Causal Democracy and Causal Contributions in Developmental Systems Theory

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In reworking a variety of biological concepts, Developmental Systems Theory (DST) has made frequent use of parity of reasoning. We have done this to show, for instance, that factors that have similar sorts of impact on a developing organism tend nevertheless to be invested with quite different causal importance. We have made similar arguments about evolutionary processes. Together, these analyses have allowed DST not only to cut through some age-old muddles about the nature of development, but also to effect a long-delayed reintegration of development into evolutionary theory.

Our penchant for causal symmetry, however (or 'causal democracy', as it has recently been termed), has sometimes been misunderstood. This paper shows that causal symmetry is neither a platitude about multiple influences nor a denial of useful distinctions, but a powerful way of exposing hidden assumptions and opening up traditional formulations to fruitful change.

1. Introduction. *Causal democracy* is Philip Kitcher's term (in press). He uses it to contrast his own reasonable "interactionism" with the well-intentioned obscurantism of Richards Lewontin and Levins (1985, for instance), and of the developmental systems perspective. Kitcher declares that standard "interactionism" is doing just fine: no conceptual change is needed (though he admits that practitioners could occasionally be a little more careful). But I have argued that those standard views are actually laden with unjustified privileging; to the extent that Kitcher is a thoroughgoing causal egalitarian about biology, perhaps he is voting with the

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wrong party. In fact, I use scare quotes around "interactionism" throughout this paper, to signal my skepticism, and to contrast it with my own *constructivist interactionism.*

The body of writings on which I draw is sometimes referred to as an approach, framework, or perspective, to avoid excessively narrow connotations of *theory.* I use DST here for convenience. (It is already in use, and, unlike alternative labels using *approach* or *perspective,* it can be used without a definite article.) The DST works Kitcher cites are Griffiths and Gray 1994 and my 2000b. Though I do not attempt to speak for Levins and Lewontin here, I will deal with a few of Kitcher's complaints from the vantage point of my own work. A kind of symmetry, or parity, figures importantly in it, and I use *causal democracy* in my title to take it back for developmental systems theory (DST). I do so in order to make a point about Kitcher's critique. I do not, however, wish to adopt it more generally *as a term.* The sociopolitical connotation, while perhaps rhetorically useful in certain contexts, introduces into already-complicated discussions rather more additional baggage than is likely to be helpful.

2. Puzzling Privileges: The Analytic Use of Parity Arguments. Parity of reasoning is a standard tool in philosophy. Something like it appears in informal discourse as well, often with reference to fairness or equity. ("Would you say that if I were white, male, tall . . . ?") In DST we might ask whether a *directive* or *controlling* function can consistently be attributed to just some interactants (participants) in development, while the others are merely supportive, or whether the asymmetrical assignment of labels reveals some unprincipled privileging. I begin with some examples of this critical use of parity questions. Then I turn to the constructive uses, and finally, I say something about what parity does and doesn't do in DST.

2.1. Determination of Variance: Contributions to Variation. There are many senses of *genetic, innate, inherited,* or *biological.*[1] They are seldom distinguished, though their empirical correlates, when there are any, are diverse. Each implies a polar opposite: *environmental, learned, acquired, cultural.* Consider the following: A phenotypic character (P) varies with an environmental factor ($Es_{1, 2}$ & $_3$) when genotype (G_1) is held constant (Fig. 1). This shows *environmentally determined, or environmentally controlled, variation.*

1. Distinctions become increasingly refined over time. This old and disordered complex of beliefs about biological nature is widely agreed to be troublesome, but the disorder resists resolution for interesting reasons. The important issue of ambiguity cannot be discussed here (though I will touch on several uses of *determination).* See Bateson 1991; Lehrman 1970; Wimsatt 1986, 1999; Oyama 2000a, 2000b.

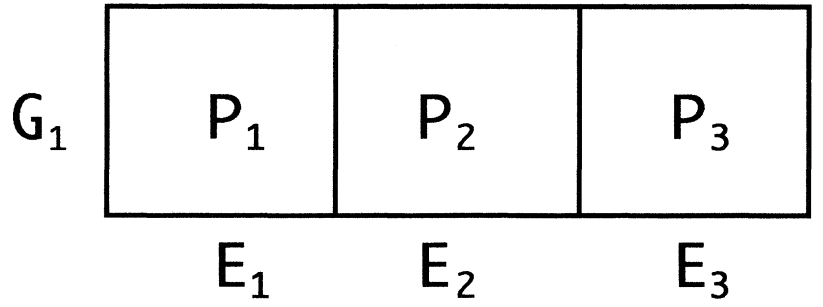

Figure 1. Environmentally controlled or determined phenotypic variation.

