4. A. R. Armstrong and H. E. Peart, *Am. Rev. Respirat. Diseases* **80**, 588 (1960).
5. R. S. Mitchell, D. K. Riemensnider, J. C. Bell, *ibid.* **80**, 108 (1959).
6. R. A. Knight, M. J. Selin, H. W. Harris, *Trans. 18th Conf. Chemo. Tuberc.* **19**, 21 (1960).
7. D. Levy, W. F. Russell, G. Middlebrook, *Tubercle* **41**, 23 (1960).
8. We are deeply indebted to Prof. Katsumi Tanaka, Department of Human Genetics, Tokyo Medical and Dental College, for valuable suggestions and cooperation, and to the medical staffs of the Central Chest Hospital, Thailand, the research Institute for Tuberculosis, Okinawa, and a number of national sanatoria in Japan for their assistance in collecting blood samples. The research was financially supported by the U.S. Army Research and Development Group (Far East), and we wish to express our sincere thanks to Colonel G. S. Gersoni.

12 May 1961

Preference for Clear versus Distorted Viewing in the Chimpanzee

Abstract. Young chimpanzees preferred to look through a clear window rather than through a window that produced a distorted image of viewed objects. Performance did not appear to be affected by familiarity with the viewing stimulus.

The tendencies to look, to manipulate, and to remain alert and on the move appear very early in life, and much of the daily activity of a primate consists of responses which keep the animal in touch with what is going on in the environment. Thus, for example, rhesus monkeys will work persistently to look out of an enclosed cage and see objects (*1*). The purpose of the experiment reported here was to determine whether chimpanzees, in addition to being motivated to look at objects, also prefer a clear to a distorted view of an object. Such a preference would be expected by Woodworth, who states, "the seeking of clear vision is built into the individual organism. It is an immediate drive of great potency without regard to any ulterior motivation. . . . All the visual mechanism [of clear vision] requires for its activation is the presence of visible objects. . . . What we said . . . in defense of an exploratory drive is pertinent here, for perception is evidently the core of exploration. The direct goal of exploration is to find or perceive 'what is there' "(*2*).

Four chimpanzees approximately 2½ years old (No. 188, Jenda; No. 173, Falweb; No. 175, Peck; No. 194, Saki) were given 28 5-minute trials, which were spaced over 7 days of testing. On each trial the subject was given access to two plastic windows (1½ by 2½ inches) mounted with their centers 12 inches apart in a wall of the home cage. One window afforded a normally clear view, but in the other the plastic was bent so as to produce (for humans) sharp but distorted images of viewed objects. By sighting through the latter window from different orientations, variations in the degree and type of distortion could be produced, but in general the distortions were gross. Double images and elongation of the visual image in multiple dimensions were the most frequently occurring types of distortion.

The positions of the clear and distorted windows were varied in balanced order from trial to trial. A seated human being, positioned 3 feet away from, and between, the two windows, served simultaneously as the viewing stimulus and as observer. By pressing a key he made a recording on a 2-channel operations recorder whenever the animal's face appeared in front of a window. To control for possible biases in response attributable to the presence of a particular person, two observers were used, a male and a female.

The amount of looking was scored from the recorder tapes by estimating the total number of marks representing 2 seconds spent by the subject in front of a window. By this criterion, the chimpanzees looked through one or the other of the windows about 30 percent of the total time; 65 percent of their looking was through the clear window. The clear window received higher scores than the distorted one on, respectively, 26 of 28, 22 of 26, 15 of 27, and 26 of 28 trials with the four animals (tied scores are excluded). For each animal except Peck (who had a position bias), $p < .01$ by sign test, and $p < .05$ by t test, with 3 degrees of freedom. "Testing" behavior—that is, moving rapidly back and forth before settling down for a time at the clear window—accounted for many of the responses to the distorted window.

It is unlikely that the viewing stimulus employed—a passive human being—produced the preference for clear viewing solely because it was familiar to or had special significance for these chimpanzees. Two of the animals (Jenda and Falweb) were raised for the first 21 months of life under special conditions that severely restricted their environmental and social experience. At the time of this testing they had had a total of less than 100 hours of social experience, and their social behavior was in many respects deficient. However, they performed in essentially the same fashion as did Peck and Saki, who were wild-born animals that had been in constant contact with other chimpanzees and with people since their arrival at the Yerkes Laboratories 1½ years prior to this experiment (*3*).

