necessary to keep the probabilities of making both kinds of errors at a certain level if one has an estimate of variability of the experimental material.

I have made this calculation using percentages of total aberrations (breaks plus gaps) (*2*). The percentages were transformed to the square roots of their arc sin values for obtaining an estimate of the variance. A normal distribution, necessary for the proper application of this method, is assumed arbitrarily. Six replications per group would be required to detect a mean difference of 5 percent per subject with a 5 percent risk of rejecting a true hypothesis and a 25 percent chance of accepting a false hypothesis. Only four replications were used in the experiment.

What this means is that the authors may be taking a greater than 1 in 4 chance of accepting the hypothesis that there is no difference between users and nonusers, even if a true difference of 5 percent total aberrations actually exists. I do not know whether the specified difference of 5 percent is appropriate; this is a medical question. It is, however, a rather large difference, with respect to the overall mean percentage, of 6.8 percent aberrations.

The importance of decisions based on the results of this experiment seems to warrant an attempt to reduce the risk of making a type II error by increasing replications. This is especially true in light of the fact that Sparkes *et al.* recognize that their results are at variance with other published work.

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19 August 1968

We agree that, given the mean differences between control and LSD exposed subjects in our study, there is danger of a type II error when significant differences cannot be demonstrated and that since type I and type II errors are inversely related they can be minimized only by simultaneously increasing sample sizes. Situations in which the power of a test is reduced and the importance of both types of error are equal permit lowering of the accepted confidence limit (*1*). However, in the case of our combined results and counting all aberrations (breaks plus gaps), the null hypothesis could not be rejected even at the .20 level for control versus "users" and at the .10 level for the control versus LSD-treated subjects.

Further, it might be questioned whether the two types of error should be weighed equally. It might be argued that the acceptance of a fallacious hypothesis may be more detrimental to scientific progress than rejection of a true one.

We are unaware of any theoretical reason to anticipate whether LSD should have a damaging effect on chromosomes. Therefore, the answer to this important question has to be based on observations and there is no reason a priori for weighing levels of significance on the basis of what a reasonable result should be. We thus used the standard 5 percent confidence limit.

Because studies of chromosome damage often demonstrate a skewed distribution, with a few individuals showing a large number of aberrations relative to the rest of the sample, the use of a "distribution-free" test of significance, as applied in the evaluation of our data, seemed appropriate. Whether assumptions of normal distribution can be made, thus allowing for more powerful inferences, depends on one's judgment regarding the "robustness" of such procedures.

As Kruskal and Haberman note, complete random selection of subjects is difficult, and, the populations from which our three groups were drawn are different. Our greatest concern was with the exposure or lack of exposure to drugs. Second, the cells were evaluated blindly for chromosome damage, a point inadvertently omitted from our initial report. Third, portions of the same blood sample were analyzed in the two laboratories, and not "separate samples" as suggested by Kruskal and Haberman. The results from each laboratory were evaluated separately, results from each group of subjects were compared between laboratories, and then the results were combined. Comparisons between laboratories as noted in our Table 3 (*2*) indicate that the null hypothesis is sustained for the following P values: controls versus controls (breaks plus gaps), $P = .057$; controls versus controls (breaks), $P = .171$; users versus users (breaks plus gaps), $P = .557$; users versus users (breaks), $P = .443$; treated versus treated (breaks plus gaps), $P = .243$; and treated versus treated (breaks), $P = .443$. Results between groups in each laboratory were in the same direction for both laboratories.

With regard to the "substantive" significance of our findings in seven of the eight comparisons (*2*, Table 3) of controls with subjects exposed to LSD, the controls show a higher percentage of aberrations; the one exception is that in which controls had fewer breaks than the "users." Therefore, despite the above-mentioned limitations of the statistical evaluation of our data, we are still inclined to conclude that our studies do not show either "statistical" or "substantive" evidence of chromosomal damage by LSD.

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9 October 1968

Factors Determining Spatial and Size-Frequency Distributions of Gemma gemma

Jackson (*1*) has used some data on the spatial and size-frequency distributions of *Gemma gemma* Totten in a bay near Guilford, Connecticut, to support his conclusion that "generalizations on the paleoecological significance of one sort of size-frequency distribution or another seem inappropriate without some idea of the life histories involved." Although we would not disagree with this conclusion, we feel, on the basis of our own work of the last 2 years at Barnstable Harbor, Massachusetts (*2*), that Jackson's data on *Gemma* and some of the conclusions drawn from them are misleading.

Jackson washed his *Gemma* samples through a 1-mm sieve. According to Sullivan (*3*), Sellmer (*4*), and our own work, newly released *Gemma* range in

length from 0.29 to 0.45 mm. We have found that a 0.25-mm sieve must be used to retain all *Gemma*, and that *Gemma* of less than 1.2 to 1.4 mm length will pass through a 1-mm sieve. It is not easy to estimate how many *Gemma* Jackson may have lost, because *Gemma* growth rates vary with latitude. Sellmer (*4*) found that in New Jersey *Gemma* reached 1.5 to 2 mm length by the fall of the year they were released. On the north side of Cape Cod this length is not attained until the second fall. If the Long Island Sound regime is more like that of New Jersey than that of Cape Cod, it is likely that one-third to one-half of the 1st year class are missing from Jackson's November data and from his Fig. 1.