If instead, the environment (E_1) is held constant and the phenotype varies with genotype ($Gs_{1,2}$ & $_3$), as in Figure 2, those differences in developmental outcome are properly termed *genetically determined, or genetically controlled.* This is one way of identifying *heritability* or *a genetic base*; it refers to genotype-associated differences under standard conditions.[2] So far, so good, in the sense that parallel reasoning is apparently being used for parallel statistical patterns. Veterans of the sociobiology and IQ wars will recall that strict usage dictates that only *differences* may be genetic or environmental. The features themselves, being phenotypic, are supposed to be incapable of being partitioned by degrees of genetic and environmental causation; nor can anything be inferred from heritability figures alone about their relative developmental flexibility under other conditions. So quantifying causal contributions to *variation* isn't the same as quantifying causal contributions to *organisms* (Lewontin 1974, Sober 1988). (If the rule about variation and phenotypes were being followed, of course, one would not generally hear of innate or acquired *traits,* depending on the sense intended, but that is part of my point.)

2.2. Determination of Invariance: Contributions to Phenotypes? These first two cases presuppose population variation, and the reasoning appears straightforward. What if we shift our focus to *in*variance? Let's look at a species universal, like the human smile, or walking (Fig. 3). Now there is no correlation, because there is no variation, at least none that we are

2. Throughout, I will use *gene* and *genetic* as loosely as they tend to be employed in the research areas in question. Multiple meanings of *gene* obviously add complexity that I do not treat here. I am also accepting the language of genetic correlates even though they are often established by tracing pedigrees. On meanings of *gene* see Griffiths and Neumann-Held 1999, Neumann-Held 1999, and various entries in Keller and Lloyd 1992.

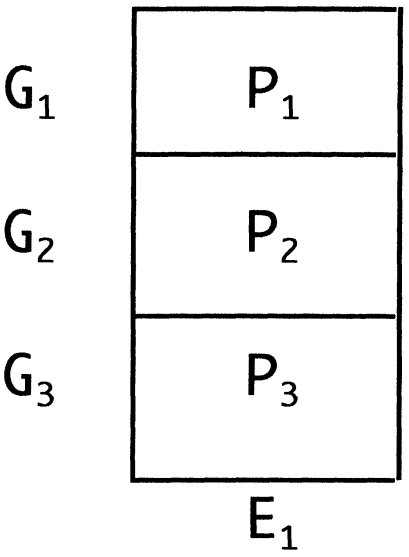

Figure 2. Genetically controlled or determined (heritable) phenotypic variation.

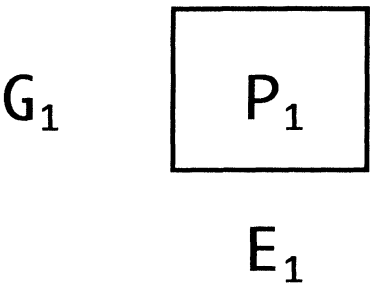

Figure 3. Universal (no phenotypic variation, normal genotype and environment): Outcome typically said to be genetically controlled, programmed, determined, etc.

interested in (an important distinction). Such traits are generally said to be encoded in or determined by, the genes. If the developmental environment is acknowledged, it tends to be only as a source of materials and conditions. Note that the genes that accounted for *differences* in Figure 2 now break free of the methodological strictures that say that manipulated factors show us something about causes that controlled ones do not: now genes are invoked to account for *lack* of differences. Meanwhile the environment fades to a vague permissive background. Such asymmetrical

explanation seems odd even if we complexify the diagram to show the diversity of genotypes and developmental environments concealed in the previous one (Fig. 4). There seems to have been a shift from statistics to a trickier mixture of the semantic and causal. The genes are given special creative, executive, directive functions that are denied other interactants; hence my term, *homunculoid gene*.

