They determined the erosional state of craters in the size range 100 m to kilometers and converted the erosion data to crater frequencies, possibly erroneously [figures 1 and 14 in (2) are inconsistent]. This is discussed by G. Neukum (*Moon*, in press).
12. The meaning of the Apollo and Luna radiometric ages for the lunar cratering record and of terrestrial age data for the North American cratering record is discussed by Neukum *et al.* (5) and G. Neukum and P. Horn (*Moon*, in press).
13. G. Neukum, B. König, J. Arkani-Hamed, *Moon* **12**, 201 (1975).
14. The constant power law distributions also shown in Fig. 2 for comparison are taken from Baldwin (3), Hartmann and Wood (16), and Shoemaker *et al.* (15).
15. E. M. Shoemaker *et al.*, *NASA Spec. Publ. SP-325* (1970).
16. W. K. Hartmann and C. A. Wood, *Moon* **3**, 3 (1971).
17. G. Neukum and D. W. Wise, *NASA Tech. Memo. TM X 74316* (1976).
18. V. R. Oberbeck and R. H. Morrison, *Moon* **9**, 415 (1974).
19. C. R. Chapman, *Icarus* **22**, 272 (1974).
20. M. H. Carr, *NASA Tech. Memo. TM X 3364* (1976), p. 152.
21. K. L. Jones, *J. Geophys. Res.* **79**, 3917 (1974).
22. The polynomial fit of the standard curve is (for the age of the Alba cone)

$$[\log N = a_0 + a_1 \log D + a_2(\log D)^2 + a_3(\log D)^3 + a_4(\log D)^4]$$

with $a_0 = -2.605$, $a_1 = -2.998$, $a_2 = 0.578$, $a_3 = 0.637$, $a_4 = -0.498$, N = cumulative crater frequency per square kilometer, and D = crater diameter in kilometers.
23. We have not distinguished in our counts between single impacts and crater doublets produced by broken-up projectiles [V. R. Overbeck and M. Aoyagi, *J. Geophys. Res.* **77**, 2419 (1972)]. The effect of doublets on the size-frequency curve is considered minor because of their relative rarity (19).
24. The asteroids with perihelions inside the earth's orbit are called Apollo asteroids, and those with perihelions between 1 and 1.3 A.U. Amor asteroids. Those with perihelions (crossing Mars' orbit) greater than 1.3 A.U. are called Mars crossers. The relative impact velocities have been given (9).
25. Shock theory would suggest the law $D \propto v^{2/3}$ for constant mass, where D is the crater diameter and v is the impact velocity. Chemical explosion data [M. D. Nordyke, *J. Geophys. Res.* **66**, 3439 (1961)] show a variation of crater diameter as the 1/3.4 power of energy expended. Use of this relationship gives essentially the same results as use of the 1/3 power.
26. Soderblom *et al.* use impact flux (mass-related) as synonymous with cratering rate (diameter-related), and thus do not account for any velocity differences. In fact, they do not set the cratering rate at Mars equal to that at the moon over the whole past. In their crater chronology diagram, the martian crater frequency (time integral of impact rate) is about a factor of 1.5 higher than the lunar frequency for ages > 2 billion years and about the same as the lunar frequency for ages < 1 billion years.
27. D. E. Gault, *Radio Sci.* **5**, 272 (1970).
28. C. R. Chapman and R. R. Haefner, *J. Geophys. Res.* **72**, 549 (1967).
29. J. B. Pollack *et al.*, *Icarus* **17**, 394 (1972). These authors considered the Phobos crater population saturated. We do not agree with respect to the larger craters ($D > 2$ km) because they lie far apart from each other.
30. The crystallization ages of meteorites also fall in this age span. It is quite possible that some of the meteorites stem from asteroid-type bodies that are similar in size to Phobos. The meteorite ages reflect the time of solidification of these bodies. This is a corroborating argument for solidification of Phobos 4.5 to 4.6 billion years ago.
31. We have not corrected Hartmann's original martian cratering frequency data given for a crater size distribution law to the –2 power in our reduction to $D = 1$ km. The velocity effect in going from large crater sizes to 1 km would lower Hartmann's values by a factor of about 2.
32. H. J. Moore, *U.S. Geol. Surv. Prof. Pap. 812-B* (1976).
33. S. W. Johnson, J. A. Smith, E. G. Franklin, L. K. Moraski, D. J. Teal, *J. Geophys. Res.* **74**, 4838 (1969).
34. E. M. Shoemaker, paper presented at the Planetary Program Principal Investigators meeting, Flagstaff, Ariz. (1976).
35. For a distribution law $N = f(D)$, $N \propto \delta N$. The data of Soderblom *et al.* in Fig. 1 are the original data in terms of δN, and those of Neukum *et al.* are the original data in terms of N. The relation between N and δN used here is derived from the calibration distribution of Fig. 2 (13): $N(D = 1$ km$) = 96\ \delta N$. Baldwin originally gave his data as cumulative crater frequencies N for craters ≥ 161 km in diameter. We used his distribution law $N \propto D^{-1.8}$ for reduction to $N(D = 5$ km) and the calibration distribution for reduction to $N(D = 1$ km). Hartmann gave his crater frequency data originally "in arbitrary units relative to an average mare frequency." We used his Apollo 14 (Fra Mauro) counts for absolute reduction to $N(D = 1$ km) in applying our calibration distribution. In this way, all data have been reduced by application of a consistent method and are directly comparable.
36. This work was supported by the Deutsche Forschungsgemeinschaft, and the Planetology Office of the National Aeronautics and Space Administration, and was completed while D. U. Wise was a guest scientist at the Max-Planck-Institut für Kernphysik, Heidelberg. B. König was most helpful in the data reduction. Discussions with R. Arvidson, K. Blasius, M. Carr, J. Cutts, R. Greeley, J. Guest, H. Moore, R. W. Shorthill, and J. Veverka helped clarify our ideas.

