which movements of subcrustal material are assumed to be taking place.

2) Kato (*2, 3*), in 1933–34, published the results of a considerable number of magnetic observations in the Japanese area and showed quite clearly a direct relation between the occurrence of earthquakes, volcanic eruptions, and local magnetic anomalies. He explained the anomalies as being due to variations in the permeability of the rocks following the rise of temperature resulting from the earthquakes and eruptions. However, in the case of the Sanriku earthquakes of 1896 and 1933, he pointed to the fact that the resulting anomalous fields were in opposite directions, and the first motions of the accompanying tsunamis were also observed to be in opposite sense. This would seem to suggest that the magnetic anomalies were not caused by temperature changes, but by the movement of crustal or subcrustal material. Further support is given to this supposition by the fact that, in the case of the Mauna Loa eruption of June 2, 1950 (*4*), the local magnetic intensity was found to have increased considerably between May 9 and June 9, the rise probably coinciding with the beginning of the eruption. Had the anomalies been due to temperature changes, one would have anticipated a decrease in intensity.

From these observations, it would seem reasonable to infer the possibility of fairly strong magnetic anomalies over areas that are tectonically active.

3) The synthesis of mica for commercial purposes has long been a major problem. Under the pressure of war requirements, intensive research was carried out by Dietzel and others (*5, 6*), at the K. W. I. Ceramics Institute, Ostheim, Germany. It was found that the growth of large sheets of mica, orientated in the required directions, was greatly facilitated by the introduction of a weak magnetic field (13 gauss) at right angles to the length of the crucible containing the melt. The mica formed in large sheets parallel to the magnetic lines of force. Dietzel attributes this effect to the paramagnetism of mica. However, it was found that, although in successive experiments the iron content of the melt was reduced considerably, the degree of orientation was not affected. It is suggested, therefore, that the effect is not due to the paramagnetism of the mica, but to some directional effect of the magnetic field upon the moving ions in the melt.

Taking these various observations together, the tentative suggestion is made that a further variable which should be considered in connection with metamorphic phenomena is that of the local state of the earth's magnetic field. It is clearly possible, from Dietzel's results, that the orientation, particularly of mica minerals, may be affected by comparatively small magnetic gradients; it is suggested further that such conditions may also have some directional effect on migrating ions.

It is emphasized that this suggestion is put forward very tentatively, with the object of encouraging research in these directions, which may prove extremely useful quite apart from the validity or nonvalidity of the hypothesis.

The lines of investigation proposed are:

1) Full and detailed study of magnetic phenomena in a tectonically active area, such as the West Indies, together with field investigation of young metamorphic areas in the same general vicinity.

2) Laboratory experiments to study the effect of magnetic fields on crystallizing minerals. This study might be extended to the examination of the effects of electrostatic fields, too, since it is felt that here also may lie an enlightening field of investigation.

Studies of this sort would be too extensive and varied in their nature for one man to carry through alone, and it is hoped that other persons interested in these topics may be encouraged to undertake and intensify investigations along these lines.

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The Action of Thrombin on Fibrinogen[1]

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Investigations of the author and his associates (*1–10*) aimed at elucidating the role of thrombin in clotting fibrinogen have shown that thrombin is not involved in the polymerization of the fibrinogen molecules, but that thrombin acting as an enzyme makes some alteration on the fibrinogen molecule (*2*). It was found that the enzymic action of thrombin does not involve oxidation or reduction (*3*); thus it is probably a hydrolytic enzyme. Experiments with papain showed that it clots fibrinogen just as thrombin does (*4*), and that it is the proteolytic enzyme itself that clots fibrinogen (*5*). These experiments and the finding that the fibrin molecule, otherwise identical with the fibrinogen molecule (*4, 6, 8–10*), has a different isoelectric point (*6*) suggest that thrombin may split a bond, such as a peptide bond, freeing a small molecule from the fibrinogen, leaving a slightly altered fibrinogen molecule. The final proof of such a mechanism would be the finding of this predicted small molecule in the supernatant after clotting.

