3) The Forestry Service has for some years been studying and effecting the germination of tambalacoque seeds without avian intervention (*2*). The germination rate is low but not more so than that of many other indigenous species which have, of recent decades, showed a marked deterioration in reproduction. This deterioration is due to various factors too complex to be discussed in this comment. The main factors have been the depredations caused by monkeys and the invasion by exotic plants.

4) A survey of the climax rain forest of the uplands made in 1941 by Vaughan and Wiehe (*3*) showed that there was quite a significant population of young tambalacoque plants certainly less than 75 to 100 years old. The dodo became extinct around 1675!

5) The manner in which the tambalacoque seed germinates was described by Hill (*4*), who demonstrated how the embryo is able to emerge from the hard woody endocarp. This is effected by the swollen embryo breaking off the bottom half of the seed along a well-defined fracture zone.

It is necessary to dispel the tambalacoque-dodo "myth" and recognize the efforts of the Forestry Service of Mauritius to propagate this magnificent tree of the upland plateau.

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References and Notes

1. S. Temple, *Science* **197**, 885 (1977).
2. Young *Calvaria major* plants that are 9 months old or more can be seen at the Forest Nursery in Curepipe.
3. R. E. Vaughan and P. O. Wiehe, *J. Ecol.* **19**, 127 (1941).
4. A. W. Hill, *Ann. Bot.* **5**, 587 (1941).

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The plant-animal mutualism that may have existed between the dodo and *Calvaria major* became impossible to prove experimentally after the dodo's extinction. What I pointed out (*1*) was the possibility that such a relation may have occurred, thus providing an explanation for the extraordinarily poor germination rate in *Calvaria*. I acknowledge the potential for error in historical reconstructions.

I disagree, however, with the conclusion of Owadally (*2*) that the dodo and *Calvaria* were geographically separated. There have been virtually no bones of dodos or any other animals found in the uplands of Mauritius not because the animals were never there, but because the island's topography does not cause alluvial deposits there. Catchment basins in certain lowland areas accumulated many bones of animals that were washed into these areas from the surrounding uplands. Accounts of early explorers, summarized by Hachisuka (*3*, p. 85), definitely refer to dodos occurring in the uplands, and Hachisuka makes a point of clarifying the misconception that dodos were strictly coastal birds. Early forestry records from Mauritius (*4*) indicate that *Calvaria* was found in the lowlands as well as on the upland plateau. Although native forests only occur in the uplands today, one of the surviving *Calvaria* trees is located at an elevation of only 150 m. Thus, the dodo and *Calvaria* may have been sympatric, making a mutualistic relation possible.

Taxonomic authorities on sapotaceous plants of the Indian Ocean region recognize seeds of *Calvaria major*, as well as the smaller seeds of *Sideroxylon longifolium*, from alluvial deposits of the Mare aux Songes marsh (*5*), but this has little relevance to the question of mutualism. Mutualistic species will not necessarily be fossilized together.

The Mauritius Forestry Service has only recently succeeded in propagating *Calvaria* seeds, and the unmentioned reason for their recent success strengthens the case for mutualism. Success was achieved when the seeds were mechanically abraded before planting (*6*). A dodo's digestive tract merely abraded the endocarp naturally the same way the staff of the Mauritius Forestry Service does artificially before the seeds are planted.

The reference Owadally cites (*7*) is equivocal about the age of the surviving *Calvaria* trees because there is no easy way to accurately date them. Coincidently, Wiehe, the coauthor of the paper Owadally cites, was also my source of the estimated age of over 300 years for the surviving trees. I agree that there were more trees surviving in the 1930's than today, which further supports the notion that *Calvaria major* is a declining species and may have been so since 1681.

I erred in not citing Hill (*8*). However, Hill does not describe how and under what conditions he induced a seed to germinate. Without these details, his description is of little relevance to the question of mutualism.