The Enigma of Radiation Effects in *Drosophila*

Linear relation between induced mutation and x-ray dose and related inconsistencies are discussed.

E. Novitski

It is very well known that the frequency of radiation-induced mutation in *Drosophila* is essentially linear with dose, from very low doses up to at least 5000 or 6000 roentgens (r units). What is not so widely appreciated is that there exists a number of inconsistencies in the theory of radiation-induced mutation and chromosome breakage in *Drosophila*. For instance, the apparently simple and almost self-evident linear relationship of mutation with dose is considered both perplexing and unsolved. A simple solution to some of these problems is proposed in this article.

Even the earliest radiation studies with *Drosophila* indicated that induced sex-linked recessive lethals may be associated with chromosome rearrangements (1) and that the percentage of such lethals increases with dose (2–4). From these findings it has been concluded that such lethals are produced predominantly, if not entirely, as a result of chromosome breakage (3, 5–7). The most precise formulation of the relation between breaks and lethal mutations was proposed by Lea and his colleagues (5–7). However, at that time, Fano pointed out (8) a serious and inexplicable flaw in their reasoning, which, in essence, is the following. For every viable rearrangement of the simplest two-break type, there should exist an inviable, dicentric type which should be lethal. These nonrecoverable rearrangements should cause a loss of the induced sex-linked lethals, leading to a depression from linearity of about 20 to 30 percent at 3000 r units. Furthermore, with increasing dosage the frequency of inviable types should go up markedly, not only because of the increase of two-break arrangements with a power of the dose greater than 1, but also because of the appearance of three-break and higher order events, of which a smaller proportion form viable rearrangements. Thus, the depression at higher doses should be even more extreme. Such a departure from linearity is not at all borne out by the existing data. In fact, if anything, the data obtained by Edington (9) in a carefully controlled experiment over a wide dose range shows a slight but significant excess of induced lethals at the higher doses.

Herskowitz (10) demonstrated that no simple combination of hypotheses of le-

The author is professor of biology at the University of Oregon, Eugene 97403.

thal origin adequately explain the linear relationship, and he suggested that a combination of three different origins—point mutation, lethals associated with chromosome breakage, and position effect lethals—might produce a linear relationship, with the higher frequency of position-effect lethals associated with rearrangements at the higher doses compensating for the loss of breakage-associated lethals lost in inviable rearrangements. By a judicious selection of proportions of the three types, he was able to reconstruct a linear relationship of induced mutations to dose consistent with the amount of chromosome breakage theoretically proposed by Lea and co-workers (*5, 7*) for those doses. In whatever way one might regard the likelihood that there would exist by chance two nonlinear relationships, of opposite curvature, which, when added together, would closely simulate an approximate linear relationship, the fact remains that this has been the best (and only) explanation of the linearity. Furthermore, it might be mentioned that the class of position-effect lethals, hypothesized to occur with substantial frequencies at high doses in order to straighten out the curve, is one for which, even now, there is no independent evidence. Nevertheless, in the absence of any better explanation, most workers have felt compelled to accept this one.