Since the probability always exists that a small molecule may be adsorbed to fibrinogen as an impurity and might appear in the supernatant after clotting, iodinated fibrinogen (*7*) was prepared and the supernatant studied for the presence of a sub-

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stance that might have been split off by the action of thrombin. Iodinated fibrinogen differs so much from fibrinogen, and its preparation involves so many additional steps, that it is more likely to be free of possible adsorbed impurities than the original fibrinogen. Working with iodinated fibrinogen has the further advantage that it is soluble in water, clots in water, and its solution can be deproteinized by adding salt to it. It was found that the supernatant of clotted iodinated fibrinogen gave a spot detectable by ninhydrin on paper chromatograms and contained a substance that stimulated isolated frog heart.

The experimental procedure is as follows. Iodinated fibrinogen is clotted with a small amount of thrombin. In a few hours a firm gel is formed. By warming the gel it reversibly liquefies. To the liquefied gel a concentrated Ringer solution is added (final salt concentration corresponds to a Ringer solution) to remove the protein. The supernatant when tested on frog heart in contrast to similar unclotted supernatant stimulates the heart. The nature of this substance found in the supernatant of clotted fibrinogen is under investigation. Lorand (personal communication) believes that it is a peptide. Work is also in progress to find further evidence that this substance is really split from fibrinogen by thrombin and whether it is a specific heart stimulant.

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The Radiation Dose-Response Curve and Bacterial Mutations

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The shapes of the curves obtained when plotting biological response against radiation dose have yielded information on the nature of the reaction. Demerec and Latarjet (*1*), Hollaender *et al.* (*2*), and Mefferd and Wyss (*3*) have shown that some induced mutations increase linearly with dose, some increase exponentially, and still others exhibit breaks in the dose-response curves. The causes of these breaks that have been observed with ultraviolet irradiation are subject to conjecture. We have observed a different type of break which is superimposed on the ultraviolet dose-response curve and also results with x-ray and nitrogen mustard treatment. This is observed when the survivors of the mutagenic action are placed in broth and permitted to grow for several generations before the assay for mutants is made. This is often done with bacteria because with a number of bacterial mutations there is considerable delay between the action of the mutagen and the appearance of the mutations in the population; with the *Escherichia coli* mutation to phage resistance the maximum number is not attained until each organism has made about 10–13 divisions following the application of the mutagenic agent. Consequently, in order to observe the maximum of induced mutants the bacteria surviving mutagenic action are usually placed in a condition favorable to growth before an assay of the mutants is made. With some mutations additional induced mutations appear up to 13 generations after the application of the mutagen; with others no more appear after a very few divisions. It is when these so-called end-point mutants are plotted against the dose of the mutagenic agent that a break in the curve is superimposed on the less drastic break that has often been observed with the "zero-point" mutants. This first came to our attention when doses of ultraviolet light sufficiently large to kill all but a small fraction of *Bacillus anthracis* spores failed to increase the incidence of mutants in a culture grown from the survivors, although lower doses gave readily measurable increases. This was especially evident when rare mutations were sought but also applies to biochemical mutants and streptomycin resistance induced by ultraviolet light as well as when x-rays or nitrogen mustard are the inducing agents.

TABLE 1

EFFECT OF THE NUMBER OF SURVIVORS ON THE INCIDENCE OF END-POINT MUTANTS TO STREPTOMYCIN RESISTANCE IN *B. anthracis*

Inoculum size	Organisms inoculated	Mutants per million
	No irradiation	
.1 cc	1.4×10^6	13.8
.001	1.4×10^4	15.0
.00001	1.4×10^2	14.8
	Irradiated 40 sec	
1.0	2.8×10^5	49.5
.1	2.8×10^4	50.5
.01	2.8×10^3	17.3
.001	2.8×10^2	17.6
	Irradiated 60 sec	
1 cc	4.6×10^4	63
.1 cc	4.6×10^3	20
.01 cc	4.6×10^2	14

In the experiment reported in Table 1 a population of *B. anthracis* was subjected to ultraviolet light as indicated. Then inocula of various sizes were removed from each treatment and planted into broth and permitted to grow for 6 hr. The incidence of streptomycin-resistant mutants in the resulting population in unirradiated cultures was not affected by inoculum