shows that adaptation to a grating at this frequency will make the spatial frequency channels less responsive to the high-frequency components of the rectangle and should make the rectangle appear wider; in fact, in our experiment this adaptation did produce perceived widening (as opposed to no change as predicted by the size-specific model). The center arrow shows a possible equilibrium grating bar size. Although adaptation should reduce sensitivity at this frequency, there should be no subsequent size changes; none were observed. The arrow at the left shows that adaptation to a grating at this frequency (bars eight times wider than the rectangle) should make the spatial frequency channels less responsive to the low-frequency components of the rectangle, and should make the rectangle appear narrower; this adaptation produced the predicted narrowing.

Thus, in our investigation of how prior viewing of a grating affects the perceived size of a rectangle, we found support for the hypothesis that the visual system codes size on the basis of the spatial frequency components rather than the width of the stimulus per se (*13*).

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4. I. D. G. MacLeod and A. Rosenfeld, *Vision Res.* **14**, 909 (1974); J. P. Thomas, *Psychol. Rev.* **77**, 121 (1970).
5. A single bar has a continuous frequency distribution proportional to $(\pi f w)^{-1} \sin(\pi f w)$. For simplicity of exposition the effect of finite grating size is not discussed, nor are harmonics mentioned.
6. T. N. Cornsweet, *Am. J. Psychol.* **75**, 485 (1962).
7. All ten subjects observed widening up to a ratio of 4:3. Horizontally oriented gratings used as adapting stimuli yielded essentially the same results: After adaptation, the rectangle appeared broader on the dimension orthogonal to the bars of the grating; in other words, the rectangle appeared taller.
8. Eleven additional subjects have shown similar shifts when run under somewhat different conditions [F. S. Frome, C. S. Harris, J. Z. Levinson, *Bull. Psychon. Soc.* **6**, 433 (1975)].
9. In experiment 1 the height of the adapting stimuli was the same for all spatial frequencies.
10. Similar results were obtained for two other observers and for both 10′ and 7.5′ rectangles.
11. It is not obvious what to call this or other aftereffects. In order to relate our findings to a wide variety of previous ones, we have referred to our results interchangeably as a size or width aftereffect. Further tests would be necessary to determine if the aftereffect reported here is a shift in perception of size, shape, height, width, squareness, extent, or some still-undetermined factor.
12. D. H. Kelly has noted (personal communication) that on a log-frequency plot, the rectangle's spectrum extends endlessly toward lower frequencies. It would thus appear impossible to find an effective adapting frequency lower than the "center of gravity" of such a distribution. Weighting the rectangle's spectrum by a modulation transfer function bounded on the lower frequency end would provide a solution, but one that raises the question of the appropriate frequency scale altogether. In thinking of a spectrum, one may use any arbitrary frequency scale for reasons of convenience, mathematical simplicity, or physical or psychological relevance. Because we do not yet have a rational choice, there is no way to make this discussion more quantitative.
13. For other kinds of data consistent with a similar conclusion, see, for example, N. Weisstein and J. Bisaha [*Science* **176**, 1047 (1972)] and C. S. Harris [*J. Opt. Soc. Am.* **61**, 689 (1971)].
14. F.S.F. was employed by the National Institute of Mental Health Section on Perception early in the course of this work. Data from the first experiment were collected at the University of Maryland and the University of Massachusetts and were reported at the 1974 annual meeting of the Association for Research in Vision and Opthalmology. Data from the second experiment were collected at the University of California at San Diego and were reported at the 1976 annual meeting of the Optical Society of America. This report was written at SRI International and Bell Laboratories. Partially supported by NIH grant EY01640 to J.Z.L. and NIH research fellowship award EY05116 to F.S.F. We thank N. Weisstein for suggesting the conditions which produce apparent narrowing, D. H. Kelly for discussing appropriate frequency scales, and the late Brian J. Murphy for support and useful suggestions throughout the course of this work. We also thank C. S. Harris for his helpful suggestions on the manuscript.
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8 February 1978; revised 13 August 1979

Genetic Resistance to Aflatoxin in Japanese Quail

Abstract. *Progress was rapid in attempts to develop lines of quail resistant to acute aflatoxicosis induced by oral dosing with aflatoxin. After five generations of selection, 8- and 11-fold differences were present in mortality between two selected lines and their respective control lines. These quail lines should be of value in investigating the physiological basis of resistance to aflatoxin.*

Aflatoxin, a carcinogenic metabolite, is produced by the filamentous fungi *Aspergillus flavus* and *Aspergillus parasiticus* (*1*). Because this mycotoxin contaminates a number of feedstuffs that enter into the food chains of humans, its long-term influences on human health are of concern. The economic impact of aflatoxin is evident from reduced growth and productivity resulting from ingestion of this mycotoxin by domestic animals.

