might do. In the case of the virus aggregate, however, the particles appear not to be interwoven, but simply to lie in layers crossed at definite angles, and stacked one over another.

Although the particles tend to form aggregates as wide as one particle is long, in many cases compounding is evident. This appears to arise from the extension of a layer of the parallel virus rods in one direction to form a compound aggregate of two, three, four, or more of the individual units (cover illustration). The individual elongate elements may appear singly or in small clusters in the cytoplasm, or they may group together in bundles of up to a hundred or more, as shown in the figure. It is believed that these are the individual units in the fibrous bundles visible under the light microscope of fresh mounts of leaf hairs and epidermal cells infected with this virus.

These unusual aggregates have not been observed in sections of leaves infected with type TMV (*5*). They differ strikingly, both under the light and electron microscopes, from the large, plate-like hexagonal crystals produced by type TMV. They also differ from the spike-like paracrystals in that they have the particles oriented at right angles to the length of the aggregate, rather than along it.

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Genetics of Mitochondria

The comment by Van Valen (*1*) on our demonstration of complementation between two kinds of mitochondria in maize (*2*) is interesting. The possibility that mitochondria from F_1 heterozygotes are superior in oxidative activity to a 1 : 1 mixture of mitochondria from the two parental lines was not overlooked. We indicated that the mixture "approaches the behavior of the hybrid." It did appear to us at the time that the problem may be resolved either (i) by statistical analyses of additional data or (ii) by a close scrutiny of mitochondria of F_1 hybrids and their parents. We chose to follow the more definitive second alternative since the phenomenon of complementation by two kinds of mitochondria suggested that such mitochondria are different from one another.

Maize is polymorphic with respect to its mitochondria (*3*). Specifically, Ohio 45 has one type of mitochondria, as revealed by density-gradient centrifugation; Wf9 has two types, one of which is identical to mitochondria of Oh45; Wf9/Oh45 has three types, two of which are identical to those of Wf9 and a third type not found in either parent. Thus the hybrid has parental types of mitochondria plus a hybrid-specific type.

Behavior of these mitochondria, as determined by rate of oxidation of cytochrome *c*, is as follows: the single type of mitochondria of Oh45 exhibits complementation with either of the two types of mitochondria from Wf9; parental types of mitochondria from the F_1 hybrid also exhibit complementation. However, the hybrid-specific type of mitochondria accounts for an additional 30 percent of the total activity of the mitochondria of the hybrid.

Thus our original statement that 1 : 1 mixtures of parental mitochondria approach the activity of hybrid mitochondria is verified. While parental mitochondria—whether extracted from parental lines or separated out from the hybrid—exhibit complementation, the hybrid is still superior because it has an additional type of mitochondria.

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Antipodal Location of Continents and Oceans

Harrison (*1*) considers the antipodal location of continents and oceans to be nonrandom, and presumably due to convection currents. His use of Monte Carlo methods to solve an otherwise intractable problem is admirable. However, his conclusion about the unlikeliness of the continents and oceans being randomly arranged is less impressive from a statistical point of view. He concludes that " . . . there is less than 1 chance in 14 that the present antipodal distribution of continents and oceans is the result of a random process." One's confidence in this statement is lowered to considerably less than 13 to 1 by the "eyeball" selection of antipodality as a striking phenomenon worthy of testing, rather than some other hypothesis of randomness.

On the other hand, perhaps his statement was not as strong as it might have been, if we view the problem in a different way. Harrison takes 82.6 percent of the total continental area to be antipodal to ocean. But Runcorn (*2*) says " . . . only 4 percent of the area of the continent is antipodal to continent," which leaves 96 percent antipodal to ocean. From Harrison's Fig. 1, the random chance of 96 percent of the land being antipodal to ocean is far less than 1 in 14, perhaps 1 in 1000.

Harrison's use of six circular or triangular continents also seems somewhat on the conservative side. Both Eurasia and South America have three shield areas, which suggests overlapping subcontinents, especially in the case of India. Neither the circles nor the triangles were allowed to overlap in Harrison's study; this probably reduced somewhat the correlation between continent locations, and slightly increased the weight of the tails of the distribution of the portion of continent antipodal to continent. One may gain insight into the effects of his use of six continents, rather than ten, and also of his use of smooth figures rather than ragged continents, by breaking the continents up into incremental islands. If the islands are scattered independently (other than not overlapping), then the expected portion of land antipodal to ocean is $1 - p$ if the land is portion p of the total surface of the earth. Harrison uses $p = 0.334$, so the expected portion of land antipodal

to ocean is 67 percent. This value is close to the median of 68 percent found by Harrison for both circles and triangles; the closeness of the match indicates that the median is not sensitive to the number of continents. Note that this island model has a degenerate distribution of antipodal land, with probability 1 of antipodal portion $1 - p$, which again suggests that Harrison's estimate of expected continent antipodal to ocean may be conservative if it is based on too few or too smooth continents, which would cause too large a standard deviation.

