ity, to distinguish between *isolated* and *sporadic* cases. Isolated cases may be of the same origin as familial cases, and the absence of affected sibs due to chance, or they may be sporadic, of different origin (mutation, phenocopy, and so forth). To distinguish between incomplete penetrance and sporadic cases, we may recognize a quantity x, the frequency of sporadic cases among affected cases in the population, and test the hypothesis that no isolated case is sporadic ($x = 0$). For example, if a trait is sometimes due to a rare dominant gene, the probability that an affected parent with s children have at least one affected child is

$$P_{(r>0)} = (1-x)(1-q^s)$$

where $q = 1 - p$, and the scores for x give a specific, and the most efficient, test for the existence of sporadic cases not transmissible to the progeny.

If families are ascertained through parents or other relatives without regard to the children, and if $x = 0$, the probability of a segregating family ($r > 0$) is

$$P_{(r>0)} = (1-h)(1-q^s)$$

for possible backcrosses, and

$$P_{(r>0)} = (1-h)^2(1-q^s)$$

for possible intercrosses, where h, the probability that a parent of dominant phenotype be homozygous, is determined under random mating by the gene frequencies. Using this theoretical value of h, we may compare the scores for p from segregating and nonsegregating families with the scores from the distribution of r in segregating families as a test of the hypotheses that $x = 0$ and that mating is at random with the specified gene frequencies.

If $x\mu \ll (1-x)p$, where μ is the proportion affected among sibs of sporadic cases, then familial cases of sporadic origin may be neglected, and the probabilities for isolated and familial cases, respectively, are

$$P_{(r=1|r>0)} = \frac{sp\pi[(x+(1-x)q^{s-1}]}{xsp\pi+(1-x)[1-(1-p\pi)^s]}$$

and

$$P_{(r|r>1)} = \frac{\binom{s}{r}p^r q^{s-r}[1-(1-\pi)^r]}{1-(1-p\pi)^s-\pi spq^{s-1}}$$

The distribution of r for familial cases gives a third sum of scores for π and a test of the null hypothesis about p, independent of x, while the distribution of isolated and familial cases tests the hypothesis that $x = 0$, or permits an estimate if this hypothesis is rejected.

Sex-linked genes present interesting problems, such as that of distinguishing the mutation rates in the two sexes. However, for a decisive analysis, more penetrating methods are required than have so far been applied to this question. For a deleterious sex-linked recessive trait, the probability that an affected male be sporadic is

$$x = mu/(2u+v)$$

where m is the coefficient of selection against affected males, u and v are the mutation rates in egg and sperm, respectively, and it is assumed that carrier females have normal fertility and that all cases are sex-linked. The test of the hypothesis that $u = v$ reduces to testing whether $x = m/3$.

Corroboration of this test may be sought in the distribution of affected maternal uncles, assuming ascertainment through nephews. If two or more nephews are affected, the probability that at least one of s maternal uncles be affected is

$$P_{(r>0)} = (1-x')(1-q^s)$$

where the expected value of x' is ½ on the hypothesis of sex linkage. Similarly, if only one nephew is affected in a sibship of n nephews, the probability that at least one maternal uncle be affected, if ascertainment is through the nephew, is

$$P_{(r>0)} = \frac{(1-x)(1-x')q^{n-1}(1-q^s)}{x+(1-x)q^{n-1}}$$

These distributions provide a test of homogeneity of x and of x', of the deviation of x' from ½, and of x from $m/3$, the last deviation being accepted as significant only if the others are nonsignificant.

These ancillary tests are particularly important if sex-linked and autosomal cases may be confused, in which event families with autosomal or sporadic cases may be recognized if they contain at least one affected girl. The probabilities for isolated and familial cases under this condition are given in Fig. 1.

Genetic tests in man have never been carried out with the precision of these methods, the practical limitations of which cannot therefore be specified. Considerable caution should be exercised in analysis of medical literature or other biased sources, or if correct diagnosis is more likely in familial cases. However, it is still possible that the distribution of r for familial cases may be adequately described by the formulae of this report.

$$P_{(r=1|r>0)} = \frac{sp\pi[x+(1-x)q^{s-1}]}{xsp\pi+2(1-x)[1-(1-p\pi)^s-(\frac{1}{2}p+q)^s+(\frac{1}{2}p+q-\frac{1}{2}p\pi)^s]}$$

$$P_{(r|r>1)} = \frac{\binom{s}{r}p^r q^{s-r}[1-(1-\pi)^r][1-(\frac{1}{2})^r]}{1-(1-p\pi)^s-(\frac{1}{2}p+q)^s+(\frac{1}{2}p+q-\frac{1}{2}p\pi)^s-(\pi spq^{s-1})/2}$$

Fig. 1. Probabilities for different numbers of affected children under multiple selection with at least one affected girl.