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References and Notes

1. S. A. Temple, *Science* **197**, 885 (1977).
2. A. W. Owadally, *ibid.* **203**, 1363 (1979).
3. M. Hachisuka, *The Dodo and Kindred Birds* (Witherby, London, 1953).
4. N. R. Brouard, *A History of the Woods and Forests of Mauritius* (Government Printer, Mauritius, 1963).
5. F. Friedmann, personal communication.
6. A. M. Gardner, personal communication.
7. R. E. Vaughan and P. O. Wiehe, *J. Ecol.* **19**, 127 (1941).
8. A. W. Hill, *Ann. Bot.* **5**, 587 (1941).

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Developmental Dyslexia: Research Methods and Inferences

Witelson (*1*) has proposed that developmental dyslexia is associated with bilateral hemispheric representation of spatial function that interferes with specialized left hemispheric processing of linguistic information. The data presented, however, may not warrant this conclusion because of (i) inferences drawn from a failure to obtain a significant treatment effect in groups selected from a heterogeneous population and (ii) the possibility of sampling biases.

There are at least two situations that can produce a result of no significant response to an independent variable. One is that nearly all subjects within a group show little or no response to the variable, thus a measure of the variability of this group would be small. A second possibility is that some subjects respond in one direction to the variable, others in the opposite direction. This latter possibility is a special case of the broad class of situations in which the data are characterized by more than one mode—situations that would produce no mean group response to the variable, but a large within-group variability.

In Witelson's case, the idea of bihemispheric representation of spatial function in dyslexic boys stems from results of two tests in which the mean score of dyslexics was the same to left- and right-sided stimulation, whereas the mean scores of normal readers showed the predicted left-sided superiority. Dyslexics are thought to be a heterogeneous group of children with respect to cognitive and perceptual function, as Witelson and others (*1*, *2*) have noted. Thus, we might expect that the reason for the obtained null result in dyslexics stems from the second possibility described. Presentation of the distribution of signed difference scores for each group is the minimum information needed to properly evaluate Witelson's claim. As her theory pertains to individual dyslexics, we need to know whether the group mean adequately reflects the performance of the individuals in this group.

Sampling from a heterogeneous population is likely to yield a heterogeneous sample. Witelson's theory represents an effort to integrate the results from four different tests on dyslexics and normal readers, all of which involved response to lateralized stimulation. Her overall sample of dyslexics contains 85 dyslexic boys and 156 normal reader control subjects, although not all subjects in either group received all tests. From among the 85 dyslexics, on each of the four tests, $N = 62$, 82, 85, and 55. From among the 156 controls, on the same tests, $N = 100$, 85, 156, and 28. As she tested different subsets of children from her original sample on each test, she has left open the possibility that a sampling bias differentially affected the composition of each of the groups which served on each of the tests. For instance, there may be a different mix of "types" of dyslexics in the different groups of children taking the different tests.

The variable of age provides a clear example of the problem of comparing results across tests when all subjects have not participated in all tests. Although the author has matched her total dyslexic and control groups for age, it is not clear that the different subsets of subjects run on each of the tests were age-matched either between groups, or across tests. Since the children studied ranged in age between 6 and 14 years, and since performance on cognitive-perceptual tests such as those used are known to vary with age (*3*), a different mix of ages between groups on each test might distort the results.

As a remedy to the problem, Witelson might analyze data from only those age-matched subjects who participated in all tests. For a post hoc test of the hypothesis that bilateral representation of spatial function interferes with ("overloads") the left hemisphere's capacity for sequential linguistic processing, we need to know if lack of perceptual asymmetry on the two spatial tasks was correlated with poor performance on the dichotic listening task. The author offers the latter task as a linguistic task requiring left-hemisphere participation.

If one accepts the notion that there are different subgroups of developmental dyslexia, the strategy for defining these groups must be different from that which one would use when investigating a relatively homogeneous population. Single performance measures taken on a group of dyslexic subjects for comparison with a group of normal readers will produce significant results only if the measure is one on which many dyslexics perform both (i) similarly to each other and (ii) differently from normal readers. The diversity of the population of dyslexic readers makes such an outcome unlikely, unless the measure directly involves a reading skill. What may happen is that only a small percentage of dyslexic subjects will react differently from normal readers on the performance measure chosen, and thus the overall results may be viewed as negative.

