



Fig. 2. A comparison of the amount of plain water drunk from tinted tubes by drug-treated quail (solid line) and saline-treated quail (dashed line). The amount drunk is expressed as a ratio of the amount ingested on a given day to the amount consumed on treatment day (TD).

illness avoidance learning. Thus, our main concern after the first experiment was whether the results for quail were unequivocal, rather than whether rats could actually see our visual cue.

In the second experiment we attempted to answer two questions: (i) Could the quail have been relying on some subtle taste of the dyed water rather than solely upon its appearance?; and (ii) Was the effective consequence that produced aversion to blue water really the drug-induced illness, or was it the considerable trauma of being caught, handled, and injected?

Birds from each of the five earlier subgroups were assigned to one of two groups, assignment being random except for the restriction that the groups be balanced with respect to prior treatment and test conditions. Procedural details were the same as in the first experiment. On treatment day, however, both groups drank from tinted blue tubes filled with the same plain water to which they were accustomed. One group ($N = 20$) was then injected with cyclophosphamide $\frac{1}{2}$ hour after drinking, whereas the other group ($N = 20$) was injected with normal saline.

Figure 2 shows the result. Birds that received the illness-inducing drug drank less from the tinted tube when they next encountered it ($P < .001$), whereas those injected with saline did not.

Although Figs. 1 and 2 give a clear picture of the relative changes in drinking occasioned by treatment-day and test conditions, they give no information on the absolute amounts ingested or the degree of variability. Accord-

ingly, means and standard deviations are shown in Table 1 for all groups each day from the last baseline day through the first extinction test. Comparison of baseline scores with those of treatment day shows that sour water, whether blue or not, was somewhat aversive to both species at first encounter, that is, before induction of illness; blue water alone was not. The amount of plain water drunk on the two recovery days after treatment shows a return to baseline levels. Effects of the delayed-illness conditioning trial are seen best by comparing scores of treatment day with those of the first extinction test.

Despite the controls introduced in the second experiment, it could be argued that the results represent not true associative learning but only the birds' increased wariness of strange-looking fluids as a result of recent illness. However, studies now completed in our laboratory (5) show that, although such sensitization or heightened neophobia contributes to the effect, there is a significant associative learning component as well. We are confident, therefore, that at least one avian species can associate a purely visual cue with a delayed illness without mediation by means of peripheral mechanisms such as reinstated taste.

It seems reasonable to expect that

this capacity will be widespread among animals whose visual systems are highly developed and whose niches demand great reliance upon vision in foraging. If so, the implications for ecology, behavior theory, and evolutionary theory are of considerable importance.

HARDY C. WILCOXON
WILLIAM B. DRAGOIN*
PAUL A. KRAL†

Department of Psychology,
George Peabody College for Teachers,
Nashville, Tennessee 37203

References and Notes

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* Present address: Department of Psychology, Auburn University, Auburn, Alabama 36830.

† Present address: Parsons State School and Hospital, Parsons, Kansas 67357.

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Atmospheric Aerosol Background Level

In their report Porch *et al.* (1) consider the existence of a background level of atmospheric aerosol, in which they derived a "scattering component of the extinction coefficient of air (b_{scat})" from atmospheric turbidity measurements made with a Volz sunphotometer at McMurdo Station, Antarctica (2). They compare b_{scat} values with the "component due to air with no aerosol (b_{Rayleigh}). The November–December 1966 value of this ratio at McMurdo Station, Antarctica, is 1.36, whereas the value for Mount Olympus, Washington, in February–March 1968 is 1.5 and for Point Barrow, Alaska, in March 1970 is 1.95 (1). Porch *et al.* speculate that the increase from 1968 to 1970 may "represent the cumulative effect of 2 years of aerosol production," but discard this hypothesis in favor of one invoking a difference in altitude between the last two stations.

In my report I stated that four stations in Antarctica showed turbidity values lower than those measured at McMurdo Station (2). The ratio $b_{\text{scat}}/b_{\text{Rayleigh}}$ for these four stations [(2), group 1 stations] is $1.24 \pm \begin{smallmatrix} +0.05 \\ -0.06 \end{smallmatrix}$, which is lower than the value for Antarctica used by Porch *et al.* and, in fact, is lower than any value in their table 1. If the Antarctic value of 1.24 and the Mount Olympus and Point Barrow values of 1.5 and 1.95, respectively, are plotted against the time of determination, they fall very nearly on a straight line; this result tends to support the idea that the differences are indeed due to the increase of atmospheric aerosols with time. The value for McMurdo Station of 1.36 lies noticeably above the time trend line. During the austral summer there is quite a bit of activity and fuel consumption at McMurdo Station; hence the station itself undoubtedly contrib-

utes to the measured turbidity, and the value of 1.36 is therefore bound to exceed the true background value. The four other Antarctic stations were located at elevations from sea level to 3000 feet (914 m), with no correlation between elevation and the ratio. These data thus suggest that altitude is not a factor.

Despite the probable increase in the atmospheric aerosol load, it is still possible today (as Porch *et al.* noted) to observe values of the ratio of $b_{\text{scat}}/b_{\text{Rayleigh}}$ very close to unity at remote stations. There is thus evidence of a high variability of aerosol loading at the low end of the scale, just as there

is frequent evidence of extreme variability at the high end of the scale at polluted urban stations.