With careful enumeration of probands and ascertainments when π is less than 1, these methods should be sufficiently general for almost any Mendelian analysis, provided that the mating types to be analyzed do not contain a mixture of segregation frequencies. (Common genes with low penetrance will continue to defy precise analysis.) For the typical case of a rare or highly penetrant gene, the method of maximum likelihood scores for p, x, h, and π, applied to the distributions of this report, will provide a simple and more powerful analysis than has been previously available.

NEWTON E. MORTON
Department of Medical Genetics, University of Wisconsin, Madison

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Structure of Adenine Polynucleotide

The x-ray diffraction pattern of untreated individual fibers of enzymatically synthesized adenine polynucleotide has been presented and discussed by Watson (*1*). A more crystalline preparation of the polymer has been obtained from material synthesized by Roland F. Beers, Jr., (*2*), spun into a bundle of fine fibers, and stretched while bathed in a heated mixture of ethanol and dichloroacetic acid (DCA) (*3*). The distribution of intensity on the x-ray diagram of these fibers is similar to that given by untreated fibers, showing that the form of the molecule is not greatly altered; differences of detail are observed, however, chiefly on the equator and on the first layer line. The 13 reflections here observed indicate a tetragonal unit cell, 22.6 by 22.6 by 15.0 A. A fourfold screw axis is evident parallel to the fiber axis, with translation of 3.75 A. The observed density of these fiber bundles (~1.60) suggests 16 nucleotide residues and 16 molecules of DCA per unit cell (calculated density, 1.66).

Ochoa and Heppel (*4*) have demonstrated that adenine polynucleotide is a 3′5′ adenosine diester of phosphoric acid. Furberg (*5*) has given bond distances and angles appropriate for a β-D-riboside; Broomhead (*6*) and Cochran (*7*) for adenine; Dunitz and Rollett (*8*) for a phosphoric acid diester. Barton and Cookson (*9*) and Mizushima (*10*) have summarized the principles of "conformational analysis." Using the above information and assuming that the polymer has no branches, we have deduced the probable preferred orientations of the groups around each of the six single bonds in the main chain of the polymer. The method developed by Bear (*11*) for collagen was then employed to exhibit, in a systematic way, all possible configurations of such a chain which would fit the several helix-net interpretations of the fourfold screw. The configurations fell into six main groups, in each of which one or more ways of satisfying net requirements were found. However, only three models capable of containing the required number of chains with atoms confined to radial limits of about 8 A could be built. One of these was discarded because the adenine–ribose bond was parallel to the fiber axis, which is expected to result in positive double refraction in contradiction to the negative sign observed by Rich (*12*). The remaining two, termed structures I and II, both have the planar adenine moieties perpendicular to the fiber axis, and both are adaptable to the observed unit cell as chain pairs whose pair axes run through the corners and the center of the square basal cell section.

The space group of structure I is $P4_322$. The fourfold screw is left-handed, and the two chains of each pair are related to each other by the perpendicular dyad axis. They are held together by a pair of reciprocal OH—O bonds of length 2.84 A between the O′-2 of one ribose group and the O′-1 of the other in each residue pair. The bonding is similar in type and length to that between O-1 and O-5 in glucose as determined by McDonald and Beevers (*13*). The P=O's of one chain are in contact with those of the other in the next turn of the screw. The adenine groups of any one double helix are in van der Waals contact in sets of two, and between any two adjacent double helices the bases form a solid column because of interpenetration.

The two chains of structure II are related by a dyad parallel to the fiber axis and are held together by a reciprocal pair of NH—N bonds between N-10 and N-1 of the adenine groups (pairing No. 2 of Donohue, *14*). The systematic analysis of stable chain conformations led to this pairing rather than to pairing No. 1 of Donohue, which has been employed by Watson (*1*) in an otherwise quite similar model. No other intramolecular H-bonds are here possible. The screw is now right-handed, and the space group is $P4_2$. Preliminary coordinates for both models are given in Table 1.

Fig. 1 shows, with vertical bars, the position in reciprocal space (and an indication of the relative intensity) of the reflections given by DCA-treated fiber bundles of adenine polynucleotide. Under each reflection is its index in the tetragonal lattice. On the right half of the figure in curving lines is the position of intensity shown on optical transforms (see Wyckoff *et al.*, *15*) of a single double helix of type I in projection perpendicular to the dyad axis (solid line) and in projection 45° to the dyad (dotted line). On the left is shown the location of intensity due to similar transforms of structure II in projection perpendicular to the x-axis (solid line) and 22½° to this axis (dotted lines). The intensities shown on each equator were derived from projections perpendicular to the fiber axis. Except for the equator for structure I, the optical transforms included neither cation nor DCA. The equatorial intensities for structure I include 16 DCA molecules placed in the cell to bind adjacent double helices. The transform of structure II lacks intensity in the (401)-(421) region of reciprocal space. With structure I, the regular oc-

Table 1. Right-handed Cartesian coordinates for structures I and II in angstroms.

