plied cautiously. Even so, in view of the high grating acuity we have found, it would be surprising if other measures, such as the spatial contrast transfer function, did not also show that falcon vision transcends that of humans.

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5. Gratings of different manufacture, (i) engine-ruled gratings (Ronchi rulings) and (ii) film copies of the engine-ruled gratings, were used. The film gratings were made in order to provide a wider assortment of spatial frequencies. High-contrast film (Kodalith ortho film, type 3) was directly exposed to magnified Ronchi rulings projected by an enlarger, a method that yielded precise control of the number of lines imposed on the film. All gratings, both film and engine-ruled, were examined under a microscope to ensure that contour edges were sharp and that the opaque contours were completely filled. Contrast $(I_{max} - I_{min}/I_{max} + I_{min})$ measured by a microphotometer was 99+ percent for all gratings. The transparent contours of the engine-ruled gratings passed 7 to 9 percent more light than those of the film gratings. This difference in transmission was corrected by filtering the engine-ruled gratings. As a test of the equivalence of film gratings and engine-ruled gratings, identical spatial frequencies of both kinds were used in different testing sessions; no differences in discrimination performance were found between sessions for any of several frequencies compared in this way. The grating and the blank stimulus were transilluminated by collimated light from two tungsten-halogen lamps. Luminance was controlled by placing appropriate combinations of neutral density filters in the optical paths. Electronic shutters were used to completely block illumination. The gratings and the blank fields were mounted as slides and attached to rotary wheels locked with detents that permitted rapid and repeatable insertion of any desired stimulus combination. The luminance levels of the displays, as seen from the starting platform, were measured before and after each session by a Pritchard photomultiplier photometer and a Salford subjective match photometer.
6. Training followed general methods of operant conditioning with food as reinforcement. The daily food ration was obtained in the testing sessions, one in the morning and one in the late afternoon. The fresh beef heart used as a reward is not a complete diet for falcons. To supplement the diet, a mouse was fed to the bird after 5 days of testing and testing was resumed 2 days later. The bird was weighed daily before and after testing; the average weight was 100 g before and 110 g after a session. Weight proved to be a sensitive index of motivation as well as a sensitive indicator of general health.
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10. The thresholds for the two observers were obtained by the method of limits; monocular viewing was used for comparison with the thresholds obtained by S. Shlaer. Binocular viewing would likely have produced a significant reduction in threshold, on the basis of prior work on binocular summation; see, for example, the review by R. Blake and R. Fox, *Percept. Psychophys.* **14**, 161 (1973).
11. We thank J. Enoch for informative discussions concerning the data required to determine the energy absorption properties of cones.
12. We thank R. Bush for his assistance in data collection, and the Tennessee Wildlife Resources Agency and the U.S. Fish and Wildlife Service for the excellent cooperation extended to us. The work was authorized by state scientific collectors permit No. 204 and federal scientific collectors permit PRT 7-01-C-Z-NV. Support was provided by NIH grant EY00931.

10 September 1975

Flavor Aversion Studies

We applaud Bitterman's general position on comparative research (*1*), but consider his criticisms of our flavor-aversion research and theory to be unwarranted. We point to a literature so voluminous that space permits citation of only a small portion.

Bitterman says, in effect, that the evidence for associative predispositions is based on experiments where testing conditions were confounded with modality, that is, visual stimuli antedated the criterion response (eating) and gustatory stimuli followed it. It has been demonstrated in many laboratories in experiments with drinkometers where both an audiovisual signal (AV) and saccharin flavor (F) are contingent upon the criterion response (licking) that rats punished with shock become conditioned to AV not to F. Conversely, rats punished with illness become conditioned to F not to AV. Furthermore, illness can be delayed for hours without disrupting conditioning to F. By increasing saccharin concentration *and its odor*, shock-avoidance responses have been conditioned to F, but here again, when the shock is delayed 210 seconds, conditioning to AV is disrupted whereas conditioning to F is reported to be enhanced. While differing in their theoretical formulations, the authors agree that AV and F are probably handled differently by central (memory) processes and interact differently with pain and illness (*2*).