In sum, it appears that an atmospheric aerosol background level exists and that it is increasing.

WILLIAM H. FISCHER*

State University of New York
Atmospheric Sciences Research Center,
P.O. Box 7185, Albany 12224

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- * Visiting scientist at the National Center for Atmospheric Research, Boulder, Colorado 80302.

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Acetylsalicylic Acid and Chromosome Damage

Mauer *et al.* (1) state: "No significant elevation of simple chromosome aberrations (breakage) was observed at any of the concentrations of ASA [acetylsalicylic acid; aspirin] tested. . . ." Their table 1 provides a list of ASA effects from 72-hour exposure at concentrations of 0, 1.0, 12.5, 25.0, and 50.0 $\mu\text{g}/\text{ml}$; the actual number of breaks they observed may be calculated. After combining data from all subjects, the concentration at 12.5 $\mu\text{g}/\text{ml}$ produces a significant increase in chromosome breaks (11/425 versus 18/1453 in controls; $t = 2.13$; $P < .05$). The data at this concentration are not homogeneous; subjects should be compared with their own controls. At this concentration only, subject M.L. had significantly more breaks than his baseline (4/108 versus 3/397; $t = 2.41$; $P < .05$) as did subject I.M. (4/104 versus 4/411; $t = 2.20$, $P < .05$). Thus two of three subjects showed significantly elevated chromosome breaks at 12.5 $\mu\text{g}/\text{ml}$, and Mauer's conclusion stated above is not wholly valid.

The clear discrepancy between their results and those of Jarvik and Kato (2) remains, and perhaps should be resolved. I examined the effects of ASA in an unpublished pilot study. There was a significant fourfold increase in simple chromatid gaps at 6.5 $\mu\text{g}/\text{ml}$ (17/107 versus 4/100; $t = 3.01$, $P < .01$). There was no increase in simple chromatid breaks, but two different chromosome-type aberrations were seen in treated cultures and none in controls. I believe this procedure not fully justified; but conversion of these to an "equivalent" number of breaks and

adding raises the "break" frequency to significance (8/107 versus 1/100; $t = 2.30$, $P < .05$). Higher and lower concentrations gave no evidence of significant effect. It seems to me that a suspicion remains, albeit a small one; but I am inclined to agree with Mauer *et al.* that ASA probably is not much of a cytogenetic hazard.

WILLIAM D. LOUGHMAN

Donner Laboratory, University of
California, Berkeley 94720

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In their report Mauer *et al.* (1) state that ". . . in the only cytogenetic investigation which, to the best of our knowledge, exists in the world literature, Jarvik and Kato . . . reported an average breakage frequency twice the control value . . . in normal human leukocyte cultures treated with 0.1 and 1.0 μg of ASA [acetylsalicylic acid] per milliliter of medium 4 hours before harvesting of cells. . . . We have been unable to confirm these results" (1, p. 199). A look at table 1 of their own report, however, reveals that in the three persons for whom the experimental conditions described by Jarvik and Kato (2) were duplicated (donors M.L., L.K., I.M.), the results of Jarvik and Kato were replicated remarkably well. When no ASA was added (first line) there were eight breaks in the 1200 metaphases counted, or 0.67 percent. When ASA

(either 0.1 or 1.0 $\mu\text{g}/\text{ml}$) was added, there were 12 breaks in 643 metaphases counted, or 1.87 percent. Thus, there was the doubling described by Jarvik and Kato.

The test used by Mauer *et al.* (1) for the statistical significance of the differences between the experimental and the control cultures is an insensitive one. They set 95 percent confidence limits on each proportion of broken chromosomes, found that the intervals almost invariably overlapped one another, and concluded that no significant differences existed.

One defect in this procedure is that the significance level for each comparison between an experimental culture and the control culture is necessarily less than the apparent 5 percent level (how much less depends on the proportions involved), making it unduly difficult to detect significant differences. The second defect is that no incorporation is made in the analysis of the consistency, if any, of the differences from one subject to the next.

A summary chi-square test due to Cochran (3) overcomes both of these defects. It keeps the significance level at .05, say, the desired probability, and incorporates any possible consistency of differences across subjects. Applying Cochran's test to the data of Mauer *et al.*, it is found that, even though none of the chi-squares for the three individual subjects is significant, nevertheless the summary chi-square, also with 1 degree of freedom, is significant ($\chi^2 = 5.15$, $P < .025$). The reason is that the individual differences, although not large enough to attain significance by themselves, are all in the same direction and of about the same magnitude.

Several investigators using a number of drugs, especially LSD, have failed to demonstrate a dose-related increase in breaks [see review by Moorhead *et al.* (4)]. With regard to ASA, Jarvik *et al.* (5, p. 1251) stated ". . . (0.1 and 1.0 mcg/ml-dosages were combined since the frequency of breaks was similar in both). . . ." Among the possible reasons for this apparent plateau may be a selective susceptibility of damaged cells to the very drug that kills them, yielding a spuriously low relative frequency of breaks at higher doses. Drugs such as streptozotocin may exhibit a more limited spectrum of toxicity than ASA and thus damaged cells might survive more readily in the presence of the former drug than in the presence of the latter.