Figures 3 and 4 are meant to set your asymmetry detectors tingling. Look at the first two arrays again: this time the same apparently unproblematic sources-of-variation examples have some typical explanations added to their captions. Now they may look less straightforward. Keep in mind that phenotypic outcomes are always jointly specified by genotype-environment (G-E) pairings.[3] The first array (Fig. 5), with a single geno-

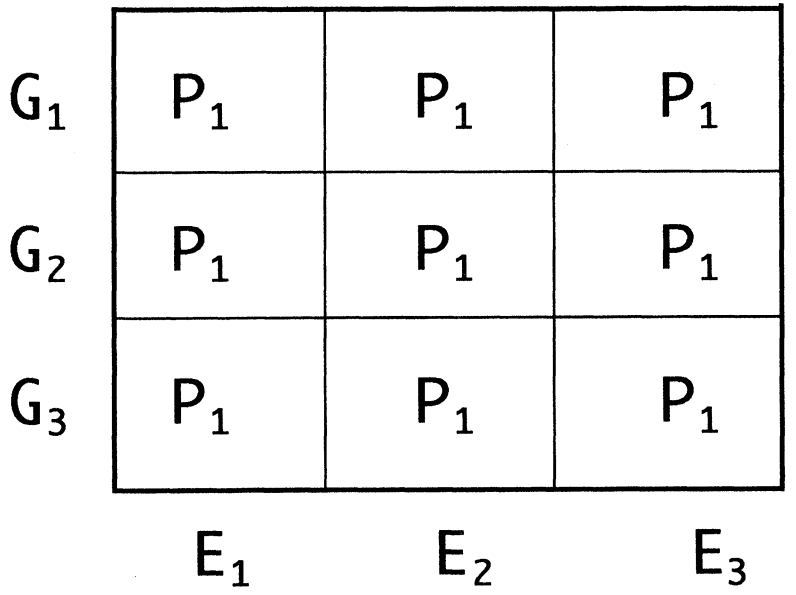

Figure 4. Universal (no phenotypic variation despite normal genetic and environmental variation): Outcome typically said to be genetically controlled, programmed, determined, etc.

3. We'll ignore for now that each G usually includes many genes in differing states, along with their associated cellular and macroscopic environments, including the organism and its relevant surround. We'll also ignore the fact that the Es include many complexly related environmental factors at many scales, that different environments may activate different genes and therefore be associated with different *effective* (active) genotypes, and that environmental manipulations typically change only a small subset of factors. Talking about genes interacting with global environments, furthermore, encourages level-collapsing travesties like "the genes interact with culture," when genes actually interact only with other molecules in an intracellular microenvironment.

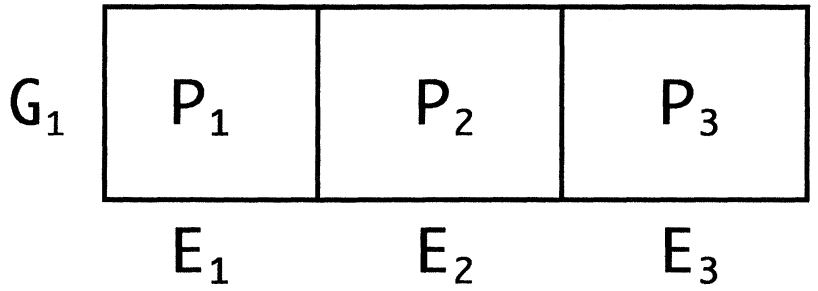

Figure 5 (Fig. 1 with expanded caption). Environmentally controlled or determined variation ("Norm of reaction, or range of possibilities, often said to be defined by, encoded by, determined by the genes").

type associated with different phenotypes in different environments, is one way (though not the standard one) of showing what geneticists call *a norm of reaction*. Recall that the variation here is environmentally determined. Yet we are often told by standard "interactionists" that the norm of reaction itself is specified by, encoded in, determined by, the *genotype*. Or that the genes contain the potentials, while the environment influences their expression.[4]

Note that for Figure 6, it seems distinctly odd to say the range of possible outcomes is *encoded in* or *defined by* the environment, though it is simply the converse of 5. It is instructive to ask oneself why the explana-

4. On this kind of explanation of phenotypic variation, found, for instance, in Freedman 1979 and Mayr 1976 (Scarr 1981 has a similar formulation), and Oyama 2000a, Ch. 3. One could say that since environmental variation determines any phenotypic variation in a norm of reaction, it is the environment that "defines" the norm of reaction for this genotype. I have never seen this said, but the opposite description is certainly found, especially when an author is trying to reconcile the existence of phenotypic variation with the concept of a fixed genetic program.