Bitterman also speculates that regurgitation or some more subtle reverse transport system returns flavor to the mouth to stimulate the taste and smell receptors at the time of illness; therefore, he says, it has not been demonstrated that flavor aversion is acquired when there is a long interval between stimulus and illness. Rats acquire aversions to harmless mouthwashes followed by toxicosis even without ingestion (*3*). Rats do not acquire aversions to untasted substances tubed directly into their stomachs before illness (*4*). Rats acquire aversions to ephemeral substances that are completely altered by digestive process long before illness is induced (*5*). Aversive effects are obtained with chronic diet deficiencies and positive effects with recuperation from dietary deficiencies where regurgitation is unlikely (*6*).

According to Bitterman this new, ethologically oriented comparative psychology leads only rarely to the comparison of different animals. The conditioning of flavor aversions by illness has been studied in man, monkey, rat, mouse, cat, wolf, coyote, ferret, cougar, blue jay, quail, hawk, turtle, fish, slug, and other species (*7*).

In addition, he argues that there is a lack of interest in functional analysis, in evolutionary history, and in relating behavior to anatomical structure. This puzzling criticism follows his reference to a paper in which Garcia, McGowan, and Green (*8*) point out, as is also done elsewhere (*9*), that natural selection favors the predisposition to associate flavor with illness and that both taste receptors and internal monitors send their fibers to the same anatomical site in the brainstem. In any case, relevant neuroanatomical research has emanated from a number of laboratories (*10*).

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10 June 1975; revised 29 September 1975

Although the literature of flavor aversion is indeed voluminous, it has some serious methodological shortcomings. To illustrate its shortcomings in my Thorndike lecture (*1*), I selected for analysis an influential experiment, first reported in these pages (*2*), which was said to show that rats associate taste more readily than visual stimuli with gastrointestinal upset but visual stimuli more readily than taste with painful electric shock. The results for illness, I noted, could be accounted for in terms of the greater persistence of gustatory than of visual stimuli, and the results for shock in terms of differences in interstimulus interval and conditions of testing. Garcia, Hankins, and Rusiniak (*3*) now suggest for our consideration five additional experiments which, in my opinion, provide no better evidence of associative predisposition.

In the first of the five experiments (*4*), there were no pseudoconditioning controls, yet illness might have produced aversion to saccharin, and shock might have produced aversion to the sound of a buzzer, quite independently of pairing. In the second (*5*), training method and source of aversion were grossly confounded. In the third (*6*), pseudoconditioning controls again were omitted. The fourth experiment (*7*) was unbalanced, with two sources of aversion (shock and illness) but only one conditioned stimulus (the taste of saccharin). Since the shocked rats stopped drinking as soon as shock was turned on but the rats intubated with LiCl continued to drink for a time, there was greater opportunity for the association of taste with illness. Moreover, there was no control for the effect of illness alone on the subsequent acceptability of saccharin. The fifth (*8*) was a very good experiment designed to examine the role of interstimulus interval, a variable frequently confounded with source of discomfort in work on conditioned aversion. Its results "could be generated by conventional principles of Pavlovian conditioning plus the assumption that the trace occasioned by saccharin consumption is more persistent than that occasioned by exposure to a light-tone compound" (*8*, p. 887).

To refute the suggestion that flavor aversions may develop with relatively long intervals between ingestion and illness because taste and smell receptors are stimulated again at the time of illness by food returned to the mouth, Garcia, Hankins, and Rusiniak call on other papers no more persuasive than those already considered. In an experiment cited to show that "rats acquire aversions to harmless mouthwashes followed by toxicosis even without ingestion," infusion of the oral cavity was followed immediately by poisoning—that is, there was no long delay—and better results were obtained even so when there was opportunity for ingestion (*9*). In an experiment cited to show that "rats do not acquire aversions to untasted substances tubed directly into their stomachs before illness," honey was applied to mask the taste of any LiCl left on the catheter and to induce swallowing in the intubated rats; we may suspect that the aftertaste was masked as well (*10*). There is, in fact, some reasonably good evidence of intravascular taste from a conditioned-aversion experiment with rats in which injection of saccharin into the tail vein was followed by x-irradiation (*11*).