Induced Mutations in Rings

Another bothersome inconsistency concerns the frequency of induced sex-linked lethal mutations in ring chromosomes. In those mathematical models already referred to, relating chromosome breakage (as evidenced by dominant lethality, chromosome rearrangements, and chromosome loss) and induced lethal mutation, a basic assumption is that the daughter chromatids of a broken chromosome, after replication, may either restitute, or the two sister strands may rejoin with each other in reverse order, or they may "rotate through half a revolution relative to one another before rejoining" (*7*) to form the equivalent of a sister strand exchange. The last event, producing a dicentric in a ring chromosome (but not in a normal rod-shaped chromosome), would lead to a loss of rings undergoing such "sister strand" type events amounting to half of the total of the recoverable breakage-associated lethals. Muller and Offermann (*11*) were the first to report that ring chromosomes, after irradiation, carried about the same frequency of induced lethals as rods—not half the frequency—and this result has been confirmed on several occasions, with the use of rod chromosomes derived originally from rings in order to minimize any extraneous differences between the chromosomes other than their conformation (*9, 12*). It is interesting to note that Lea, in his masterful synthesis of the *Drosophila* data (*7*), after concluding on the one hand, that 50 percent of all broken ring chromosomes must be lost by dicentric formation and, on the other, that induced lethal mutations result from radiation induced breaks, failed to make the further logical deduction that the induced lethal frequency in rings must be depressed, and, since it was not, that there was necessarily a serious flaw in his model.

Still another problem arises in connection with the high frequency of whole-body mutations after irradiation. The substance of the dilemma can best be understood by considering, first, what happens when the sperm of *Drosophila* males are treated with a chemical mutagen and their progeny are examined for visible mutants. A large proportion of the mutants recovered appear as mosaics, or fractionals; this is interpreted as the consequence of a local alteration of one of the two DNA strands, the mutated strand and its descendants when appearing in approximately half of the cells of the resulting individual. Because of the indeterminate nature of development in *Drosophila*, the mutated strand may be found in as many as all, or in as few as none, of the cells capable of revealing the phenotypic change. Nevertheless, after treatment with most chemical mutagens, visible mutational changes generally appear as mosaics.

This is not the case, however, for x-ray induced visible mutants, which appear predominantly as whole-body mutants, suggesting that all the cells making up the fly carry identically mutated chromosomes. How this might happen is not at all clear. Muller *et al.* (*13*) have proposed that, when visible mutational changes are induced by x-rays, excitation by a secondary electron track causes a base pair to be rotated through 180° so that precisely complementary changes are found on both strands of the DNA, which, after replication, would lead to two identically changed strands. In this way, all of the cells of the resulting individual would have the same mutational change. This interesting hypothesis, advanced at that time as the explanation for virtually all radiation-induced mutation in *Drosophila*, is not supported by our current knowledge of the mechanism of DNA mutations (*14*). That it should be suggested at all testifies to the fact that there exists a real puzzle with no simple or obvious solution.

I shall show that all of these questions may very well be answered by simply assuming that normal development of *Drosophilia* may proceed from only one of the two first-cleavage nuclei, and that this is generally true if the other nucleus carries a dominant lethal change. However, it may be worthwhile first to give in detail the reasons for advancing toptipotency of the first-cleavage products in the *Drosophila* egg as a viable hypothesis.

The first clue came from the results of an experiment designed to attach the sex-linked markers *yellow*$^+$ and *Bar* autosomal tips for use in studies of somatic crossing over. For this purpose, my coworkers and I (*15*) irradiated a doubly marked Y chromosome, synthesized by Brosseau (*16*), which had these two markers, one at each tip; and, by mating the irradiated males to attached-X females, we recovered the radiation-induced chromosomal changes in F_1 females. Altered Y chromosomes could be readily detected and perpetuated since the Y chromosome is not essential for either normal development or fertility of the female (*17*). The surprising result was that the exceptional progeny, with one or the other of the terminal markers, but not both, included a high proportion of mosaics (15 out of 58).

Mosaicism for x-ray induced breaks has been reported as isolated cases many times previously and in itself is not a cause for undue interest. What was surprising was the high frequency of its occurrence in an experiment of a type never performed previously, where a chromosomal mosaic could be readily identified by the phenotype of the individual carrying it, without affecting either its viability or fertility, and where the chromosome involved could be captured and subjected to genetic tests subsequently.