It is unlikely that the aggregation of *Gemma* which is evident with small samples of 40 cm^2 (our data) or 25 cm^2 (Jackson's data) is caused by the method of reproduction and release. Any aggregation of newly released *Gemma*, if unaffected by heterogeneity of substrate, would be broken up after several tidal cycles, much less after the minimum of several months needed to reach a length of 1 mm. Heterogeneity of the environment is probably the determining factor. Sanders *et al.* (*5*) state that the average distance from crest to crest for ripple marks in the area we have studied at Barnstable is 4 to 5 cm, precisely the value which would have the most effect on 25- to 40-cm^2 samples. Such ripple marks often occur on the sediment types where *Gemma* is common.

Jackson found the ratio of variance to mean to be greater for younger than for older *Gemma*. This ratio is a biased measure of aggregation (*6*). That is, the estimated ratio tends to be lower when samples are taken from populations of lower density, even when there is no less aggregation. The density of 2nd year class *Gemma* would typically be orders of magnitude lower than that of 1st year class *Gemma*.

Finally, our study shows marked effects of very slight tidal-height differences on growth and mortality rates, and therefore on size-frequency distributions. It is likely that the net effect of these remarks is to strengthen Jackson's conclusion.

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7 August 1968

Green and Hobson state that "it is likely that one-third to one-half of the 1st year class are missing from" my data (*1*) on the living *Gemma* population. Although doubtless some individuals were lost, I feel this estimate is much too large. Green and Hobson report that *Gemma* less than 1.2 to 1.4 mm in length will pass through a 1-mm sieve, and some must do so; but examination of the length-frequency distribution (*1*) of the dead *Gemma* shows that numerous individuals as small as 1.1 mm, mostly single valves, were retained by the 1-mm sieve. That this was not the case for live *Gemma* suggests that very few live individuals smaller than 1.2 mm in length were present at sampling.

In addition, the seasonal temperature-salinity regime in Long Island Sound is essentially similar to that in New Jersey where Sellmer (*2*) obtained the following values for shell length of 1st year *Gemma* in November:

Year	Mean length	Range (mm)
1955	2.440	1.677–3.978
1956	1.850	1.248–2.574
1957	1.347	1.053–1.950

Furthermore, for his 1956 October length-frequency polygon (only year for which such data is given) less than 10 percent of the individuals have lengths less than 1.3 to 1.4 mm. Because waters north of Cape Cod are well known to be ecologically quite dissimilar to those to the immediate south, it is probable by these data that maximum sieve loss of live *Gemma* in my samples was 10 to 20 percent of the total live 1st year population. This was the justification for use of a 1-mm sieve in the first place.

As a check of my 1967 procedure, on 15 October 1968, I collected mud (0.1 m^2 by 3.5 cm) from the same locality sampled previously and sieved this sediment with both a 1-mm and 0.42-mm sieve. Of 227 live *Gemma* obtained in this sample, 27 passed through the 1-mm sieve. If we assume, as for last year that 75 percent of those *Gemma* trapped by the 1-mm sieve are in the 1968 1st year class, then 27 of 177 (75 percent of 200 = 150 + 27 = 177) or approximately 15 percent of the total live 1st year class *Gemma* were lost through the 1-mm sieve. This is in agreement with the value range expected from examination of Sellmer's (*2*) data.

Green and Hobson question the reliability of the ratio of variance to mean as a measure of distribution for the live 2nd year *Gemma* population. However, the density of this population (141 individuals in 100 cells) is within the acceptable range of this statistic which only tends to break down when the mean is less than 1, that is, when cells consist mainly of 1's and 0's (*3*) which was not the case for my data. The fact that the live 2nd year population density is 25 percent (20 percent including probable sieve loss) that of the live 1st year population (not "orders of magnitude lower") is not relevant to the validity of the determined distribution of the live 2nd year population, which is further supported by the similar result based on the binomial (*1*).

I agree that it is intuitively difficult to imagine that aggregation of a small infaunal intertidal species would not be destroyed by tidal action. But the live 1st year *Gemma* are aggregated, and the same argument could be applied to any mechanism of aggregation caused by some subtle heterogeneity of the environment. Moreover, although the 2nd year dead *Gemma* population is aggregated, probably by tides, the live 2nd year population is randomly distributed (both variance to mean and the binominal), implying that the 0.25 m^2 sampled was homogeneous for *Gemma* and that live *Gemma* do actively control their position in this environment.

Finally, Buzas (*4*) has proposed asexual reproduction as the probable cause for aggregation of certain species of foraminifera collected for distributional analysis from mud in 1-m water depth in Rehoboth Bay, Delaware. If this is true, and if the primary distribution of foraminifera is not destroyed by currents, it seems reasonable that live 1st year *Gemma* may not be either.

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