a prominent linear valley in the Precambrian rocks. The "offset" may represent an original en echelon arrangement, but owing to its regional continuity elsewhere and the logical reconstruction of the unity of the two transverse anticlinal structures, however, I prefer the interpretation that the Ramapo fault was originally continuous and has been displaced.

The supposed right-lateral strike-slip offset of a formerly continuous Ramapo fault amounts to only 6 miles. The apparent discrepancy with the 12-mile offset based on the displaced anticlinal crests can be explained if the Hopewell fault, with its large up-on-the-west dip-slip component of displacement (*4*) is extended northward. Up-on-the-west dip-slip movement on a north-trending vertical fault would offset a formerly continuous northeast-trending, southeast-dipping Ramapo fault to the *south* on the west side of the north-trending fault. Thus, the apparent strike-slip offset of the Ramapo fault would be less than the true strike-slip component (as measured by the anticlines) by an amount equal to the southward displacement caused by dip-slip movement. The magnitude of this southward dip-slip displacement (6 miles, if the 12-mile figure is correct) is controlled only by two variables: (i) the amount of dip-slip displacement on the vertical north-trending fault (Hopewell fault extended) and (ii) the dip of the Ramapo fault. Stratigraphic evidence north of Somerville suggests that the amount of dip-slip displacement on the vertical north-trending fault is approximately 15,000 feet; with this figure, the dip of the Ramapo fault can be calculated to be 31°. Alternatively, if one assumes that the Ramapo fault dips more steeply, say 55°, then the dip-slip displacement on the north-trending fault must be 31,680 feet.

If the Hopewell fault continues northward and contains large strike-slip displacement, as is argued here, then a comparable northward extension and similar strike-slip displacement may also be inferred for the nearby Flemington fault. The case for the Flemington fault is less direct than that for the Hopewell fault, for the only known reference surface which extends across the Flemington fault or its inferred northward extension is the Ramapo fault. The originally continuous nature of this surface is debatable, for the observed offset arrangement northeast of Pittstown and north of Flemington (Fig. 1) might be regarded as an initial en echelon pattern. If, however, one assumes the original continuity of the Ramapo fault here, as north of Somerville, then it appears to have been offset along a fault which lies due north of the termination on Kümmel's map of the Flemington fault (Fig. 1). A relationship exactly similar to the one just described for the supposed northward extension of the Hopewell fault may therefore exist north of Flemington (Fig. 2). Like the Hopewell fault, the Flemington fault shows large up-on-the-west dip-slip displacement (*5*). If only dip-slip movement has occurred on this fault, then a northward extension of it would offset a formerly continuous southeast-dipping Ramapo fault to the *south* on the western block, not to the north, as is observed. This "opposite" effect is taken as suggestive evidence that the Flemington fault extends northward of its previously supposed termination and that substantial right-lateral strike-slip displacement has accompanied the dip-slip displacement on it (*6*).

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Dehydrogenases of Neurospora crassa

Abstract. Malate, isocitrate, glucose-6-phosphate, and 6-phosphogluconate dehydrogenases of the homogenate prepared from the mycelia of several wild strains of *Neurospora crassa* have been subjected to zone electrophoresis in starch gel. Four electrophoretically different malate dehydrogenases, a single isocitrate dehydrogenase, three glucose-6-phosphate dehydrogenases, and two 6-phosphogluconate dehydrogenases were obtained regardless of the strain or the media in which the organism was grown.

The existence of lactate dehydrogenase in electrophoretically different forms has been noted in purified material (*1*). The finding of several components of this enzyme in animal organs with paper electrophoresis has stimulated wide interest in this phenomenon (*2*). The use of electrophoresis in starch gel for the separation of the dehydrogenases of mammalian organs also yielded evidence of multiplicity of enzymes (*3*, *4*). To eliminate the possibility of this multiplicity being caused by indiscriminate mixing of different types of cells in organ homogenates, it has been shown with tissue-cultured mammalian cells that the multiplicity of several dehydrogenases is demonstrable in cytologically uniform material (*4*). To strengthen the evidence obtained by electrophoresis, sufficient quantities of the components of a single dehydrogenase must be secured for other studies. These studies are necessary to establish the chemical relationship between the components. In a survey of microorganisms which can be harvested conveniently in quantity, *Neurospora crassa* was found to exhibit multiplicity of dehydrogenases. This report deals with the experimental evidence of such multiplicity and the persistence of this phenomenon in several strains grown in different culture media.

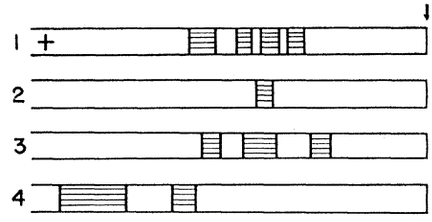

Fig. 1. Dehydrogenases of *Neurospora crassa* mycelia after fractionation by electrophoresis in starch gel and direct visualization of enzymic activity in the gel. Substrates used were: 1, malate; 2, isocitrate; 3, glucose-6-phosphate; and 4, 6-phosphogluconate. Arrow indicates the location of the sample of mycelia homogenate.