If, however, multiple measures are used for each dyslexic subject tested, possible covariation among these measures may suggest an explanation for the overall negative results on one of the measures, and may, as well, provide an empirical basis for distinguishing among the various subgroups of dyslexia. Such an analysis of Witelson's data may provide a fruitful beginning for more clearly defining the different causes of dyslexia.

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References and Notes

1. S. F. Witelson, *Science* **195**, 309 (1977).
2. A. N. Applebee *J. Child Psychol. Psychiatry* **12**, 91 (1971); A. L. Benton, *Adv. Neurol.* **7**, 1 (1975); H. G. Birch, in *Reading Disability: Progress and Research Needs in Dyslexia*, J. Money, Ed. (Johns Hopkins Press, Baltimore, 1962), pp. 161–169; E. Broder, in *Progress in Learning Disabilities*, H. R. Myklebust, Ed. (Grune & Stratton, New York, 1971), vol. 2, pp. 293–321; S. Mattis, J. H. French, I. Rapin, *Dev. Med. Child Neurol.* **17**, 150 (1975); D. P. Shankweiler, *Neuropsychologia* **1**, 267 (1963).
3. See S. F. Witelson [in *The Neuropsychology of Learning Disorders: Theoretical Approaches*, R. Knights and D. J. Bakker, Eds. (University Park Press, Baltimore, 1976), pp. 232–256] for data on developmental changes in performance on these tests and S. Satz [in *ibid.*, pp. 273–294], who has emphasized the need for isolating the effects of age on measures of perceptual asymmetry.

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The concerns of Gross and Rothenberg essentially stem from the absence of details not included in my report (*1*), but which were given in two longer papers (*2, 3*) cited [references 12 and 28 in (*1*)]. As Morrison (*4*) indicated in his recent response to Rothenberg and Gross' (*5*) other technical comment on Morrison, Giordani, and Nagy's report (*6*), also on dyslexia, "Restrictions on length and format of reports in *Science* limit presentation of certain methodological points. Further details on these methodological features are presented elsewhere." Nevertheless, I will give here some of the details requested.

Gross and Rothenberg's statement that my hypothesis of bihemispheric representation of spatial processing in dyslexic boys stemmed from the results of two tests (they are referring to the tachistoscopic test using human figures and the dichhaptic nonsense shapes test) is inaccurate. A third set of data, that based on the dichhaptic letters test interpreted in the context of the other sets of data, was also used to infer greater bihemispheric representation of spatial functions in dyslexic compared with normal boys.

In both dichhaptic tests, analyses of variance (group by age by hand) indicated significant group-by-hand interactions (nonsense shapes, $P < .01$; letters, $P < .005$) (*2, 3*). Inferences were based on a lack of difference between hands in dyslexics on the shapes test, but based on a significant difference in favor of one hand in dyslexics, opposite to that observed for the normal group, on the letters test. In each test the variances were not significantly different for any of the subgroups, nor was there a bimodal distribution in the case of dyslexic or normal subjects. As reported, χ^2 tests indicated significant differences between dyslexic and normal groups in the distribution of subjects having greater left- or right-hand scores (*2, 3*). The data from the tachistoscopic task are the least clear, mainly because this test had too inconsistent a pattern in normal subjects (*2, 3*). In fact, it was this frequently encountered difficulty with tachistoscopic tasks which in part led me to devise a task that engaged the tactual modality.

Although Gross and Rothenberg did not discuss the dyslexics' performance on the dichotic digits test, it should be noted that although in this case they showed the pattern of right-ear superiority as do normal children, the dyslexics again showed a consistent within-group pattern. There was no bimodal distribution of scores for the dyslexics, and the χ^2 test indicated no difference in the distribution of subjects with greater right- or left-ear scores (*2, 3*).