The fact of mosaicism was itself evidence that the two daughter chromatids separating at the first-cleavage division were behaving independently; and, if this were a general phenomenon, there is no reason why in other cases one of the two daughter nuclei might not carry a dominant lethal change (as a dicentric), while the other might be completely viable. It was pointed out (*18*) that this might account for the preponderance of whole-body visible mutations in radiation experiments, that most of the viable

offspring actually resulted from cases where development had proceeded from only one of the two first-cleavage products, the other carrying a dominant lethal change (such as broken but unrestituted chromosomes, dicentrics, or gross deficiencies), with the result that the possibility of mosaicism was wiped out.

The results from a completely different kind of experiment have led Leigh and Sobels (*19*) to precisely the same conclusion. After irradiating mature sperm, they were able to recover new compound chromosomes, chromosomes that consisted of two sister chromatids of an autosomal arm attached to each other. From the manner in which the experiment was set up to recover these compounds, it was clear that they would be found as balanced gene complements initially in only one of the two first-cleavage products, the other being lethal. The appearance of viable exceptions carrying newly induced compounds was convincing evidence that a normal fly could be produced from one of the cleavage products when the other was lethal.

The Hypothesis of Totipotency

This hypothesis of the developmental competence of a single first-cleavage product after loss of the other by its involvement in chromatid rearrangements suggests that interchanges predominantly involve the chromatids within each of the two nuclei, and not between the two nuclei; otherwise we should not see the vast majority of new rearrangements as modifications of a single haploid set. The random breakages and reunions of chromosomes between two haploid sets would regularly give rise—particularly in F_1 larvae which will better tolerate genetic unbalance—to a hodgepodge of rearrangements with duplications of sister chromatid material that could not be misinterpreted. This leads to the conclusion that the early interpretation of dicentric formation after breakages of ring chromosomes is incorrect, that broken sister chromatids do not as readily participate in a sister strand type exchange as they do in an ordinary restitution. If the basis for expecting a halving of the lethal frequency in ring chromosomes is incorrect, the greater overall loss of ring (compared to rod) chromosomes after radiation, which originally led Bauer (*20*) to postulate dicentric formation, must be explained in some other way. In fact, Bürki has already postulated other reasons for radiation-induced ring loss (*21*).

On the basis of the preceding arguments, several reasons can be considered why the curve of induced sex-linked lethals plotted as a function of dose does not drop sharply from the simple expectation, as Fano (*8*) suggested it should. In the first place, there are three possible fates for a newly induced X chromosome break which has an associated lethal. After replication, the two first-cleavage products will carry a broken chromosome and in each of them the broken chromosome may (i) restitute, (ii) become involved in a viable rearrangement, or (iii) fail to restitute or may otherwise become involved in an inviable rearrangement. If these choices are independent in the two nuclei, then if one of those two products carries an inviable rearrangement, as in (iii) above, it simply dies off and is "replaced" by the other totipotent cell, of type (i) or type (ii). It is only in those cases where both products carry an inviable rearrangement involving the X chromosome that sex-linked lethals would be lost. Using Fano's figures (*8*) of an expected depression of 0.2 to 0.3 at 3000 r units from inviable rearrangements, we would get instead a depression of $(0.2)^2$ to $(0.3)^2$ or 0.04 to 0.09, deviations that would be much more difficult to detect experimentally, even if they were responsible for the only perturbation from the simplest expectation.

However, a second contribution must be taken into account, one that will tend to impart an upward swing to the curve at higher doses. Some small fraction of x-ray induced lethals must involve local changes on only one of the two DNA strands, this, then, appearing in a mosaic. Since lethal tests generally rely on the complete absence of normal chromosomes in the gonads, such lethal mosaics will more often than not be classified as nonlethal. However, as the dose increases, the chance that both strands will independently acquire such lethals increases as the square of the dose; and these mosaics, half of whose cells carry one lethal and half another, will always be classified as lethal-bearing. In addition, a more substantial contribution may come from a third effect. At low doses, the mosaic lethals will often be misclassified as nonlethal because of gonadal mosaicism. As the dose increases, so does the frequency of chromatid rearrangements involving all the chromosomes of the complement. At least half, and usually more, of these will form inviable combinations, killing off that first-cleavage product and eliminating the possibility of mosaicism, since this type of mosaicism requires two viable products. In this way, a potential mosaic for a lethal, with a high probability of being classified as normal, is converted into a nonmosaic, with a certainty of being classified as lethal.

