to the objects of the research in question. Each of these has advantages and disadvantages. In the end, I think some combination of pluralism and pragmatism offers a way of treating the variety of explanatory approaches that acknowledges the contribution each makes to the overall goal of understanding behavior.

Monism honors the impulse to unity and comprehensiveness that seems to drive many researchers, especially theoretical researchers. It makes for a relatively straightforward epistemology (true or false, correct or incorrect), and it makes sense of the debates among proponents of different and incompatible approaches to the same phenomenon or class of phenomena.[16] However, it presupposes that the data that would be used to adjudicate among approaches can, at least in principle, be completely and univocally described. In the case at hand, one has to ask: is research focused on one parsing of the causal space adequate to assign values to elements in the others? I hope the above illustrations of the causal spaces presupposed by the single factor approaches suffice to give a negative answer to this question. But, one might then suppose that a different parsing, indeed, one that includes all relevant factors should do better. Of the two integrationist approaches, however, one, the DST approach, is empirically intractable, while the other is limited in its scope to disorders, not to behavior generally. Monism, pace the debates swirling in research and philosophical circles about nature vs. nurture, requires conditions not satisfiable by the approaches currently practiced. This is not to say that some approach in the future might satisfy the conditions. But the problem with monism is that it legitimates forms of argument directed to elimination of all but one of a set of contesting approaches any time such a set exists.

The pluralist is more impressed by the (apparent) fact that each of the approaches has generated productive and useful research. Single factor and integrationist approaches can muster evidential support for their claims. The pluralist will propose that our task as philosophers is not to participate in debates about which of these approaches is the correct one, but to understand and help to articulate their scope, their evidential requirements, and their limitations. But pluralism has different problems: What's the sense in which each is correct? I have proposed conformation as an umbrella term for varieties of semantic/epistemic success (including truth, similarity, approximation, isomorphism, homomorphism) that enable us, as epistemologists, to countenance multiple non-congruent accounts of the same phenomenon.[17] Is this too coarse-grained a form of evaluation? How, if multiple approaches are correct, would

[15] See Longino (2006).

[16] See the debates from which the opening quotes to this paper are drawn. Also Turkheimer and Gottesman (1991) versus Gottlieb (1991) and also McGue (1994); Maccoby (2000).

[17] Longino (2002).

we determine which to use in practical situations? Doesn't application of scientific models/theories presuppose their epistemic superiority to alternatives?

Here, it seems to me, is the appropriate place for pragmatism, as a higher order sorting procedure for approaches that meet the standard of conformation mandated by the pluralist. Pragmatism is often accused of recommending acceptance of hypotheses and theories solely on the basis of their utility, which conjures images and memories of racist and otherwise faulty science. But paired with the kinds of empirical requirement that are central to pluralism, pragmatism can help address the problem about applicability of incompatible but equally empirically adequate approaches. Pragmatism, as a second order sorting procedure, recommends that we evaluate theories and models with respect to the specific questions they set out to answer and the kinds of intervention in the world the answers make possible.[18]

Each of these approaches does specific kinds of work, reveals particular families of causal dependencies, knowledge of each of which serves useful purposes. Behavior genetics provides clues to the function of particular genes or gene complexes and narrows the search for intermediate physiological processes. (This capacity is on display in the Caspi and Moffitt work, among others.) Behavioral neuroanatomy and neurophysiology provide clues to the interrelation of neural structures and processes. That is, regardless of the extent to which they account for the expression of any given behavior, research conducted with those frameworks is likely to have cognitively and practically useful outcomes. Research conducted within the social-environment framework enables comparisons of the effectiveness of different environmental interventions in modifying behaviors. The Developmental Systems approach at least helps apply brakes to overhasty application of single factor frameworks as well as encouraging, if not research that could directly test the full set of interactions in any given instance, research that tries to identify specific (mostly pairwise) interactions. Finally, the obvious value of the G+E+N approach is that, when it achieves results, it helps to identify proximate causes of identifiable psychiatric disorder (in those cases that fit the model) and, thereby, a strategy for therapy. The answer to the question: on what approach should we rely for application in intervention and policy? must be: it depends on the kind of intervention needed and the kind of policy required.

2.5

A pluralist stance has informed the approach to analysis of this research, but it was suggested by a preliminary investigation that revealed that all approaches were home to research efforts that could claim empirical success. Pluralism is a way of trying to make philosophical sense of this situation. I have tried to show how it is that these different approaches could all be successful by showing that there is no

[18]For further discussion, see Longino (forthcoming).

common basis of evaluation, even though methods internal to the approaches are adequate to separate empirically adequate from inadequate. But, pluralism without the kind of second order pragmatism outlined above is incomplete.