Moreover, although Gross and Rothenberg have suggested that the hemisphere lateralization test results may be due to dyslexics' being a "heterogeneous group of children with respect to cognitive and perceptual function," and that such "diversity" will likely result in diverse test performance, they ignored the data dealing more directly with the level of such skills. These data indicated a consistent, depressed level of accuracy on the dichotic test (a linguistic task), and a consistent, normal level of accuracy on the three dichhaptic and tachistoscopic tests (all involving spatial processing) (*1–3*).

Thus, the mean left and right scores of the dyslexic group on each test represented the majority of dyslexics. That not every dyslexic, nor indeed every normal individual, performed in the same manner is not surprising. These perceptual tasks are imperfect indicators of hemisphere functional specialization (*7*), and are not sufficiently sensitive for use on an individual basis (for example, as a diagnostic tool). It was for this reason that I used a battery of lateral perceptual tests to allow for the possibility of converging patterns of performance from which to draw neural inferences [explained more fully in (*2*)] of (i) greater bihemispheric representation of aspects of spatial processing on dyslexics and (ii) typical left-sided representation of language but dysfunction in the left hemisphere. The possibility that either neural factor might result in the other or that both could be caused by one or more independent antecedent factors was left open (*1–3*).

Gross and Rothenberg have taken the position that there are different and definable subgroups of developmental dyslexia. Although several clinical descriptive reports and experimental studies have noted the heterogeneity of symptomotology among dyslexics and have suggested the possibility of subgroups delineated on the basis of variation in behavioral and cognitive symptomatology, reading patterns, hereditary, and birth stress factors (*8*), the results to date are not congruent as to number, description, and specific definition of the subgroups. I agree with Gross and Rothenberg that this is likely an important and fruitful issue. For the present, however, in the study of dependent variables such as cognitive skills and patterns of cerebral dominance in dyslexia, it is difficult enough to diagnose and objectively define dyslexia or specific reading disability as opposed to other childhood disorders, such as primary emotional disturbance and hyperactivity (*3, 9*). Much current research (*10*) does not use or support different subgroups.

Furthermore, whether such putative subgroups vary in aspects of hemisphere specialization remains to be determined. Different reading or cognitive patterns do not necessarily imply different neural organizations. It is an even further issue whether there are "different causes" of dyslexia as Gross and Rothenberg state.

Gross and Rothenberg's second main concern relates to possible sampling biases. They noted the different sample sizes of dyslexics and queried why different subsets of the total group were tested and whether this could account for the obtained results. The reason for different N's has been specified (*2*). All children were given all tests as soon as the tests were available. This research, like much clinical research, was done over several years. All children ($N = 85$) were tested with the dichotic test (even the few with unilateral hearing loss were tested by adjusting channel volumes); all were given the tachistoscopic test [except the few having inadequate stereopsis and therefore poor binocular fusion, which could result in improper fixation with at least one eye ($N = 82$)]; and when the dichhaptic tasks were subsequently devised, all the then-current cases were accordingly tested, except for a few who were unable to name letters sufficiently well to make the letters test a valid task ($N = 62$ for shapes; $N = 55$ for letters). There is no reason to assume a different selection of dyslexics over the years.

Gross and Rothenberg also queried whether age varied between the various dyslexic and control groups. The age range and mean age (*2, 3*) were comparable for all dyslexic and control groups. Moreover, in the detailed reports, groups were compared not only as wholes but also at four ages: 6 and 7, 8 and 9, 10 and 11, and 12 through 14 years (*2, 3*). On no test was a significant interaction of performance with age observed.