In view of human interest in relatively simple forms of "incongruous" stimuli (*4*), it is conceivable that the chimpanzee's preference for clear viewing could be reversed under some conditions. Under the conditions of this experiment, however, Woodworth's position is supported. Butler's recent and closely related finding that rhesus monkeys look more at a projected image that is in focus than at an image that is out of focus (*5*) is further evidence for the primacy of clear vision.

EMIL W. MENZEL, JR.
RICHARD K. DAVENPORT, JR.
Yerkes Laboratories of Primate Biology, Orange Park, Florida

References and Notes

1. R. A. Butler, in *Principles of Comparative Psychology*, R. H. Waters, D. A. Rethlingshafer, W. E. Caldwell, Eds. (McGraw-Hill, New York, 1960).
2. R. S. Woodworth, *Dynamics of Behavior* (Holt, New York, 1958), p. 192. See also H. W. Nissen, in *Handbook of Experimental Psychology*, S. S. Stevens, Ed. (Wiley, New York, 1951).
3. This research was supported in part by grant No. M-1005 from the National Institute of Mental Health, U.S. Public Health Service, and by a grant from the Ford Foundation.
4. D. E. Berlyne, *Conflict, Arousal, and Curiosity* (McGraw-Hill, New York, 1960).
5. R. A. Butler, *J. Genet. Psychol.* **98**, 239 (1961).

21 July 1961

Influence of Cage Type and Dietary Zinc Oxide upon Molybdenum Toxicity

Abstract. Molybdenum-fed rats housed in galvanized cages gained less weight and had lower hemoglobin levels than similarly fed rats housed in stainless steel cages. Since similar effects were produced in rats housed in stainless steel cages by increasing the zinc in their diets, it was concluded that zinc consumed by chewing on zinc-coated cages was responsible for the abnormalities noted.

Ever since the early work of Ferguson (*1*) the toxicity of molybdenum has been widely studied (*2–4*). Many of these studies have utilized rats as the experimental animals (*3, 4*), and it is likely that, in many of the studies with rats, the type of cage in which the animals were housed was not considered important. The experiments reported here were initiated as a result of the observation that rats housed in galvanized (zinc dipped) cages suffered more

Table 1. Some effects of cage type and dietary zinc oxide upon molybdenum-fed rats. Abbreviations: SS, stainless steel; Gal, galvanized.

Lot No.	Addition to basal diet	Cage type	6-wk wt. gain (g)	Hemoglobin concentration at 4 wk (g/100 ml)	Concentration in dry liver (μg/g)		
					Zn	Cu	Mo
	First experiment (average of five rats per treatment)						
1		SS	135	12.2	70	18.6	0.9
2		Gal	132	12.0	95	10.0	1.0
3	200 ppm Mo	SS	62	12.5	65	25.0	21.0
4	200 ppm Mo	Gal	41	9.8	125	42.0	37.0
	Second experiment (average of six rats per treatment)						
1′		SS	144	13.6	101	13.2	0.9
2′	0.05% ZnO	SS	119	12.3	314	8.0	1.1
3′	0.10% ZnO	SS	104	10.9	370	7.7	1.2
4′	0.15% ZnO	SS	82	10.2	339	7.2	1.4
5′	200 ppm Mo	SS	48	14.7	95	43.7	31.6
6′	200 ppm Mo + 0.05% ZnO	SS	25	12.7	145	51.4	52.2
7′	200 ppm Mo + 0.10% ZnO	SS	16	12.0	233	50.9	47.1
8′	200 ppm Mo + 0.15% ZnO	SS	16	12.1	296	45.7	48.1

from molybdenum toxicity than those housed in stainless steel cages.

The weanling rats used in these studies (Sprague-Dawley strain males) were individually housed in cages with either stainless steel or galvanized wire floors and were provided food and tap water *ad libitum*. The basal diet was essentially the same as one described elsewhere (*3*) and contained (in percent): sucrose 81.5, vitamin-free casein (Nutritional Biochemicals) 10.0, Wesson oil 5.0, mineral mixture 2.5, and vitamin mixture 1.0. Copper at the level of 1.6 parts per million (ppm) was added as copper sulfate to bring the total copper, as analyzed, to approximately 2.5 ppm. The zinc content of the basal diet was approximately 8 ppm. Hemoglobin was determined by the acid hematin method with blood obtained from the tip of the tail. After the rats were fed the experimental diets for 6 weeks, they were killed and the livers were removed and dried for analysis. Copper was determined by the carbamate method (*5*), molybdenum by the thiocyanate method (*6*), and zinc by the dithizone method (*5*).

