derived from the primary ones. The most noteworthy facts about the 180 mutants studied are that all 10 of the primary ones grew better than 17D4 on uranium nitrate medium, that none of the 180 have lost any essential growth factors, and that some of them exceed 17D4 in certain characters such as color and rate of growth.

Studies with monosporous isolates of the cream variety of the common cultivated mushroom, *Agaricus campestris*, have yielded similar results. Uranium nitrate and other uranium salts have induced mutants whose growth on artificial media is from 5 to 7 times that of the original lines, as determined by dry weight of mycelial mats. Moreover, spawn of some mutant lines has produced mushrooms earlier than that of the checks, and the color was white instead of brownish.

The agar containing uranium nitrate is mildly radioactive, as determined by Dr. Alexander Hollaender, Oak Ridge National Laboratory.

It is suggested that the addition of uranium nitrate, or other similar salts, to nutrient media may be a simple and useful means for inducing desirable mutations in at least some microorganisms.

Changes in the Blood Following Exposure to Gaseous Ammonia

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The use of ammonia as a solvent and as a reagent has opened a new field, and many chemical industries are using anhydrous liquid ammonia in large quantities. Because of leaks in reactors and the transfer of ammonia, the concentration of gaseous ammonia in the air can be of such amounts as to have definite physiological effects upon long exposure. Such has been the case in these laboratories, where investigations utilizing anhydrous liquid ammonia have been carried out over a period of 15 years. Students have noticed such physiological effects as initial exhilaration and increased frequency of respiration with subsequent exhaustion lasting several hours after exposure. There seems to be no lasting detrimental effect, however.

It was thought that it would be interesting and profitable to collect some data pertaining to the accumulation of ammonia by the blood through breathing as a by-product of these investigations.

It has been known for some time that inhalation of ammonia lowered the blood pressure, but no quantitative data pertaining to this are available. A study has been made to show how the blood pressure varies with time when breathing a constant concentration of the gas. In this paper is also presented the change in the NPN (non-protein nitrogen) and the carbon dioxide-combining power of the blood plasma.

In these studies the air in the room was kept at a constant concentration of ammonia, varying less than 30 ppm over 4 hrs. The ammonia concentration was determined twice during this time, at the beginning and at the end. Samples of air were drawn into an evacuated flask, the volume computed to 760 mm, 25 ml of water added, and the solution titrated with 0.1 normal acid. The concentrations of ammonia in the air varied between 530 and 560 ppm.

TABLE 1

1. VARIATION OF NPN AND AMMONIA WITH TIME OF BREATHING GASEOUS AMMONIA

Time of breathing (hrs)	NPN (mg %)	NH_3 (mg %)	Urea (mg %)	Creatinine (mg %)
Normal	27.0	00.0	15.0	1.5
1	37.0	12.1	15.0	1.6
2	45.0	21.9	15.0	1.6
3	50.0	27.9	15.0	1.6
4	57.0	36.4	15.0	1.6

2. CHANGE IN THE NPN AND AMMONIA WITH TIME AFTER CESSATION OF BREATHING GASEOUS AMMONIA FOR 3 HRS

Normal	27.0	00.0	13.0	1.5
1	47.5	24.9	13.0	1.5
2	40.5	16.4	13.0	1.5
3	32.5	6.6	13.0	1.5

The subject, the senior author, remained in contact continuously with the air-gas mixture. Samples of his blood were drawn at regular intervals from a vein in the

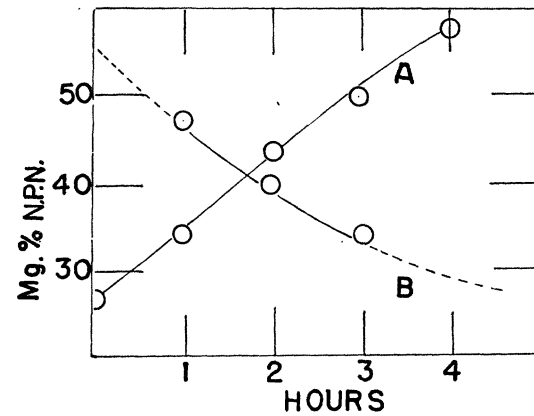

FIG. 1. Rate of change in the blood NPN upon exposure to ammonia: (A) absorption of ammonia by the blood, (B) elimination of ammonia by the blood.

arm and were analyzed for NPN, urea, and creatinine according to the method of Folin and Wu (*1*). The carbon dioxide-combining power of the blood was determined by the method suggested by Van Slyke and Cullen (*2*). pH determinations of the whole blood were made before and after breathing ammonia for 3 hrs, by means of a quinhydrone electrode. No significant change was noticed (7.35; 7.29).