One final theoretical point: Gross and Rothenberg stated that age also is important here because "performance on cognitive-perceptual tests such as those used are known to vary with age." By their footnote (their reference 3), it becomes clear that what they consider to vary is perceptual asymmetry, not overall level of performance. Certainly overall performance may change with age, and in my research, as an example, age is significant for each test; that is, children do better as they get older. However, scant evidence supports the assumption that perceptual asymmetry changes with age. The results of the studies summarized indicate that perceptual asymmetry is evident at as early an age as experimenters have tested for it and that the magnitude of the asymmetry may not increase with age (*11*). A few more recent studies (*12*) have specifically shown not only perceptual asymmetry in the youngest children tested, but also no change in the extent of asymmetry with advancing age. New behavioral evidence may support right-hemisphere specialization for face perception in 3-month-old infants (*13*). Thus, my data for dyslexics may support atypical functional asymmetry in dyslexics at least as old as 14 years, yet such functional asymmetry is probably present at birth in the normal child. This suggests that age is not a crucial factor in explaining the differences between dyslexics and normal children and that the hypothesized atypical neural organization and neural dysfunction in dyslexics, if it does exist, is more likely to be a different neural substrate then a neural maturational lag.

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References

1. S. F. Witelson, *Science* **195**, 309 (1977).
2. ______, in *The Neuropsychology of Learning Disorders: Theoretical Approaches*, R. Knights and D. J. Bakker, Eds. (University Park Press, Baltimore, 1976), pp. 232–256.
3. ______, in *Psychopathology and Brain Dysfunction*, C. Shagass, S. Gershon, A. J. Freidhoff, Eds. (Raven, New York, 1977), pp. 15–49.
4. F. J. Morrison, *Science* **200**, 802 (1978).
5. S. Rothenberg and K. Gross, *ibid.*, p. 801.
6. F. J. Morrison, B. Giordani, J. Nagy, *ibid.* **196**, 77 (1977).
7. For example, S. Blumstein, H. Goodglass, V. Tartter, *Brain Lang.* **2**, 226 (1975).
8. For example, S. T. Orton, *Reading, Writing and Speech Problems in Children* (Norton, New York, 1937); O. L. Zangwill, in *Reading Disability*, J. Money, Ed. (Johns Hopkins Press, Baltimore, 1962), pp. 103–113; H. R. Myklebust, *Development and Disorders of Written Language, Picture Story Language Test* (Grune & Stratton, New York, 1965), vol. 1; M. Critchley, *The Dyslexic Child* (Heinemann, London, 1970); E. Boder, *Dev. Med. Child Neurol.* **15**, 663 (1973); S. Mattis, J. H. French, I. Rapin, *ibid.* **17**, 150 (1975); A. L. Benton, *Adv. Neurol.* **7**, 1 (1975); D. G. Doehring and I. M. Hoshko, *Cortex* **13**, 281 (1977).
9. P. Satz and J. M. Fletcher, in *Handbook of Minimal Brain Dysfunctions*, H. Rie and E. Rie, Eds. (Wiley, New York, in press).
10. For example, J. M. Finnucci, J. T. Guthrie, A. L. Childs, H. Abbey, B. Childs, *Ann. Hum. Genet.* **40**, 1 (1976); C. K. Leong, *Reading Res. Q.* **2**, 204 (1976–1977); R. G. Rudel, M. B. Denckla, M. Broman, *Brain Lang.* **6**, 52 (1978); L. S. Mark, D. Shankweiler, I. Y. Liberman, C. A. Fowler, *Mem. Cognit.* **5**, 623 (1977); F. R. Vellutino, *Harv. Educ. Rev.* **47**, 334 (1977).
11. For a review, see S. F. Witelson, in *Language Development and Neurological Theory*, S. Segalowitz and F. Gruber, Eds. (Academic Press, New York, 1977), pp. 213–287.
12. For example, G. W. Hynd and J. E. Obrzut, *Neuropsychologia* **15**, 689 (1977); C. Schulman-Galambos, *ibid.*, p. 577.
13. M. E. Barrera, A. Dalrymple, S. F. Witelson, paper presented at the annual meeting of the Canadian Psychological Association, Ottawa, 7 to 9 June 1978.

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