In the first experiment (lots 1 to 4) the rats were housed in stainless steel or galvanized cages and were fed either 200 ppm molybdenum, added as sodium molybdate, or none at all, as indicated in Table 1. The galvanized cage environment resulted in a significant ($t > t_{.05,8}$) reduction in blood hemoglobin concentration, a highly significant ($t > t_{.01,8}$) reduction in weight gain, and a highly significant ($t > t_{.01,8}$) increase in concentrations of molybdenum and copper in the livers of the animals fed molybdenum, when comparison is made with animals receiving molybdenum but housed in stainless steel cages (lot 3 compared to lot 4). The zinc level in the livers of the rats fed molybdenum and housed in galvanized cages was approximately twice the level in the livers of those fed molybdenum and housed in stainless steel cages. This difference may not be real, however, because of variability within treatments.

In the second experiment (lots 1′ to 8′) the rats were housed in stainless steel cages, and three different levels of zinc oxide and one level of molybdenum were added to the basal diet of the seven treatment groups as indicated in Table 1. The rats fed 0.05 percent zinc oxide had a weight gain of 83 percent of the controls, while those fed 200 ppm molybdenum and 0.05 percent zinc oxide had a weight gain of only 52 percent of the molybdenum-fed rats. Statistical analysis showed there was a significant interaction between the molybdenum and zinc oxide treatments when measured by weight gain ($F > F_{.05,3,39}$). This would substantiate the report that there is an interaction between molybdenum and zinc (*7*). However, statistical analysis further showed there was no significant interaction between molybdenum and zinc oxide upon hemoglobin levels or copper concentration in the liver. That dietary zinc at a relatively low level (0.75 percent) is toxic has been established (*8*). In addition, G. Matrone (*9*) has indicated that zinc at the level of 0.05 percent in the diet was toxic for the rat.

The lowest level of zinc oxide fed with the molybdenum in the second experiment (0.05 percent) probably furnished more zinc oxide than the molybdenum-fed rats in the first experiment obtained from chewing on the galvanized cages. Food consumption records indicated that the rats in lot 6′ consumed approximately 6 g of food per day (between the 3rd and 5th weeks) which provided a daily zinc oxide intake of approximately 3 mg. Although it is conceivable that a rat could obtain this amount of zinc oxide by chewing on a galvanized cage, it is unlikely that as much as 3 mg/day was consumed by the animals housed in zinc-coated cages during the first experiment, since their growth was more rapid than the growth of the animals in the second experiment, which were fed the same diets, plus zinc oxide, but were housed in stainless steel cages. Although the possibility remains that the increased toxicity of molybdenum was due to the presence of some contaminant other than zinc on commercially produced galvanized metal cages, it appeared that the zinc from the galvanized cages was responsible for this effect.

It is evident then that in the future when molybdenum, copper, or zinc studies are being conducted with animals in cages, the type of cage used should always be considered, as has been noted when other trace minerals were studied; perhaps some earlier experiments should be reevaluated in this light (*10*).

G. L. Brinkman*
Russell F. Miller
Department of Biochemistry and Nutrition, Virginia Polytechnic Institute, Blacksburg

References and Notes

1. W. S. Ferguson, A. H. Lewis, S. J. Watson, *Nature* **141**, 553 (1938).
2. A. T. Dick, *Australian Vet. J.* **30**, 196 (1954).
3. R. F. Miller, N. O. Price, R. W. Engel, *J. Nutrition* **60**, 539 (1956).
4. C. F. Mills, K. J. Monty, A. Ichihara, P. B. Pearson, *ibid.* **65**, 129 (1958).
5. Association of Official Agricultural Chemists, *Official Methods of Analysis* (AOAC, Washington, D.C., ed. 9, 1960), pp. 80, 333.
6. H. J. Evans, E. R. Parvis, F. E. Bear, *Anal. Chem.* **22**, 1568 (1950).
7. L. F. Gray and G. H. Ellis, *J. Nutrition* **40**, 441 (1950).
8. A. C. Magee and G. Matrone, *ibid.* **72**, 233 (1960).
9. G. Matrone, personal communication.
10. The above work was supported in part by a grant from the Nutrition Foundation, Inc., N.Y.

* Cooperative fellow, National Science Foundation.

29 May 1961