Given this confusion over *determination*, it is perhaps unfortunate that Kitcher chooses to follow vernacular (or at least nonbehavior-genetic) usage in his discussion of norms of reaction. He employs *genetic determination* for a flat norm of reaction (lack of relation between phenotypic and environmental variation *when genotype is held constant*). Many critics of genetic determinism do this; see also Block 1995–96. The advantage is that it fits one aspect of popular understanding: the genes as explanations of rigidity. It does not, however, touch the more subtle, and ultimately more troublesome, issues of underlying tendency or essence, discussed in the next subsection. Since any attempt to clarify issues of biological determinism must contend with behavior-genetic research, furthermore, I wish these writers had decided differently, perhaps adopting Lehrman's (1970) phrase, *developmental fixity,* and reserving *determination* for the more technical, source-of-variation sense.

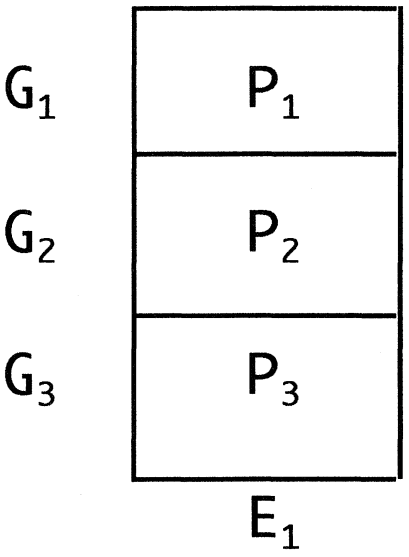

Figure 6 (Fig. 2 with expanded caption). Genetically controlled or determined (heritable) phenotypic variation ("Range of possibilities defined by, encoded by, determined by *the environment*"?).

tions sound so different, and how one would justify each.[5] This sort of asymmetry makes parity of reasoning an attractive device: explicitly asking whether the logic employed for one case can be used for another one that is usually treated differently can bring to light assumptions that otherwise remain hidden.

2.3. Cryptic Representations. Such privilegings are, I believe, attempts to capture the intuition of an underlying truth or reality, of something essential and deep that stands behind the mere phenotype. That intuition survives one technical correction after another—because it's not *about* method, but about *meaning* and *being*. The homunculoid gene plays *mind* to environment's *matter*. It carries a pre-scribed—already written—representation of what the organism is *meant* to be. According to this reasoning, some phenotypes approach their cryptic ideals more closely than

5. Some versions of the nature-nurture dichotomy include a "symmetry" of a different sort, whereby the genes specify innate characters while the environment specifies learned (or acquired) ones. This is traditional developmental dualism, and has no place in the notion of a developmental system. For a description of these two models, see Bateson 1983 and Johnston 1987.

others; for some, an organism can apparently misrepresent its own genotype.[6]

Challenging such convictions takes more than the piecemeal critiques that Kitcher advocates: the pieces also have to be put together differently. While important, local corrections are unstable to the degree that the larger network of nature-nurture images, concepts and practices remains in place. Intentional genes resist methodological critique just because their relations to actual data and analyses are obscure. Kitcher's own investigation of genetic determinism focuses on norms of reaction, for example, and thus bypasses these more elusive issues. Yet only by examining them can we begin to address the "social forces" he blames for scientists' slips.

Lumping nongenetic factors under the umbrella term *environment* conceals the multiplicity and changing complexity of environments. We cannot undo a dichotomy at one scale without considering others, or rework one opposition without attacking related ones. The issues are underspecified and incompletely distinguished in the first place, so the extent of the mess is easy to miss (even, occasionally, when one steps in it). Perhaps this is where the question of the "radicality" of the developmental systems approach comes in. Because the complex of ideas about biological causes is too large and slippery to yield to local fixes, critical strategies must be applied repeatedly and systematically, and followed to their sometimes unorthodox-sounding conclusions. That's when the scandalized outcries erupt ("Equal, but not *that* equal!"), even as particular points are not only conceded, but declared to be common knowledge, and thus hardly worth mentioning at all.