Genetic variation for resistance of animals exposed to aflatoxin is suggested by differences noted between the New Hampshire and other breeds of chickens (*2*). Through artificial selection, it may be possible to not only demonstrate genetic variation to aflatoxin, but also to develop populations of domestic animals resistant to the adverse effects of aflatoxin. Such animals would be useful for the investigation of the physiological differences between resistant and nonresistant populations and serve as a model to study the mechanism of aflatoxin toxicity within the organism. The objectives of our study were to investigate the genetic factors of selection for resistance to aflatoxin with the Japanese quail as a model and to develop resistant lines for determining systems influenced by the selection process.

Aflatoxin was produced on polished rice (*3*), with incremental increases in temperature during the incubation period (*4*). After the rice was autoclaved to kill the mold, it was ground to a fine powder, and the aflatoxin was extracted from the substrate (*5*) and quantified by high-pressure liquid chromatography. The fer-

Table 1. Mortality of Japanese quail from acute aflatoxicosis by line (control and selected) and generation.

Generation	Aflatoxin at 2.5 mg/kg					Aflatoxin at 3.0 mg/kg				
	Control		Selected		Ratio†	Control		Selected		Ratio†
	*N**	Mortality (%)	*N**	Mortality (%)		*N**	Mortality (%)	*N**	Mortality (%)	
S_1	65	40.0	139	14.4	2.8	74	71.6	121	39.7	1.8
S_2	91	72.5	179	36.9	2.0	87	88.5	157	42.0	2.1
S_3	119	79.0	152	22.4	3.5	100	97.0	163	35.6	2.7
S_4	69	78.3	129	11.6	6.8	69	78.3	185	12.4	6.3
S_5	75	75.0	175	6.3	11.9	70	82.9	180	10.0	8.3

*Total number of birds challenged. †Control mortality:selected mortality.

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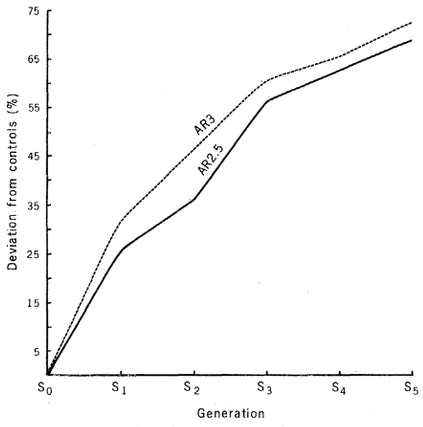

Fig. 1. Mortality deviation of selected lines from control lines.

mented rice contained 2.95 mg of total aflatoxin per gram (90.1 percent B_1, 8.6 percent G_1, 1.1 percent B_2, and 0.2 percent G_2). Weighed amounts of the fermented rice were mixed with deionized H_2O to yield a concentration of either 250 or 300 μg/ml. In all experiments, a measured amount (1 ml per 100 g of body weight) of the resulting slurry was administered to each bird by intubation through the crop. All birds were 29 days old at the time of aflatoxin dosing. The quail used were from a nonselected random-bred control population. The base generation (S_0) consisting of 250 quail were given 3.0 mg of aflatoxin per kilogram of body weight. Mortality was recorded for 5 days after treatment. Normally mortality at this age (4 weeks) is extremely low, therefore, all mortality during this 5-day period was considered due to acute aflatoxicosis. Surviving quail in the S_0 generation were used as breeders (30 pairs) to produce progeny for the S_1 generation. Quail from the random-bred population were reared intermingled with S_1 generation quail and served as nonselected controls. Quail from hatch 1 in the S_1 generation were given 2.5 mg of aflatoxin per kilogram of body weight. Two lines were therefore established, one selected on the basis of a challenge of 2.5 mg of aflatoxin per kilogram of body weight (AR2.5) and a second selected with a challenge of 3.0 mg/kg (AR3). The selected lines (AR2.5 and AR3) in the S_2 generation were produced by 36 pairs per line. Where more survivors were available than needed for breeders in these lines, family selection was used to identify survivors from the more resistant families. Reproduction of lines occurred approximately 6 to 8 weeks after the aflatoxin challenge in all generations.

