conceivable for studies of the canalization of behavior. Consequently, alternative techniques must be used and applied to the types of data available or potentially available for analysis.

Most experimental studies of canalization have been based on characters that are almost completely invariant normally. Such characters were selected because an increase in variance caused by the application of environmental or genetic stress is much easier to demonstrate than an increase of variability in a character that has an appreciable variability normally. Apart from certain reflexes, there are probably few (if any) aspects of behavior that are analogous to the strongly canalized characters used by the experimenters. Studies in behavior will most probably involve characters that are variable at the norm.

A feature of canalized characters that has been emphasized in some of the experimental studies (see Rendel 1959) is that the form of the variability distribution of a canalized character will change under environmental or genetic stress. Consider first the relationship of the first and second moments of a frequency distribution (the mean, k_1, to the variance, k_2). The variance of a canalized character will be at a minimum when the distribution of the character is centered on the norm. The variance will increase with deviation of the mean from the norm, reaching a maximum when the deviation is of such magnitude that the buffering mechanisms are not effective.

Next, consider the relationship between the first and third moments, the mean, k_1, and the degree of skew, k_3. As the mean of a character deviates from the norm we expect the distribution of the character to be skewed toward the norm. The degree of skewing is expected to increase with increasing deviation up to a maximum, and then to decrease to zero with further deviation of the mean from the norm. The expectation is that populations whose mean deviates below the norm will show skewing upward toward the norm, whereas populations whose mean deviates above the norm will show skewing downward toward the norm. Such changes in the degree of skewing are expected for canalized characters, and can be considered a very useful diagnostic.

Finally, consider the relationship between the first and fourth moments, the mean and the degree of kurtosis, k_4. The occurrence of buffering mechanisms causes a reduction of variability, which can result in a marked degree of kurtosis. Deviation of the mean above or below the norm will cause a reduction of kurtosis (k_4).

How may these observations be applied to behavioral characters? The essence of the above is that if a canalized character is stressed beyond the limits of the buffering system of that character, then specific relationships between the mean and k_2, k_3, and k_4 should be found. Turning the problem around, if populations have different means for a particular behavioral character, and the relationships between the mean and k_2, k_3, and k_4 are similar to those found for stressed canalized characters, then it may be inferred that the character is canalized.

There are several ways in which the relevant data may be obtained. Populations that differ either genetically or environmentally can be measured on some character(s), the population means, k_2, k_3, and k_4 computed, and their relationships examined. For the mean to variance relationship, for example, consideration is centered on the population with the lowest variance, and changes in variance with changes in population mean are examined. If variance increases with increasing distance from the "centered" population, then canalization is inferred. For examining skew and kurtosis, similar techniques are employed.

Such measurements cannot identify without question whether or not a particular character is canalized, since the available range of environments and genotypes may be within the zone of the buffering mechanisms, or they may all be outside the buffering zone. In both cases the distribution of variability will not show the predicted changes. This approach will only lead to diagnoses of canalization if the series of populations to be compared differs environmentally or genetically such that the means transit the zone of canalization (the evolutionary norm).

The experimental analyses of canalization that use environmental stresses may not be feasible for human and infrahuman populations, but those that use genetic stresses are feasible since the analogues of the mutant genes used in the experiments on mice and Drosophila do occur in other species. The basis of this approach is to identify mutant types that cause a shift of the behavioral characters outside the norm. Such mutant types should, if the behavioral characters are canalized, have an increased variance and a decreased kurtosis, and they may show a skewing toward the norm.

The following is one way in which mutations can be used. First, a mutation is chosen, for example, Down's syndrome, that affects both the behavioral character in question and a nonbehavioral character. The effect on the nonbehavioral character will be variable, ranging from nearly normal to extremely defective, indicating varying degrees of mutant expression. If the mutant types are grouped into relatively homogeneous, that is, equal variance, populations based on the degree of defect of the nonbehavioral character, then the mean, k_2, k_3, and k_4 of the behavioral character can be computed for each population, and their relationships can be examined. If these relationships are similar to those found in canalization experiments, it is likely that the behavioral character is canalized.