As shown in Table 1 and Fig. 1, the NPN and ammonia vary regularly with time, while the urea and creatinine content of the blood show no variation what-

soever. Upon cessation of breathing ammonia, the NPN drops regularly to the normal value, but at a slower rate than it was absorbed. It was thought at first that there would be an increase in the urea content of the blood through conversion of ammonium carbonate into urea, but such does not seem to be the case. An examination of the urine for excess urea and ammonium salts over the normal gave no further clue. The amount of increase in the urea or ammonium salt formed by the ammonia inhaled would be insignificant compared to the normal amount excreted in a 24-hr sample.

TABLE 2

CARBON DIOXIDE-COMBINING POWER OF THE BLOOD PLASMA

Time (min)	Volume (% CO_2)
Series I	
0	56
120	57
180	57
Series II	
0	52
90	48
Series III	
0	52
60	53
120	50

The carbon dioxide-combining power of the blood plasma apparently is not impaired by the accumulation of ammonia, as is illustrated in Table 2.

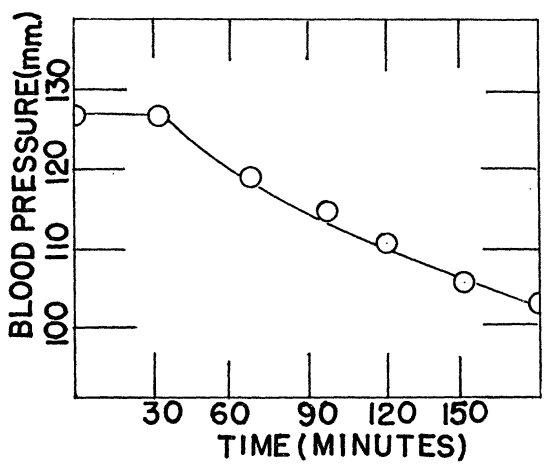

FIG. 2. Change in blood pressure with time upon exposure to gaseous ammonia.

The pulse rate, determined at regular intervals during each experiment, was found to be constant. Blood pressures taken on the subject showed a regular drop after the first 35 min of inhalation. A typical determination is shown in Fig. 2.

No attempt is made to explain the mechanism of the absorption of ammonia by the blood. The authors feel sure, however, that it is a chemical process and not a physical one. It seems to be a second-order reaction. The data are presented in the hope that more work will be done on this problem.

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Inhibition of Mitotic Poisoning by *meso*-Inositol[1]

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The studies briefly reported here stem from several observations recorded in the literature. The strong insecticidal agent, γ-hexachlorocyclohexane (Gammexane) has a cytological effect on *Allium Cepa* similar to that of colchicine. It produces the phenomenon known as c-mitosis, which is characterized by the arrest of nuclear division in the metaphase and, on longer exposure, formation of c-tumors. Other isomeric hexachlorocyclohexanes are either ineffective or only slightly active (*5*). The

TABLE 1

TUMOR FORMATION

Medium* (mM/liter)		c-Tumors†	Medium* (mM/liter)		c-Tumors†
Mitotic poison	Inhibiting agent		Mitotic poison	Inhibiting agent	
0.025 C	...	− (2)	0.035 G	...	+ (4)
0.25 C	...	+ (4)	0.35 G	...	+ (3)
0.25 C	0.33 mI	± (2)	0.035 G	0.33 mI	± (3)
0.25 C	3.3 mI	− (6)	0.035 G	3.3 mI	− (5)
0.25 C	2.0 S	+ (2)	0.35 G	3.3 mI	± (3)
0.25 C	3.3 dI	+ (2)	0.035 G	3.3 dI	+ (2)
....	...	− (5)	0.035 G	2.0 S	+ (3)
....	0.66 mI	− (2)	....	0.25 S	− (2)

* C = colchicine; G = Gammexane; mI = *meso*-inositol; dI = *d*-inositol; S = D-sorbitol.

† + = tumors on all roots; ± = small tumors on some of the roots; − = no tumors. The figures in parentheses indicate the number of bulbs examined.

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