The following strains of *Neurospora crassa* were obtained from the American Type Culture Collection: 9279, 12758, 10767, 10336, 10337, 10815, and 10816. Strain 5297 was a gift of H. J. Blumenthal. Minimal medium (*5*), Sabouraud medium (Difco), and a medium containing 5-percent sucrose and 0.5-percent each of malt extract and yeast extract were used. Cultures were grown from conidial inocula in 1 liter of medium in a Fernbach flask rotated at 150 rev/min at 28° to 30°C. Mycelia were harvested after 5 to 10 days, collected on a Buchner funnel over a layer of gauze, washed

thoroughly with distilled water, lyophilized immediately, and kept at −20°C until use. The lyophilized mycelia were ground to a fine powder in a chilled mortar before the addition of buffer. Previously described procedures were followed from then on for the preparation of a homogenate, the application of homogenate to the starch gel column, the electrophoretic separation of the components of dehydrogenases, and finally the direct visualization of the loci of dehydrogenases on gel column (*4*). The only modification made was the use of malate at one-tenth of the concentration previously used to reduce excess precipitation of formazan from neo-tetrazolium in the incubation media. The solution for the direct visualization of 6-phosphogluconate dehydrogenase activity in the starch gel has not been described previously. The substrate concentration used was 0.002*M* and the other constituents were the same as those for the detection of glucose-6-phosphate dehydrogenase. To assure the identity of various bands of formazan, the homogenate prepared with strain 5297 was used as the standard to which the positions of the bands from other strains were always referred.

The results of the electrophoretic fractionation of the dehydrogenases are summarized in Fig. 1. Lactate dehydrogenase and α-glycerophosphate dehydrogenase were not detected. Visual estimation of the color of the formazan precipitate which appears at the loci of specific dehydrogenase activity does not give reliable quantitation of enzyme concentration. However, there seem to be strain differences in the relative intensities of various bands. Otherwise, strain or culture media do not affect the number or location of the bands for each dehydrogenase. Since there is a concentration limit below which activity is not detectable, the observed number of components for each dehydrogenase represents the minimum number present. In spite of the care taken in preserving enzyme activity, it must be assumed that labile components of dehydrogenases, even if present in the organism in sufficient quantities, might also have escaped detection.

It is clear that the heterogeneity of specific dehydrogenases can occur in relatively simple organisms as well as in the mammalian tissues. Results from studies on yeast lactate dehydrogenase support this observation (*6*). The fact that similar patterns of dehydrogenases were observed with different strains and media indicates that there is intrinsic diversity which is not influenced by the external factors tried. One might speculate that this diversity rises as a result of subcellular differentiation or due to limited randomization during the synthesis from subunits of enzyme proteins. Furthermore, this diversity very likely was evolved before the apparent strain difference occurred and was controlled by genes other than those responsible for the characteristics that set these strains apart (*7*).

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Amygdaloid Suppression of Hypothalamically Elicited Attack Behavior

Abstract. Electrodes were implanted in the hypothalamus and in the amygdala of adult cats. Electrical stimulation of the amygdala suppressed attack behavior elicited by the hypothalamic stimulation.

Much experimental evidence implicates the amygdala in the control of emotional behavior (*1*). The present study presents direct evidence that the amygdala modulates the emotional activity of the hypothalamus, supplementing data from ablation (*2*) and unit-activity (*3*) studies, and supporting speculations based on anatomical and theoretical grounds (*4*).

Bipolar electrodes were implanted aseptically in the hypothalamus and in the amygdala of ten cats. Preoperative tests had shown that these cats would not normally attack rats, which were used as attack objects during experimental sessions. Two other cats, which did attack rats normally, were eliminated from the study. The hypothalamic electrodes were aimed at a region known to produce directed attack (*5*, *6*), which served in this experiment as the model of emotional behavior.

Nine of the ten operated cats, when stimulated in the hypothalamus, savagely attacked a rat placed in the cat's cage. The attack ceased immediately when the stimulation was turned off. The electrical stimulation was a train of 62.5 pulse/sec, 2-msec biphasic square waves lasting usually only until the attacking cat touched the rat with tooth or claw. However, in some trials stimulation lasted from 10 to 25 seconds regardless of the cat's behavior; during such trials, several of the cats killed a rat virtually each time before the hypothalamic stimulation was terminated. Between trials, none of the cats appeared to pay any attention to the rats.

The threshold current for eliciting the attack response was generally about 0.30 ma. There was a tendency for this threshold to increase slightly with repeated stimulations.

As reported previously (*6*), two forms of hypothalamically elicited attack were observed: a stalking form characterized by biting and the relative absence of concomitant autonomic signs, and a more "rage-like" form characterized by repeated striking with the paw and a full complement of autonomic signs such as piloerection and salivation. The stalking attack was elicited by electrodes slightly lateral to those eliciting the "rage-like" form. However, amygdaloid stimulation did not seem to act differently on these two forms of attack.

The amygdaloid electrodes, either bilaterally or unilaterally, were connected to a second stimulator identical to that used to stimulate the hypothalamus. For the simultaneous stimulation of amygdala and hypothalamus which was used in this experiment, the pulses from the two stimulators were interlocked so that a 2-msec pulse in one region was followed 6 msec later by a similar pulse in the other region. Temporal separation of the pulses is necessary to avoid stimulating intervening structures.

In five of the nine cats tested, when amygdaloid stimulation was added to the hypothalamic, either the attack occurred after a longer period of stimulation or, on many trials, it did not occur at all. The effective amygdaloid suppressing current, both unilateral and bilateral, was generally 0.15 to 0.25 ma. Histological examinations of the brains of these five animals at the end of the