Totipotency, the capacity for development of a complete individual from one of the early cleavage products, is a long-held embryological concept; its relevance to *Drosophila* embryogenesis has only recently been established (*22*). Unfortunately, these observations have been appreciated primarily by developmental geneticists, and their possible significance to the study of mutagenesis has been largely unrecognized.

Furthermore, apart from theoretical arguments of the sort presented here, evidence for it comes from the appearance of isolated exceptions in specialized experiments, generally of little interest to (and with less impact on) anyone except those doing the experiments. I have attempted several different kinds of tests to try to verify totipotency without success. Perhaps the most convincing test would have an experimental design that would predict an otherwise incomprehensible result and, to this end, I have thought that the predicted decline in frequency of mosaic types with increasing dose of radiation would serve such a purpose. Accordingly, Carlson and I (*23*) have checked the frequency of chemically induced mosaics at the dumpy locus, with and without subsequent radiation, and have found no decrease after radiation, contrary to this theory. In contrast, I would like to call attention to a different sort of experiment, also involving induced mosaics, carried out by Inagaki and Nakao (*24*). They measured the frequency of whole-body mutations at the yellow, white, miniature, and forked loci at doses from 1000 to 4000 r units, in intervals of a thousand, and obtained frequencies of 0.047, 0.119, 0.234, and 0.330. However, the frequency of fractionals stayed at a low level for all doses, so that the ratio of fractional changes to whole-body changes, over this dose range, was 1 : 1, 0.46 : 1, 0.23 : 1, and 0.16 : 1, a shift with increasing dose of precisely the type predicted by totipotency.

In conclusion, it is clear that the hypothesis of totipotency of the cleavage products of *Drosophila* explains many hitherto puzzling results from radiation experiments. Possibly, a more profitable approach in assessing its validity in interpreting mutagenic effects in *Drosophila* will come from developmental, rather than radiation, studies.

References and Notes

1. H. J. Muller and E. Altenburg, *Genetics* **15**, 283 (1930).
2. C. P. Oliver, *Science* **71**, 44 (1930); *Z. Indukt. Abstammun. Verebungsl.* **61**, 447 (1932); M. Demerec, *Cytologia* (Fujii Jubilee Volume) (1937), p. 1125.
3. I. H. Herskowitz, *Am. Nat.* **80**, 588 (1946).
4. M. Demerec and U. Fano, *Proc. Natl. Acad. Sci. U.S.A.* **27**, 24 (1941).
5. D. E. Lea and D. G. Catcheside, *J. Genet.* **47**, 10 (1945); J. B. S. Haldane and D. E. Lea, *ibid.* **48**, 1 (1947).
6. D. G. Catcheside, *Adv. Genet.* **2**, 271 (1948).
7. D. E. Lea, *Actions of Radiations on Living Cells* (Cambridge University Press, London, 1946).
8. U. Fano, *Science* **106**, 87 (1947).
9. C. W. Edington, *Genetics* **41**, 814 (1956).
10. I. H. Herskowitz, *ibid.* **36**, 356 (1951).
11. H. J. Muller and C. A. Offermann, unpublished data, cited by H. J. Muller, *J. Genet.* (1940), p. 401.
12. E. Novitski, *Drosophila Inf. Serv.* **26**, 115 (1952).
13. H. J. Muller, E. Carlson, A. Schalet, *Genetics* **46**, 213 (1961).
14. F. W. Stahl, *The Mechanics of Inheritance* (Prentice-Hall, Englewood Cliffs, N.J., ed. 2, 1969).
15. E. Novitski, J. Cohen, C. Laird, *Arch. Julius Klaus Stift. Vererbungsforsch. Sozialanthropol. Rassenhyg.* **37**, 431 (1962).
16. G. E. Brosseau, *Drosophila Inf. Serv.* **40**, 71 (1965).
17. This is an important point. I. I. Oster reports no such mosaics [see F. H. Sobels, Ed., *Repair from Genetic Radiation Damage and Differential Radiosensitivity in Germ Cells* (Macmillan, New York, 1963), pp. 179–204] after irradiating a similar chromosome, but his method of detection depended on finding fertile F_1 males, which would necessarily eliminate those cases with a broken or fragment Y chromosome lacking fertility factors.
18. E. Novitski, *Drosophila Inf. Serv.* **38**, 71 (1963).
19. B. Leigh and F. H. Sobels, *Mutat. Res.* **10**, 475 (1970); F. H. Sobels and B. Leigh, *ibid.* **12**, 100 (1971).
20. H. Bauer, *Naturwissenschaften* **27**, 821 (1939).
21. K. Bürki, *Arch. Genet.* **47**, 105 (1974).
22. K. Illmensee and A. P. Mahowald, *Proc. Natl. Acad. Sci. U.S.A.* **71**, 1016 (1974).
23. E. A. Carlson and E. Novitski, *Genetics* **50**, 239 (1964).
24. E. Inagaki and Y. Nakao, *Mutat. Res.* **3**, 268 (1966).