3. The Constructive Use of Parity Arguments in DST. Parity analysis can having a "pushing" function (Oyama 2000b, Afterword): flushing out hidden assumptions, provoking tighter and more principled arguments or exposing untenable ones. But there's more to DST. The constructive use of parity arguments provides an alternative to treating the motley of nature-nurture discriminations as a set of alternative (albeit imperfect) ways of glimpsing a unitary hidden nature. The discriminations must stand on their own if they are to stand at all. This does not mean the various nature-nurture questions are always independent of each other, just that they are different; relations must be empirically established. If one really is interested in abilities that are present at birth, for instance, one can

6. See Block 1995–96; Lindzey, Hall, and Thompson 1978, 42; Nash 1970, 29. Neander's (1995) treatment of misrepresentation and evolutionary function shows how similar ideas are dealt with in certain biological approaches to philosophy of mind. This is one of the intriguing points of contact (and conflict, it would seem) between developmental systems work and that literature.

certainly study them. One can also see to what extent they are associated with other indicators of "nature," like presence in phylogenetic relatives. These do not automatically go together. One has only to reflect on the fact that heritable variation and its lack have *both* been taken as signs of innateness. The same is true of presence and absence in related species: Incest avoidance (allegedly universal in humans) has been supposed to be innate in us because it is seen in other animals, and language is supposed to be innate because it is not. Again, the point is not that all notions of nature are meaningless, but that their meanings are often unclear, and that the various senses are apt to be run together, improperly inferred from each other, needlessly opposed—in short, to suffer all the indignities that plague such large, disorderly, but (for many, apparently) indispensable ideas.

3.1. Similarities Between Genes and Environments. Start with the following uncontroversial statements about genes and environments:

a) Both (as classes) are necessary for development; neither is sufficient.
b) Variation in either can, but often does not, account for variation in outcome.
c) Normal outcomes are often possible despite abnormalities in either (assuming these abnormalities can be independently assessed).
d) Variants in either may affect all, many or few organisms in a population.
e) Variants in either may enter into all, many or few successive life cycles.
f) Variation in either may be correlated to varying degrees (including zero) with reproductive lineage.

3.2. Interdependence Between Genes and Environments. Such similarities between the two traditional categories are significant. Both kinds of factors also have contingent effects, and contingencies may reach across the very skins and membranes that are generally used to discriminate these two kinds of factors as classes of developmental causes. This interdependence threatens the causal primacy and inherent meanings of the homunculoid gene.

a) What counts as a developmental interactant, and what aspects of it are relevant, depends on others; the constitution of the system is defined in interaction. (The effective stimulus and the organism define each other; some noises are famously audible to dogs and not to their owners, so they are stimuli only to the former, and only the former are hearers. Whether a bit of DNA is "read"

depends on its chromosomal neighbors and the cellular surround, and whether a molecule in that surround becomes a resource for biosynthesis depends partly on the availability and state of the DNA.)

b) What kind of interaction occurs is system-dependent. (Whether two animals play or fight depends on the situation and the state of each. Whether and how chemicals interact depends on conditions and the identity of the substances.)

c) The effects of the interaction are, too. (Milk ingested in childhood may be easily assimilated, but cause indigestion a decade later. A gene product may have drastically different impact in different cells, on an organism in different circumstances, or in organisms of different species.)

d) Interactants often bring about concrete changes in each other. (An insect is nourished as it ravages the plant. Though the DNA is often regarded as unchanging, even as it brings about changes in other entities, many intra- and transgenerational effects on the chromosomes have been reported, for example, by Jablonka and Lamb 1995.)

Together, systemic interdependence and parity (for more examples, see Jervis 1997, Lewontin 1982) answer the bewildered query, "If not designer genes, then *what*?" The constructive use of parity blocks both causal and semantic privileging, giving us the systems of heterogeneous resources or interactants that produce, maintain, and reproduce phenotypes and their developmental environments.

At any level, these developmental systems encompass a focal entity, which can be the organism itself, and its causally relevant surround. The same applies at other scales, say molecules in their subcellular surrounds. There is no central organizer, no repository of goals or instructions, no prime mover. In DST "nature" is not a phantom reality standing behind the phenotype: the phenotype in its surround is *all the nature there is*, and this is plenty. It is always a nurtured nature—that is, a changing organism located in time and space: in short, in a world. It is not enough to say that psychological development is due to interaction, while the body is programmed, or that some modules are in the genes while others must develop.[7]

3.3. Routes to the Next Generation: One, Two, or Many? Applied to the concept of heredity itself, constructivist interactionism reconfigures the

7. Important points are made by those who stress a developmental perspective on modularity (Elman et al. 1996, Karmiloff-Smith 1992). From my point of view, though, they do not take their "rethinking" of innateness far enough.

relationship between development and evolution. In DST, developmental interactants or resources are "transmitted" in the sense of becoming available to a life cycle. This happens in a wide variety of ways, including simple persistence in the niche. Phenotypic traits, on the other hand, are not transmitted but must be constructed in development. The usual flow of disembodied genetic "information" between the generations, with or without a second channel for culture, is replaced by more or less faithfully repeating systems, each of whose operation helps (or not) to make available the interactants for the next life cycle. If one wishes, one can still trace the distributions of any interactants, including genes, in successive generations; the point is not to prohibit research, but to understand it differently. In addition, more novel questions now can be posed as well.