Mortality from challenge with aflatoxin at 3.0 mg/kg in the S_0 generation was 76 percent. Mortality in the S_1 generation of control birds dosed with 3.0 mg/kg was similar (71.6 percent), but the mortality in selected AR3 birds was 39.7 percent (Table 1). Mortality of selected and control quail dosed with 2.5 mg/kg in the S_1 generation was approximately one-half the mortality of quail dosed with 3.0 mg/kg (Table 1). Mortalities for both control lines in the S_2, S_3, S_4, and S_5 generations were higher than mortality in the S_1 generation, perhaps as a result of different ambient temperatures during and after induction of aflatoxicosis (*6*). Correction for environmental variations between generations was accomplished by plotting the selected lines as deviations from the control (Fig. 1). The greatest deviation occurred in the S_1 generation, indicating an immediate selection response. Larger deviations of selected lines from the controls in the S_2, S_3, S_4, and S_5 generations show continued genetic improvement in reducing mortality. The ratios of control line mortality to selected line mortality (Table 1) show that, after a single generation of selection, the difference between mortality of the selected and the control lines was twofold and after the fifth generation of selection, the difference was 8- to 11-fold.

After the fourth generation of selection (S_4), reciprocal crosses were made between the selected lines and the base control line. Two replications (hatches) were produced in order to evaluate the resistance of progeny to acute aflatoxicosis. The administration of the aflatoxin stress was identical to the procedure used in the development of the AR2.5 and AR3 lines. The mortality levels of the controls (C × C) was three to four times higher than the mortality levels of the cross progeny (for example, AR2.5 × C and C × AR2.5). The similar responses of the reciprocal crosses is supporting evidence for genetic change and negates the possibility of extranuclear factors passed through the eggs being the basis for the resistance observed in the AR2.5 and AR3 lines. Although the crosses had higher mortalities than the pure lines (AR2.5 and AR3), they more closely approximated these lines than the control.

The rapid initial response to selection shows that relatively few loci may be involved in resistance to aflatoxin in quail. Because cellular resistance to Rous sarcoma viruses is controlled by a limited number of autosomal loci (*7*), the resistance of quail to aflatoxin may involve a similar mechanism that interferes with the transport of aflatoxin into the liver or its subsequent metabolism. Resistant and nonresistant lines in other species may be useful as a system to determine the role of genetic resistance in the production of the carcinogenic metabolite aflatoxicol (*8*).

Table 2. Mortality of pure line and reciprocal crosses to acute aflatoxicosis. C designates the nonselected control line. AR2.5 and AR3.0 designate the lines selected after dosing with 2.5 and 3.0 mg of aflatoxin per kilogram of body weight, respectively.

Aflatoxin (mg/kg)	Line (male × female)	Mortality (%)		Mean
		Exp. 1	Exp. 1	
2.5	AR2.5 × AR2.5	0.0	4.0	2.0
	AR2.5 × C	8.0	19.6	13.8
	C × AR2.5	14.0	32.0	23.0
	C × C	47.4	75.6	61.5
3.0	AR3 × AR3	2.0	8.0	5.0
	AR3 × C	18.0	28.0	23.0
	C × AR3	14.0	18.4	16.2
	C × C	68.6	85.4	77.0

These data show that rapid progress is possible in developing lines of quail resistant to acute aflatoxicosis induced by oral dosing with 2.5 and 3.0 mg of aflatoxin per kilogram of body weight. Lines resistant to aflatoxin should be useful in investigating the mode of action of aflatoxin. These lines could provide a model for testing differences in the metabolic systems between resistant and nonresistant lines. This information may offer new insights into techniques to eliminate the adverse effects of aflatoxin on animal production and its possible relation to human health.

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20 October 1978; revised 3 October 1979