Atom	Structure I			Structure II		
	x	*y*	*z*	*x*	*y*	*z*
C′-1	4.33	2.34	0.00	6.45	2.15	0.00
C′-2	4.37	1.67	1.32	7.50	2.00	−1.03
C′-3	3.03	2.05	1.83	6.80	2.90	−2.11
C′-4	2.08	2.00	0.65	6.25	4.05	−1.33
C′-5	0.95	2.90	1.10	4.90	4.80	−1.74
O′-1	3.03	1.87	−0.50	5.85	3.40	0.00
O′-2	4.62	0.30	1.40	8.85	2.10	−0.69
O′-3	2.36	1.36	2.88	7.75	3.20	−3.10
O′-5	2.20	0.35	5.00	4.50	5.85	−0.80
P	3.17	1.30	4.20	3.25	6.75	−0.60
OH	4.67	1.45	4.55	4.10	7.90	−0.90
=O	2.30	2.45	4.40	2.05	5.90	−0.60
N-1	3.55	7.40	0.00	1.50	1.10	0.00
C-2	2.65	6.35	0.00	2.10	2.30	0.00
N-3	2.95	5.10	0.00	3.45	2.45	0.00
C-4	4.30	4.90	0.00	4.10	1.25	0.00
C-5	5.30	5.85	0.00	3.55	0.00	0.00
C-6	4.90	7.20	0.00	2.15	−0.15	0.00
N-7	6.55	5.25	0.00	4.55	−0.95	0.00
C-8	6.25	3.95	0.00	5.65	−0.20	0.00
N-9	4.95	3.67	0.00	5.45	1.10	0.00
N-10	5.70	8.20	0.00	1.50	−1.25	0.00

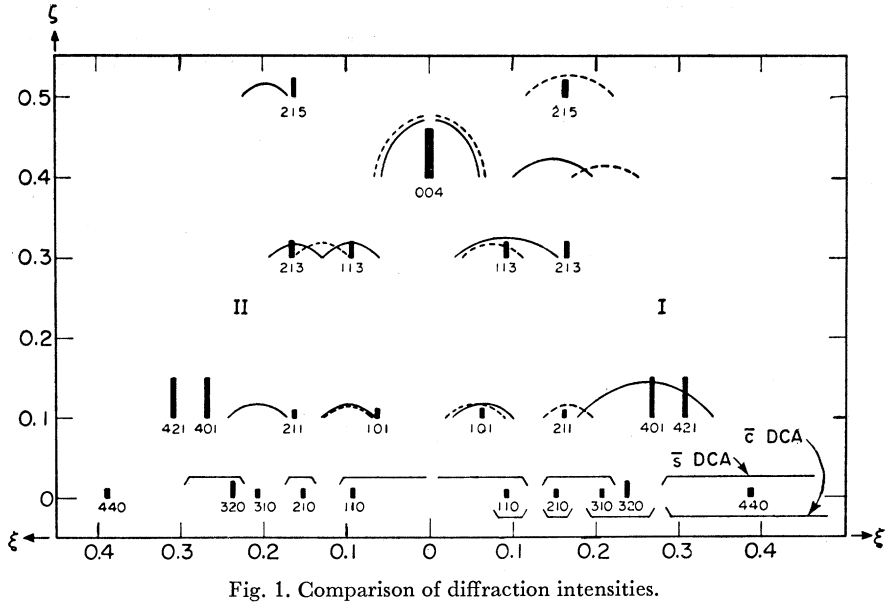

Fig. 1. Comparison of diffraction intensities.

currence of nearly planar H-bonded furanose rings, separated by approximately 5 A and tilted 20° to the fiber axis, is responsible for the great intensity seen in this area. It is these reflections, in fact, which constitute the most striking similarity between the diagrams of adenine polynucleotide and ribonucleic acid (RNA) and their greatest contrast to that of deoxyribonucleic acid (DNA).

