described in detail, has been termed *sensory deprivation,* a term more convenient than accurate.

The first systematic investigation of the effects of confinement on human subjects was carried out at McGill University by Bexton, Heron, and Scott (*2*). In that study the conditions of confinement emphasized the reduction of variability in sensory stimulation. The subjects wore semitranslucent goggles through which they could make out brightness but could not form discriminations. There was constantly present a masking noise which served to minimize the effectiveness of sound from outside the cubicle. Each subject wore cardboard gauntlets that extended from just below the elbow to beyond the fingertips. The subjects were confined under such conditions for various periods of time up to 5 days. It was found that in tests of cognitive ability, continued isolation produced inferior performances, later referred to as deterioration of the intellect (*3*), which fortunately disappeared upon the subject's release from confinement. Confinement also produced in all subjects hallucinations that were mostly auditory or visual, but in one case tactual. Immediately upon being released from confinement, subjects reported difficulty in focusing, as well as a tendency to perceive the environment as two-dimensional, with colors more saturated than usual. They also reported that during confinement there was an inability to sustain extended thought.

The present study is preliminary in nature (*4*) and was not intended as a replication of the McGill study. The confinement conditions are very dissimilar. The Princeton study was conducted in the following manner: Four male college seniors served as subjects under conditions of confinement and isolation for a period of 48 hours. Isolation was provided by a floating room (15 ft by 9 ft), which was lightproof, and through which there was an 80-db sound loss. Subjects were fitted with ear plugs and instructed to make as little noise as possible while they utilized free access to a bed and a chair. The confinement cubicle was only 4 ft by 9 ft, which allowed little activity. Further restriction of activity was provided, as in the McGill study, in the form of cardboard gauntlets. The confinement period was interrupted only for meals, tests, and toilet needs. For meals and tests, subjects were removed from the isolation cubicle to the antichamber of the floating room, where a 15-w red bulb provided illumination when necessary. Lightproof goggles were used when the subjects attended to toilet needs outside the confinement cubicle. During the interruptions, no conversation was allowed except that necessary for the conduct of tests. Smoking was permitted at these times.

Inasmuch as the McGill study had found that confinement affected cognitive ability adversely, it was decided in the present study to test the effects of sensory deprivation on learning rate. The learning tasks were 12-item adjective lists, presented aurally. A subject's ability to learn by the anticipation method with a 2-second interstimulus interval was determined before confinement, after 24 hours of confinement, after 48 hours of confinement, 24 hours after release from confinement, and 48 hours after release from confinement. All tests were conducted in the antichamber of the floating room to render all distractions relatively constant.

Figure 1 presents the effects of sensory deprivation on the ability to learn adjective lists, indicating mean values for the four subjects. The results clearly indicate that the ability to learn adjective lists improves with continued sensory deprivation. That this improvement is not a function of either the particular adjective lists or a learning how to learn is indicated by the performance of a control group.

Attempts to measure the effect of sensory deprivation on suggestion, by the Hull body-sway technique, proved unsuccessful.

Upon release from confinement, the subjects were required to give full accounts of the confinement experience, which was recorded on tape. They were then questioned about any of the following items that they had not mentioned: hallucinations, focusing difficulty, lack of ability to engage in extended thought, increased saturation of hues, and lack of three-dimensional perception. In each case, the subject's report was contrary to the McGill findings.

It would be unwise to stress the differences between the McGill and Princeton findings in view of the few subjects used in the latter case. However, it is possible that the differences in the confinement conditions were in part responsible for the divergent results. For example, it may have been that hallucinations were actually generated in the McGill study by the amorphous visual stimuli or the repetitious masking noise or both. Furthermore, these same stimuli may have served as distractions contributing to the decrease in the general mental performance of the McGill subjects.

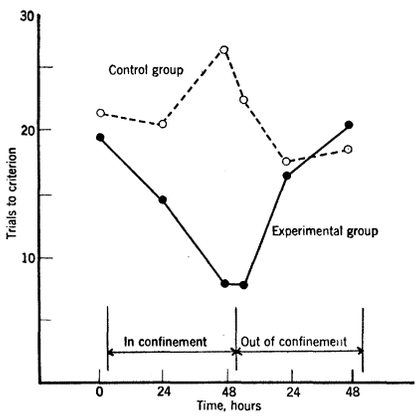

Fig. 1. Effect of sensory deprivation on learning rate in human beings. Each point is the mean value of four subjects.

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Nature of Protein Synthesis in Sweetpotato Infected with Ceratostomella fimbriata

An increase in respiratory rate is commonly observed in the tissues of seed plants when they are infected by pathogens (*1*), and we have found that in sweetpotato infected with *Ceratostomella fimbriata* (black rot), the rate of respiration of the sound tissue adjacent to the area in which the pathogen has developed is higher than that of a control (uninfected sweetpotato). The increase was accompanied by anabolic events, as shown by the accumulation of organic phosphate (P_o) and the synthesis of protein (*2*).

A description of a possible mechanism for the respiratory increase follows. As a result of the penetration of the fungus, the protoplasm of the sound tissue next to the infected area is stimulated to synthesize P_o and protein, and, concomitantly, the reaction

$$ATP \rightarrow ADP + P$$

is accelerated, resulting in an increase in respiratory rate. The details of this metabolic stimulation and protein synthesis are examined in this report (*3*).

To determine the pattern of protein synthesis, slices (1 cm thick) were cut from sweetpotatoes. Some of the slices were inoculated with a spore suspension of *C. fimbriata*; the rest served as a control sample. The sound tissue adjacent to the infected areas in the inoculated sample, which is referred to as tissue A, was removed after 48 hours. Sound tissue (referred to as tissue B) was also ob-

Table 1. Amounts of RNA-phosphorus and protein in the cytoplasmic fractions of sliced sweetpotato tissue. Values are expressed in micrograms per gram of fresh tissue.