When these systems of developmental processes and products change, in constitution or in prevalence, evolution occurs. Without this conceptual reworking of heredity and evolution we are left with a conglomeration: phenomena described with a fairly "interactionist" vocabulary at one level but not another, or construction posited for some traits and inheritance for others, always with constituents obediently segregating for admission to the next generation, either through the door marked *Genetic* or the one that says *Other*. And the nature-nurture complex, with its ambiguities and puzzling privileges, gets passed on as well.

4. Conclusion: What Parity Does and Doesn't Do in DST.

4.1. Separate But Equal? There has been some confusion about what parity does. It clearly doesn't lead to the conventional "interactionism" that accepts traditional categories of nature and nurture, biology and culture, even if both are "important," and "interact." This is a separate-but-(not quite)equal nonsolution.

4.2. Denial of Difference? Nor does parity mean that in any particular analysis, all things are equally important or "the same," that no distinctions can be made. It is precisely the careful and principled discrimination of a whole set of issues that DST accomplishes, most notably with the disambiguation of nature-nurture terms (see fn. 1). This remedies a certain *lack* of discrimination in much of the existing literature. The single metric implied by accusations of blanket sameness, furthermore, is precisely one of the things that DST denies. Distinctions rise from particular research projects, and from the questions being pursued. DST helps to distinguish both. The point is not that everything's the same, but that things that are "the same" in one analysis won't always be so in another, or at another time.

Our main quarrel is with developmental and evolutionary dualism. The distinction between traits that are formed (more) from the inside and ones

shaped (more) from the outside is indeed one that we find mischievous, whether we are talking about ontogenetic timespans or phylogenetic ones. It may even be subtly at work in one of Kitcher's confrontations with DST. Developmental systems theorists Griffiths and Gray (1994) wrote a critique of Sterelny and Kitcher's 1988 paper on the notion of "genes for." In the more recent paper I've been referring to, Kitcher (in press) conceded that the same logic that warrants saying a gene is "for X," in the sense of reliably making a difference in the phenotype, could also be used for cytoplasmic or ecological features "for X." He asserted that this confirms the adequacy of his democratic "interactionism."[8] Now it is one thing gracefully to acknowledge that one has missed an insight that could—conceivably—have been produced by one's own reasoning, but in fact wasn't. It is quite another to claim that the insight flows naturally from the very framework one was using when one missed it. The developmental systems framework *generates that serious attention to possible symmetry as a matter of course.* In contrast, the standard one minimizes the probability that such unorthodox symmetries will be seen, even when they are not logically prohibited. Remember Figures 1–4.

These are quite general conceptual revisions, not specific empirical predictions. (This is why I tend to use less restricted, hypothesis-bound terms than *theory*.) DST's emphasis on development should not be confused with certain other "developmental" approaches in evolutionary theory. Taking a more symmetrical view of both development and evolution isn't to insist that evolution is more influenced by development than by selection, say, any more than it licenses particular predictions about any given developmental course. It is also important to distinguish this rethinking of evolution in developmental terms from what people are apt to imagine, with well-schooled horror, when they hear the two words juxtaposed:

8. Speaking of a landscape feature "for X," in parallel with a gene "for X" (always against a fixed causal background), seems to me to be unarguable in context but still completely nonstandard, despite Kitcher's implication that it fits comfortably with standard "interactionism." He cites talk of proteins "for" polarity in molecular developmental genetics, apparently intending it to be taken as evidence that conventional thought is causally democratic. The *Drosophila* literature, he says, "has already begun to emphasize the causal role of proteins deposited by the mother in the cytoplasm of the ovum." We could ask, though, why researchers, at century's end, would have just "begun" to stress the importance of maternal proteins. Maternal contributions have been known for a long time. The question is how they are treated within the overall explanatory framework. For the researchers in question, they may well be seen as more instances of "genetic control," rather than as something that requires them to put other influences on comparable formative footing with genes and their products. (For examples of this kind of reasoning, see Bonner 1988). In my *Ontogeny of Information* (2000b, Ch. 8), I discussed a case in which maternal behavior in cross-fostering research was handled in just this way.