Because the x-ray diffraction pattern of adenine polynucleotide resembles that of RNA (*16*) (and differs from that of DNA), it is expected that the structures of adenine polynucleotide and RNA are similar. The form of H-bonded backbone described for structure I above can serve as a basis for the structure of RNA, since any sequence of purines or pyrimidines can be accommodated on either chain and the configuration depends, for its regularity and stability, only on that feature of the chain which distinguishes RNA (and the synthetic polyribonucleotides) from DNA—namely, the hydroxyl group on C′-2 of the sugar moiety. Furthermore, the 6-keto and 6-amino groups of both purines and both pyrimidines would, in this kind of structure, fall at almost exactly equivalent positions. This offers the possibility of their bonding to amino acids or other molecules in a structurally regular way despite their seeming disparity of size. Chargaff and Elson (*17*), who have demonstrated the numerical equality of these groups, have also suggested such a type of bonding (*18*).

RICHARD S. MORGAN*
RICHARD S. BEAR†
Department of Biology, Massachusetts Institute of Technology, Cambridge

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* Present address. Children's Cancer Research Foundation, Boston, Mass.
† Present address: Division of Science, Iowa State College, Ames.

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Role of Parenchyma Cells in Graft Union in Vanilla Orchid

The successful grafting of numerous monocotyledons has been previously reported (*1, 2*). This work demonstrated that grafts could be made in several grass species and in certain monocotyledonous tropical lianas. It was shown that, contrary to accepted usage, a cambium was not essential for graft union but that any meristematic tissue was suitable for this purpose. The principle that any meristematic tissue is capable of forming a union between scion and stock was extended to another monocotyledonous group, the Orchidales, in the study described in this report.

The vanilla orchid of commerce, *Vanilla planifolia* Andr., is susceptible to a root rot, *Fusarium* sp., which has nearly destroyed the industry in Puerto Rico. Attempts were made, therefore, to graft *Vanilla planifolia* on another species, *V. phaeantha* Reichenb., which is resistant to the fungus. The method used was similar to that previously described for grafting lianas (*2*). Like the lianas, *Vanilla* has no definite intercalary meristem, but the actively growing tips remain meristematic. The stem of the *V. phaeantha* stock was broken in the third to fifth internode. A paper tube dipped in paraffin was slipped over the stock, and a scion of *V. planifolia* of equal diameter was inserted into the tube. The scion, also, had been obtained by breaking of the parent stem in the actively growing region. The scion was placed firmly in contact with the stock and tied, to hold it in place. Both intra- and interspecific grafts were attempted. The graft union in the *Vanilla* was different from that in the grasses and lianas in that the process of union was arrested at the point where parenchyma bridges were formed between scion and stock. Two months after grafting of the *Vanilla*, parenchyma bridges were found, but after 2 years there was no evidence of the formation of vascular tissues across the graft union. This was true both of intra- and interspecific grafts. In the grasses, vascular connections were found within 6 to 8 weeks after grafting and in the lianas, within 12 to 16 weeks.

Approximately 5 percent of the *V. planifolia* scions survived for over 2 years. Growth was extremely variable. Some scions began growth within 2 months and grew to a length of several feet, whereas others lived for a year or more before growing. During this time, they retained their original leaves. Growth was slow in the majority of cases. If the nodal roots which hold the plant to its support were permitted to grow into the soil, the stock died, and the scion then persisted on its own roots.

Tests were made in which the bases of scions were dipped in coconut milk, in coconut milk plus one part per million of 2,4-D, and in one part per million of 2,4-D in water solution. Coconut milk appeared to have a beneficial effect, and it was possible to pick out the scions dipped in coconut milk by their better color and vigor. Dipping in coconut milk plus 2,4-D, or in 2,4-D water solution, was deleterious.

Wardlaw (*3*) found that if the shoot apex of certain ferns was isolated from the surrounding leaf primordia and stelar tissue by vertical cuts, it continued to grow and produce both vascular tissue and leaf primordia. No connection took place between the newly formed vascular tissue and that formed prior to the surgery, and all translocation was across parenchyma cells. The experiments reported in the present paper demonstrate that *Vanilla* grafts can survive and grow for at least 2 years on parenchyma unions. These parenchyma cells must, therefore, serve for both upward and downward translocation, since they form the only union between scion and stock. These results suggest the possibility that parenchyma cells could play a similar role in intact plants.

THOMAS J. MUZIK*
Federal Experiment Station, Mayaguez, Puerto Rico

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* Present address: Department of Agronomy, State College of Washington, Pullman.

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Amino Acid Factor in Control of Abscission

The role of indoleacetic acid in the control of abscission of plant organs has been demonstrated repeatedly since Laibach (*1*), in 1933, first observed that orchid pollinia retarded the abscission of debladed petioles. Addicott and Lynch (*2*) have reviewed other factors which may affect abscission, including acidity, ethylene, mineral metabolites, carbohydrate level, auxin gradients, oxygen, and carbon dioxide. Several of these have been shown to exert indirect effects in the control of abscission. The effect of