25. This article was written while I was the recipient of a John Simon Guggenheim Foundation Fellowship in residence at the University of Leiden. Help from Prof. F. Sobels, A. T. Natarajan, B. Leigh, J. H. Postlethwaite, and K. Sankaranarayanan is gratefully acknowledged.

Ergotism and the Salem Village Witch Trials

Records of the events of 1692 do not support the hypothesis that ergot poisoning was involved.

Nicholas P. Spanos and Jack Gottlieb

In a recent article in *Science* (*1*) it was suggested that the residents of Salem Village, Massachusetts, who in 1692 charged some of their neighbors with witchcraft did so because of delusions resulting from convulsive ergotism. The author of the article, L. R. Caporael, argued that (i) the general features of the Salem crisis corresponded to the features of an epidemic of convulsive ergotism, (ii) symptoms manifested by the girls who were the principal accusers were those of ergot poisoning, (iii) the symptoms shown by other accusing witnesses were also those of convulsive ergotism, and (iv) the abrupt ending of the Salem crisis suggests ergot poisoning. We shall attempt to show that these arguments are not well founded.

Features of Convulsive Ergotism Epidemics

Ergot is a fungus (*Claviceps purpurea*) that under some conditions infests rye and other cereal grains. When ingested the ergotized grain may produce a variety of cardiovascular effects leading, among other things, to gangrene (gangrenous ergotism), or neurological effects leading, among other things, to convulsions (convulsive ergotism) (*2–5*). Epidemics of convulsive ergotism have a number of general features that differ substantially from the events that occurred in Salem.

According to Barger (*2*), epidemics of convulsive ergotism have occurred almost exclusively in locales where the inhabitants suffered severe vitamin A deficiencies. Ergot poisoning in individuals with adequate vitamin A intakes leads to gangrenous rather than convulsive symptoms. Vitamin A is found both in fish and in dairy products. Salem Village was a farming community and Salem Town, which bordered the village, was a well-known seaport; cows and fish were plentiful. There is no evidence to suggest a vitamin A deficiency in the diet of the inhabitants, and it would be particularly unlikely for the so-called "afflicted girls," some of whom came from well-to-do farming families. The absence of any instance of gangrenous symptomatology makes it highly unlikely that ergot played any role in the Salem crisis.

Young children are particularly susceptible to convulsive ergotism. Barger states (*2*, p. 39):

> All accounts of convulsive ergotism agree that children were more liable to convulsive ergotism than adults; thus 56 percent in the Finnish epidemic were under 10 years of age; 60 percent of Scrinc's cases were under 15 years of age. . . .

Only 3 of the 11 afflicted girls at Salem were under 15 years of age and only one of those was under 10 (*6*, p. 57). There is no evidence either in the trial records or in eyewitness accounts to indicate a high rate of convulsive symptoms in the young children of Salem Village during the witch crisis (*7–11*). In fact we could find references to only two cases of convulsions in children under ten during the period of the crisis. One of these was the afflicted girl mentioned above. The other was an 8-week-old infant that convulsed before it died (*11*, vol. 1, p. 95). An 8-week-old infant would not yet have been weaned, and nursing infants do not suffer ergot poisoning even if their mothers have a very severe case of the disease (*2*, p. 38); it is therefore unlikely that this infant died from ergotism.

The fact that most of the individuals (including young children) living in the same households as the afflicted girls showed no symptoms is attributed by Caporael to wide individual differences in susceptibility to ergot poisoning. While there are wide individual differences in susceptibility to gangrenous ergotism, convulsive ergotism is another matter. According to Barger it was common for all members of a family to develop symptoms of convulsive ergotism during epidemics (*2*, p. 27). This tendency was so pronounced that convulsive ergotism was long (but erroneously) thought to be infectious.

Dr. Spanos is an assistant professor and Mr. Gottlieb a doctoral candidate in the Department of Psychology, Carleton University, Ottawa, Ontario, Canada K1S 5B6.