Two of the papers cited to show that "rats acquire aversions to ephemeral substances that are completely altered by digestive process long before illness is induced" describe experiments in which the animals were poisoned after eating either dry food accompanied by water (given on some days) or the same food mixed with water (given on others). The earlier paper (*12*) reports no differential conditioning. In the later paper (*13*), it is claimed that "rats learned to discriminate wet food from dry food plus water," but no data are presented on the consumption of wet food after poisoning with dry food, or on the consumption of dry food after poisoning with wet food. The earlier paper describes a parallel experiment with two different concentrations of the same flavor which is said to show differential conditioning; but even if we disregard statistical deficiencies, errors in procedure "discovered through major perturbations in the data" (*12*, p. 427), and what the author himself terms the "rather anomalous" finding (*12*, p. 428) that intake of the poisoned concentration did not drop appreciably (instead, intake of the safe concentration increased), we cannot overlook the fact that poisoning with the lower concentration produced no reliable preference for the higher. If the experiment shows anything at all, it is the importance of concentration. Garcia, Hankins, and Rusiniak also make the point that "aversive effects are obtained with chronic diet deficiencies and positive effects with recuperation," but these results are irrelevant to the problem of conditioning with long interstimulus intervals. Creating a deficiency guarantees close contiguity of taste with illness, and supplying it may bring prompt symptomatic relief. To quote from one of the cited papers (*14*): "Patients with a thiamine deficiency often report that injections of thiamine make them 'feel better' soon afterward."

Certainly there have been some experiments on conditioned aversion in animals other than rats, but they can hardly be called "comparative" in any strict sense of the term. They do, however, have many of the same shortcomings as the experiments with rats. In my Thorndike lecture I referred to a paper which reports two flawed experiments on visual aversions in quail, one in which food was colored with blue dye (despite the recognized possibility that it may have had a distinctive taste), and one in which blue light rather than dye was used but with no control for the effect of illness alone (*15*). The claim in a recent paper on slugs (*16*) to which Garcia, Hankins, and Rusiniak make reference that aversion develops with a 1-hour interval between ingestion and poisoning is based on inappropriate statistical analysis. A still more recent paper on conditioned aversion to sexual attractants in hamsters (*17*) displays utter disregard of the principle that the demonstration of "new associations" between stimuli requires control for the effects of the stimuli themselves (or the conditions used to generate them) apart from their temporal relation.

I stand by my opinion that the loose speculation about adaptive value which is so characteristic of ethological thinking tends to discourage functional analysis. What could be more indicative of functional disregard than the flat assertion by Garcia, McGowan, and Green that whether an aversion is the product of conditioning or pseudoconditioning "doesn't matter" (*18*, p. 19)? Nonassociative procedures show us "the

same adaptive behavior" as do associative procedures, they say; in both cases "the propensity of the animal to adapt by rejecting a novel or distinctive fluid is revealed" (*18*, pp. 18–19). As to mechanisms of associative predisposition, speculation seems to me to be premature. It might be wiser to wait until we have evidence that there is such a thing—evidence which the literature of flavor aversion, however voluminous, fails to provide.

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2 January 1976; revised 1 March 1976

Stability of Species in Geologic Time

In assessing the distribution of evolutionary rates in phylogeny, Harper (*1*) has misconstrued my contribution (*2*). I did not feel compelled to choose between the two extreme alternatives: (i) that all evolutionary change is phyletic, or occurs as gradual transition within established species, and (ii) that nearly all evolution is associated with multiplication of species. In fact, I recognized that a spectrum of intermediate possibilities exists, but conducted four tests of the fossil record which showed that phyletic change is "generally slow and of minor consequence relative to changes that frequently occur in speciation events." No claim was made that phyletic change necessarily accounts for "considerably less than" 10 percent of all evolution.