References

Brunner, H.G., et al. (1993a): "Abnormal Behavior Associated with a Point Mutation in the Structural Gene for Monoamine Oxidase A." *Science* 262: 578–580.

Caspi, A. & Moffitt, T. (2006): "Gene-environment Interactions in Psychiatry: Joining Forces with Neuroscience." *Nature Reviews: Neuroscience* 7: 583–590.

Caspi, A., Sugden, K., Moffitt, T. et al. (2003): "Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene." *Science* 301: 386–389.

Coccaro, E., Gabriel, S. & Siever, L. (1990): "Buspirone Challenge: Preliminary Evidence for a Role for Central 5-HT-1a Receptor Function in Impulsive Aggressive Behavior in Humans." *Psychopharmacology Bulletin* 26: 393–405.

Gottlieb, G. (1991): "Experimental Canalization of Behavioral Development: Theory." *Developmental Psychology* 27: 4–13.

Gottlieb, G. (2001): "A Developmental Psychobiological Systems View: Early Formulation and Current Status", in: Oyama, S., Griffiths, P.E. & Gray, R. D. (eds.): *Cycles of Contingency*. Cambridge (MA): MIT Press, pp. 41–54.

Kellert, S., Longino, H. E. & Waters, C. K. (2006): "The Pluralism Stance", in: Kellert, S., Longino, H. E. & Waters, C. K. (ed.): *Scientific Pluralism (Minnesota Studies in Philosophy of Science vol. XIX)*, Minneapolis (MN): University of Minnesota Press.

Lavigueur, S., Tremblay, R. & Saucier, J.-F. (1995): "Interactional Processes in Families with Disruptive Boys: Patterns of Direct and Indirect Influence." *Journal of Abnormal Child Psychology* 23: 359–378.

Longino, H. E. (2001): "What Do We Measure When We Measure Aggression" *Studies in History and Philosophy of Science* 32, 4: 685–701.

Longino, H. E. (2002): *The Fate of Knowledge*. Princeton (NJ): Princeton University Press.

Longino, H. E. (2006): "Theoretical Pluralism and the Sciences of Behavior", in: Kellert, S., Longino, H. E. & Waters, C. K. (ed.): *Scientific Pluralism (Minnesota Studies in Philosophy of Science vol. XIX)*, Minneapolis (MN): University of Minnesota Press.

Longino, H. E. (forthcoming): *Understanding the Sciences of Human Behavior* (working title). Chicago, IL: University of Chicago Press.

Luntz, B. & Widom, C. (1994): "Antisocial Personality Disorder in Abused and Neglected Children Grown Up." *American Journal of Psychiatry* 151: 670–674.

Maccoby, E. (2000): "Parenting and its Effects on Children: On Reading and Misreading Behavior Genetics." *Annual Review of Psychology* 51: 1–27.

Mason, D. & Frick, P. (1994): "The Heritability of Antisocial Behavior: A Meta-Analysis of Twin and Adoption Studies." *Journal of Psychopathology and Behavioral Assessment* 16: 301–323.

McGue, M. (1994): "Why Developmental Psychology Should Find Room for Behavioral Genetics", in: Alexander, C. N. et. al. (eds.): *Threats To Optimal Development: Integrating Biological, Psychological, and Social Risk Factors*, Hove: Lawrence Erlbaum Associates, pp. 105–119.

Oyama, S. (1985): *The Ontogeny of Information*. New York: Cambridge University Press.

Risch, N., Herrell, R., Lehner, T. et al. (2009): "Interaction Between the Serotonin Transporter Gene (5-HTTLPR), Stressful Life Events, and Risk of Depression: A Meta-analysis." *Journal of the American Medical Association* 301: 2462–2471.

Scarr, S. (1995): "Commentary" *Human Development* 38: 154–157.

Tellegen, A., Lykken, D. T., Bouchard, Jr., T. J., Wilcox, K. J., Segal, N. L. & Rich, S. (1998): "Personality Similarity in Twins Reared Apart." *Journal of Personality and Social Psychology* 54: 1031–1039.

Turkheimer, E. & Gottesman, I. (1991): "Individual Differences and the Canalization of Human Behavior." *Developmental Psychology* 27: 18–22.
Wahlsten, D. & Gottlieb, G. (1997): "The Invalid Separation of Effects of Nature and Nuture: Lessons From Animal Experimentation", in: Sternberg, R., Grigorenko, E. et. al. (eds.): *Intelligence, Heredity, and Environment*, Cambridge: Cambridge University Press, pp. 163–192.