Given that analyses similar to the above lead to a diagnosis of canalization, then further discussions of the variability can be envisaged to identify the mechanisms of canalization, that is, those features of development that respond to perturbations by altering development such that the effects of the perturbations are minimized. Although there is not a great deal of evidence on the nature of the buffering mechanisms in the canalization of morphological characters, it seems clear that their development does involve the occurrence of critical periods. We would, thus, expect the development of a behavioral character to involve one or more relatively short periods during which development is sensitive to stress. Consequently, any characteristic of behavior that is diagnosed as canalized from the analysis of its pattern of variability, would be expected to be based on a series of buffering mechanisms that become operative during fairly restricted stages of development.

The identification of critical periods is usually based on the exposure of a number of populations to a specific stress at different stages of development. This procedure is not available for human and many infrahuman species; consequently, other approaches have to be used. An extremely useful method has been to identify individuals who exhibit abnormalities for the character of interest, and then attempt to locate the stresses and critical periods by detailed analyses of their environments during development. This approach was used to identify the stress and critical periods for thalidomide and German measles.

An organism, evolved to fit its ecological niche, will be buffered against perturbations of the environment within the range of the niche. If it develops outside the limits of the niche, then the system of buffering will be ineffective and the distribution of variability of characters will show the projected changes of variance, skew, and kurtosis (Fraser 1977). Comparisons then, over the various levels of Johnston's schema could provide another dimension for attribution of generalities.

Am I a closet general process learning theorist?

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In the section of the target article entitled "Subservience to the general process tradition" Johnston (1981) quotes extensively

from part of the introduction to an article by Galef and Osborne (1978) on the role of novel tastes in potentiating visual-cue-toxicosis learning in rats. Johnston uses this material to illustrate "the power that general process theory may have to constrain experimentation." He concludes that the paragraphs he quotes "can only imply a belief [presumably on the part of the paragraphs' authors] in some general process underlying poison avoidance, which is equally well revealed by studying any form of poison avoidance in any species." Johnston's interpretation of the Galef and Osborne manuscript strongly implies that we arrived at an inappropriate series of experiments because of an extreme commitment to general process theory, a lack of awareness of interspecies diversity in behavioral capacity, and an insensitivity to the possibility that the plasticity exhibited by members of particular species might be shaped by ecological pressures. Are Osborne and I apologists for such a biologically naive view of learning processes? I think not. I certainly hope not.

Although it may be the case that general process learning theories can "constrain experimentation," as Johnston suggests, the sole example he selects to illustrate this point is not particularly well chosen. The subject species in the Galef and Osborne studies was determined by the nature of the question in which Osborne and I were interested rather than by the theoretical issues on which Johnston chose to focus attention. Johnston states that Galef and Osborne (1978) "sought to investigate the hypothesis that aposomatism (bright coloration) in many poisonous insects is an adaptation to the ability of potential predators to learn to avoid such prey, which implies that predators should indeed demonstrate the appropriate learning ability." On the contrary, Osborne and I postulated that aposematisms are an adaptation to the ability of potential predators to learn to avoid warning colored prey. Rather, as is explicitly stated in the introduction to the studies under discussion, Osborne and I were testing the hypothesis that organisms such as rats, which have been found (in standard aversion learning paradigms) to have considerable difficulty in learning to avoid visual cues associated with toxicosis, might be able to use visual cues to learn aversions under conditions reflecting the sequence of events occurring during prey capture and ingestion in natural circumstances.