Furthermore, Harper's inference that my tests can apply only to the extreme alternatives is incorrect. The fossil record offers crucial evidence for resolution of the question. Elaboration of the test of adaptive radiation (*2*) will make these points. This test begins with the observation that species durations within higher taxa are extremely long with respect to rates of large-scale evolution. For example, an average species of late Cenozoic mammals has survived 1 to 2 million years, and yet most orders of mammals arose from primitive ancestors during only about 12 million years of the early Cenozoic. Clearly, ten or so species-to-species phyletic transitions are insufficient to produce the enormous degree of change that occurred in the origin of such divergent taxa as bats or whales. It seems inconceivable that rates of phyletic evolution were somehow dramatically accelerated for an enormous variety of early Cenozoic mammalian taxa occupying unrelated niches in diverse habitats. On the other hand, we know that speciation was rampant, as a multitude of niches was invaded in the replacement of extinct reptiles. It is unlikely that this close association between speciation and rapid large-scale evolution was coincidental.

Mean longevity of mammal species was originally calculated by a technique that did not permit estimation of skewness or variance (*2*). Is the distribution of species durations strongly skewed, allowing for many short-lived species that might have undergone rapid phyletic transition? A new kind of analysis (Fig. 1) eliminates this possibility. The analysis is deceptively simple but should be highly accurate. Each point of Fig. 1A is derived from fossil data for a particular stage of the Plio-Pleistocene of Europe. It represents the percentage of all mammal species of the stage that survived into the Würm (last Pleistocene stage). Use of the Würm rather than the Recent as an end point in this particular example avoids the effects of the famous sub-Recent mass extinction. No bias is introduced because nearly every species of the preceding Eem stage is also recorded for the Würm. Even though the fossil data and absolute time scale come from different sources, the resulting curve is quite smooth. It can be transformed into a survivorship curve, depicting attrition from some imaginary time zero, as follows. Consider a single point of Fig. 1A representing a time α, approximately 1 million years before the Würm, when 30 percent of all existing species were to survive into the Würm. As will be justified below, it is assumed that an average species of the total fauna existing at any time, including α, was in mid-duration. The stratigraphic ranges of an idealized set of all species existing at time α are plotted in Fig. 1B. Figure 1B happens to display more species of medium duration than of long or short duration, but the shape of the distribution is immaterial to the analysis. An average species of each duration is placed in midrange. As required by Fig. 1A, 30 percent are extant at the start of the Würm. In plotting a survivorship curve, the time for decline to 30 percent will be longer than the interval from α to earliest Würm because survivorship represents decline starting with a "cohort" of brand new species. More precisely, realignment of the set of hypothetical species so that all originate simultaneously (Fig. 1C) doubles the decline time. This effect can also be seen by inspection of the symmetry of Fig. 1B. The 30 percent point is therefore plotted at 2 million years in Fig. 1D and, by extrapolation, a complete survivorship curve is produced by doubling the time scale of Fig. 1A. Finally, a histogram of species durations derived from Fig. 1D is plotted as Fig. 1E.

The assumption that at any time depicted in Fig. 1A an average species was in mid-duration amounts to the assumption that rates of speciation and extinction were constant, or that a stable age distribution of species was maintained (*3*). There is no theoretical reason to believe that these conditions should have been met. On the other hand, only major departures from the conditions would have caused significant deflection of the empirical curve. Sufficient adherence to them for the purpose of this analysis is indicated both by the smoothness of the empirical curve (Fig. 1A) and by the general similarity of age-frequency distributions for species entering the four final stages (Fig. 1F). These represent the critical portion of the curve because the "shoulder" adjacent to the ordinate attests to the presence of few short-lived species. The technique used to obtain the histogram avoids the sources of error attributed to similar curves plotted directly from recorded stratigraphic ranges (*4*). From the standpoint of preservation, it requires only that the fossil faunas analyzed be good