Altogether, it seems reasonable to conclude with Harrison that the observed portion of land antipodal to ocean is unlikely to have occurred by chance, even allowing for the a priori selection, especially if Runcorn's statement is correct.

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Thompson questions the use of the figure of 17.4 percent of continental area being antipodal to continent. In the report by Harrison (*1*) the figure was derived from Vening Meinesz (*2*), and it had also been used by Evison and Whittle (*3*) in their study. As to whether the figure of 17.4 percent is more correct than Runcorn's (*4*) figure of 4 percent, we have no information. The figure of 17.4 percent is supposed to be for continents out to the edge of the continental shelf, whereas the figure of 4 percent is presumably for the continental areas above sea level. We are at the moment engaged in refining the study started by Harrison. We are now using the actual shapes of the present-day continents, rather than circular or triangular continents, another factor mentioned by Thompson. This study will incidentally give a new measurement of the percentage of continental area antipodal to continent for the present-day distribution.

Finally, the problem of how many continents one should use, while being fascinating, is much more speculative, as one has to decide (i) what were the original continents, and (ii) how much one is going to allow them to overlap. At present we feel that the study should not include such speculative decisions, but we may consider them at a later date.

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Toxicity of Antibiotics in Laboratory Rodents

The deaths of a number of experimental rodents that had been treated with antibiotics impressed upon us our ignorance of the toxicity of certain of these compounds to small animals. In an attempt to arrest a bacterial infection that appeared in a group of hamsters (100 to 150 g) we administered a preparation of procaine penicillin G in dihydrostreptomycin solution. The dosage recommended in the package insert was ¼ cc for small animals (0 to 5 lb). Each hamster was therefore injected intramuscularly with ¼ ml of the preparation. Within several minutes, all the hamsters were dead. When we injected normal mice apparently free from infection to study further this fatal reaction, similar results were obtained. Mice weighing 18 to 22 g having each received either intraperitoneally or intramuscularly ⅛ ml of a different batch of the same preparation or of a preparation containing streptomycin without penicillin died within 5 to 6 minutes.

It seemed probable that the carrier or other material included in the formulation of this antibiotic was lethal. The chemical firm of J. D. Copanos and Company, Incorporated, Baltimore, Maryland, was contacted, and it kindly provided us with the following useful information on the toxicity of streptomycin and dihydrostreptomycin compounds.

Data released by the National Academy of Sciences and the National Research Council after a search of the literature show that streptomycin and dihydrostreptomycin are extremely toxic to mice, rats, and other rodents. A mouse weighing 20 g apparently has a 50 percent chance of survival if it receives a 4-mg dose of streptomycin or dihydrostreptomycin intravenously, regardless of the volume in which this amount is contained. Also, a mouse weighing 20 g has a 50 percent chance of survival if it receives 18 mg of streptomycin or dihydrostreptomycin parenterally (other than intravenously); a similar mouse has a 50 percent chance of survival if it receives 180 mg of streptomycin or dihydrostreptomycin orally. Therefore, a single dose of penicillin-dihydrostreptomycin to be administered parenterally (other than intravenously) to a 20 g mouse should not exceed 0.08 ml of the standard product; a dose to be administered orally should not exceed 0.8 ml of the standard product.

Apparently the toxicity of certain antibiotics in rodents is generally known throughout the field of chemotherapy. There are several accounts concerning the lethality of small doses of penicillin in the guinea pig, and recently Farrar, Kent, and Elliott (*1*) described similar lethal effects of bacitracin in the same rodent.

One of the antibiotic preparations that we were using is intended mainly for use by veterinarians; this preparation was not accompanied by a warning regarding dosages for small experimental animals. Consultation with several local veterinarians indicated a lack of familiarity with the toxicity of these compounds for rodents. In view of this, and as a result of our own experience, we would suggest that other investigators use caution in treating small laboratory rodents with antibiotics.

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Quasi-Stellar Objects: Possible Local Origin

Terrell (*1*) discusses the arguments favoring and contradicting the local-origin hypothesis of quasi-stellar objects and concludes that the galactic origin suggested by him (*2*) can account for all known characteristics of quasi-stellar objects.