"Why, they think evolution is like development!" where development is understood as internally driven, predictable, and goal-oriented. *In DST that internalist view of development is exactly what is called into question.* I do think the contrast between the two processes looks different once it has been recast in developmental systems terms. One is about organismic life cycles, and the other, about changes in, and relations among, large numbers of such cycles, similar and dissimilar, successive and contemporaneous. (See Griffiths and Gray 1994, 1997; for some agreement and some dissent, see Sterelny, Smith, and Dickison 1996).

We reframe a picture, not to make it an unintelligible smear, but to bring out some qualities and differences and reduce the salience of others. Doing so allows us to see things in a different way.

4.3. All or Nothing? Other complaints about DST are similarly inaccurate. Our emphasis on causal interdependence doesn't mean that everything is so connected to everything else that analysis is impossible, or that in order to study anything, you must study everything.[9] It means you take possible interdependence into account when you analyze and interpret. It is not clear whether Kitcher (in press) intends his accusation of near-incoherence about causality to apply to DST as well as to Levins' and Lewontin's dialectical biology. As I noted earlier, I do not presume to speak for the latter, but DST, far from making causal inquiry impossible, can make it both more precise (by discouraging certain overgeneralizations) and more ample (by pointing to potentially significant paths of influence on the focal phenomenon that other approaches are apt to miss). Making explicit the contingency of DNA function on cytoplasmic conditions hardly prevents the investigation of the DNA's effects on the cytoplasm, though it should make it harder to say silly things about genetic primacy (something Kitcher does not do in his critique, but that is common enough in the areas both he and I are discussing). It also broadens the potential investigative field *in nonarbitrary ways* by stressing the importance of understanding how those cytoplasmic conditions are main-

9. Another source of Kitcher's (in press) skepticism. This is often the first, anguished worry of the researcher as well. Yet as any empirical worker knows, however confidently "extraneous" factors are assumed to be irrelevant, one must always be ready to be shown wrong, just as any worker routinely chooses a few things to study, knowing full well that these are not the only things that could be studied. Causal interconnectedness in the world is not DST's invention. We just deal with it differently, by including it in our formulations from the start, rather than bringing it in only when forced to do so, and then perhaps marginalizing it in some way. Foreswearing the implied null hypothesis of genetic programming helps to keep open and present the possibility that considerations relevant to the investigation at hand will issue from other fields, perhaps necessitating a redrawing of research boundaries.

tained, reconstructed, or changed. The usefulness of manipulation and control for identifying operative factors is not nullified by our awareness of causal contingency, as Kitcher seems to think, though that awareness may increase the probability that the conclusions we draw will reflect precisely the caution he so rightly counsels. A person working from a DST perspective may employ standard techniques for "separating" or "isolating" influences in research, that is, but have a different understanding of what is being done.

Finding system boundaries—delineating a set of interesting factors that are connected in relevant ways—is a pragmatic matter, tied to the specifics of the inquiry. We can take for granted the processes that bring an organism to the start of our study, but we should also realize that this is provisional blackboxing, not an explanation of what's in the box and how it got there, much less its future prospects.

4.4. Zero Privilege? I don't want to imply that it is possible to do away with all privileging, if this means having a theory-free, interest-free God's eye view from nowhere. I have focused my own parity plays on a set of historically influential problems, and this decision suggests *my* priorities: to elaborate an account of development free of the inconsistencies of traditional ones, to give more weight to the actual role played by a developmental interactant than to its location vis-à-vis the organism's skin, to regard the ways in which the heterogeneous resources for developmental construction do or do not become available to a life cycle as more important than staying inside the lines of a restrictively nucleus-bound heredity.

The emphasis on developmental construction arises partly because so many notions of biological causation give it short shrift. The focus on actual interactions comes from a desire to test the rhetoric of magic molecules. The wholehearted inclusion of environmental factors in the developmental story comes from a concern about the consequences of such rhetoric, both inside and outside science. I highlight the interdependence of these interactants and their effects, finally, because I have seen the ways the language of genetic primacy, autonomy, and essence can be used, and believe it more important to undo this tangle of ideas about nature than to respect certain aspects of current practice.

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