Measurement	Mw	Pw	S_2	Total
RNA-P of sound tissue from infected slices (A)	1.84	3.54	13.0	18.38
Protein of sound tissue from infected slices (A)	88.0	74.5	390.0	552.5
RNA-P of sound tissue from uninoculated slices (B)	1.74	2.01	14.5	18.25
Protein of sound tissue from uninoculated slices (B)	66.7	47.3	309.0	423.0

tained from the control sample. Both kinds of tissues were then treated in the following fashion.

The tissues were homogenized with 0.25*M* sucrose solution and, after a low-speed centrifugation to remove sediment such as nuclei, starch granules, and the debris of cell walls, the supernatant was fractionated according to Schneider's method (*4*) into three parts: mitochondria (Mw), microsomes (Pw), and supernatant (S_2). Protein nitrogen and ribonucleic acid (RNA) were determined by micro-Kjeldahl digestion and by the modified method of Ogur (*5*), respectively. As is shown in Table 1, protein nitrogen in every fraction of the tissue was higher in the sound parts of infected slices (tissue A) than in corresponding tissue from untreated control slices (tissue B), but the total amount of RNA was the same. However, it can be seen that in tissue A the amount of RNA in the smaller particles (designated as the microsomal fraction) had increased at the expense of the supernatant. Recent studies have led to the conclusion that the microsomes, rich in RNA, are the site of all of the protein synthesis in the cells (*6*). On the basis of these investigations, it may be suggested that the growth of the pathogen in the sweetpotato leads in some manner to a stimulation of synthetic activity (as shown by the large increases in protein in each cell fraction) in adjacent parts of the sweetpotato tissue that are not infected.

The possibility that the observed increases in protein might be due in part to the synthesis of enzyme protein was investigated by comparing the oxidative activity of mitochondrial fractions prepared from tissues A and B.

It is clear (Table 2) that tissue A was superior to tissue B by each of the criteria utilized. The rate of oxygen uptake in mitochondria from the sound part of infected slices was about twice that in those from uninfected slices, and phosphorylative activity was also greater. There was a greater amount of protein nitrogen in the preparation from tissue A, but despite this, the activity of the mitochondria of tissue A (expressed as oxygen uptake per milligram of nitrogen, per hour), was higher than in those from tissue B. The greater intrinsic activity of the mitochondria may be one factor contributing to the higher respiratory rate that has been observed in the intact tissue from which the mitochondria were derived. The P/O value for oxidative phosphorylation was higher in tissue A than in tissue B, but it was lower in both cases than it was in the mitochondria prepared from the tissue of fresh sweetpotato (that is, one which had remained unsliced and untreated). This might be attributed to difficulties experienced in the preparation of the mitochondria which results from the oxidation of polyphenols by a polyphenol oxidase that increases in the tissues when the sweetpotato is sliced or infected (*8*).

It would thus appear that increases in the amount of mitochondrial enzymes (functional protein) do occur in parts of slices adjacent to infected areas and contribute to the protein recovered in this fraction. In addition, however, the intrinsic activity of the mitochondria is apparently increased.

Table 2. Respiratory oxidation and oxidative phosphorylation by mitochondria from sweetpotato. The reaction mixture contained 20 μ*M* phosphate buffer at *p*H 7.2; 200 μmoles of sucrose; 20 μmoles of α-ketoglutaric acid; 100 μmoles of glucose; 3 μmoles of ATP, 10 μmoles of $MgCl_2$; 10 μmoles of Versene; 20 μmoles of NaF; and 1 ml of enzyme in a total volume of 2.3 ml. The mixture was incubated in Warburg vessels for 60 minutes at 30°C. The mitochondria were prepared according to a previously described method (*7*), using 45 g of each tissue, and finally they were suspended in 3 ml of the 0.5*M* sucrose solution containing 0.02*M* phosphate buffer and 0.2*M* NaF as a phosphatase inhibitor.

Source of mitochondria	Uptake (μatom/flask, per hr)		P/O	Protein N (μg/flask)	O_2 uptake (μatom/mg of N, per hr)
	O_2	P			
Tissue A (sound) from infected slice 48 hr after cutting	6.7	3.6	0.54	577	11.6
Tissue B (from uninoculated control slice) 48 hr after cutting	3.6	0.59	0.16	419	8.6
Tissue A 72 hr after cutting	7.61	3.2	0.42	595	12.8
Tissue B 72 hr after cutting	2.77	0	0	426	6.5
Fresh sweetpotato	2.0	2.15	1.08		

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New Insoluble Active Derivative of an Enzyme as a Model for Study of Cellular Metabolism

It has long been recognized by those interested in cytochemistry and cell physiology that an interface would be the preferred model structure for the study of certain enzymatic reactions (*1*). If the enzyme forms the surface of a stationary phase and the substrate is dissolved in the adjacent liquid or mobile phase, the system simulates conditions inside a living cell (*2*). Other conditions which are imposed on such an ideal working model are that the enzyme is not inactivated in forming the model and that the model responds to environmental changes.

In these studies, catalase was combined with cellulose anion exchanger; this combination was enzymatically active. It is believed that this is the first recorded demonstration of an active derivative of an enzyme resulting from the electrostatic interaction of the enzyme and an insoluble polyelectrolyte. The new derivative is remarkable because of the ease with which the soluble enzyme can be liberated.

The new catalase-cellulose form can be easily demonstrated with crude or crystalline preparations of catalase and