I infer from Johnston's redirection of our hypothesis and his subsequent remarks that he would have preferred us to conduct experiments asking whether known insectivores (for example, blue jays or toads) have the capacity to learn to avoid toxic species on the basis of their appearance. Such experiments have appeared frequently in the biological literature, and the role of predator learning in aposematic prey avoidance is already well established. It seemed to Osborne and me more interesting to inquire whether a generalist feeder, not specialized for insectivory, might be able to use aposematic characteristics to avoid ingesting visually distinctive toxic prey, especially given the indications in the psychological literature that they might not be able to do so. Our experiments thus required a subject species not specialized for dealing with toxic prey and known to have difficulty in learning visual-cue-toxicosis associations in standard tests of aversion learning. The rat seemed a suitable choice. There is no reason or need for Johnston to infer a belief on our part that the processes of aversion learning are the same in all species.

In fact, neither Osborne nor I hold the theoretical position Johnston attributes to us, that there is "some general process underlying poison avoidance, which is equally well revealed by studying any form of poison avoidance in any species." It is difficult to see how anyone with even a superficial familiarity with the taste-aversion literature could be committed to such a view. I certainly did not intend the paragraphs quoted by Johnston, or any others in the Galef and Osborne paper, to imply such a theoretical position. I believe Johnston has read too much into the sentences he quotes concerning the central position of rats as subjects in the psychological study of the proximal mechanisms of taste-aversion learning. As a consequence, he has imputed an approval of the current focus on rats as subjects in studies of taste-aversion learning where none was intended or proffered, and he has attributed an extreme and unrealistic position to Osborne and myself. If we had believed that poison avoidance was equivalent in all species, we would not have raised the issue of choice of subject species in the first place; it is certainly not customary to do so in the psychological literature.

While it is in some sense flattering to serve as a straw man, it is uncomfortable to be saddled with theoretical positions one does not hold or even seriously entertain. As Rozin (1981) pointed out in his commentary on the target article, there is reason to expect to find both generality and diversity in learning processes. Unfortunately, predicting either the boundaries of generality or the occurrence of specializations is beyond the current state of the art. A more ecologically oriented approach to the study of learning principles, such as the one Johnston proposes, holds promise of achieving such predictive power.

From observation to principles of learning: A long and problematic route

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As psychologists with an ecological commitment, we find little to criticize either in Johnston's (1981) analyses of modern learning theory or in his desire for an ecologically based general theory. Our difficulties concern his strategy for achieving that goal. For Johnston, one begins with task descriptions for particular animal–environment systems. Next, one determines their local solutions. The identification of what is invariant over these local principles reveals general principles. And these general principles, in turn, provide the ingredients for a general theory.

At issue for us is whether such a bottom-up approach (see Johnston's Figure 1) is the one that ought to be adopted. In what follows, we discuss two reasons for our hesitation. Like some of the other commentators, we worry about what observers will themselves contribute to a task description. It seems that this fear is well placed because several commentators reveal concepts in whose terms they would make a presumably neutral task description (e.g., Rozin 1981, p. 157 "Animals face the problem of developing useful *representations* of the world and effective *plans* of action," emphases added, or the "cognitive maps" of Barlow & Glickman 1981). But there is a more fundamental problem: Even if one can avoid bringing concepts to observation, one cannot avoid bringing measurement techniques to observation. And the yardstick with which one describes and quantifies natural phenomena itself embodies a host of assumptions about which dimensions of those phenomena are salient *and* about how quantities vary along those dimensions. What are needed are yardsticks that can bend and stretch in a way that makes them fit naturally to the entities that they quantify. Johnston appears to recognize this when he observes that ecological structure (e.g., of a city) may be different from "geometrical" (actually, Euclidean) structure, but he offers little guidance as to how an environment or an animal is quantified, or even what ought to be our concerns. Ecologically appropriate measurement requires not only that the measured system be natural but also that the measurement system be natural (Shaw & Cutting 1980). Otherwise, the rules that relate the measure to the phenomenon may be mistaken for the rules (laws) of the phenomenon. Put another way, we must guard against the prospect that the properties ascribed to learning are artifacts of