Moh's hardness of massive sodium chloride is 2.

Unfortunately, no data are available on the tensile strength of diamond, but, judging from the examples I have given, the ideal hardness should be at least several times the observed value. Tabor shows measured values for the indentation hardness to be 8000 to 10,000 kg/mm² for diamond. This yields a tensile strength of 4 to 5 × 10^6 lb/in.², a truly remarkable value.

It seems obvious that greatly improved abrasives might be made from relatively inexpensive substances such as aluminum oxide and silicon carbide. These materials in ideal form might be harder than diamond. The attempt to make substances in ideal form for abrasives has several attractive features as compared with an attempt to make massive pieces ideally hard, or with attempts to make superstrong metals: (i) large pieces are not required—small grains can be used as an abrasive powder; and (ii) the consequences of transformation to the non-ideal form are less severe.

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Lactic Dehydrogenase Activity of Serum in Mice with Transplantable Leukemia

The finding that the serum activity of lactic dehydrogenase (LD) is elevated during acute and chronic human leukemia (*1*) prompted the study reported here of the alterations of serum lactic dehydrogenase (SLD) during the course of mouse leukemia (*2*). The purposes of this study were (i) to ascertain the relationship, if any, between SLD and the course of experimental mouse leukemia and (ii) to determine whether there was any correlation between SLD and the various types of experimental transplantable mouse leukemia.

Four lines of leukemia in mice were examined to determine whether alterations in the activity of SLD occurs during the course of the disease. Lines I, 8174, and 82B are acute stem-cell leukemias probably lymphocytic in character. Line C1498 arose spontaneously as a myeloid leukemia. Line-I leukemia, carried in C58 mice, was supplied to the Sloan-Kettering Institute by E. C. MacDowell and has since been kept in passage in F1 (C58 by BALB) mice by the intraperitoneal inoculation of 10 to 20 million cells from minced spleen of leukemic donors. The survival time of the inoculated animals is 4 to 7 days.

Table 1. Serum lactic dehydrogenase activity in mice inoculated intraperitoneally with a suspension of specified leukemic cells.

Days of inoculation	SLD* (units/ml)			
	82B	C1498	I	8174
1	1000	1200	3200	2500
2	1900	1600	6200	2900
3	3000	3300	24,000	5300
4	5400	5500	no survivors	7100
7	3700	3200		6300
8	2900	4700		10,000
9	3700	7200		15,000
10	3900	5200		18,000
11	3200	6200		40,000
15	11,000	14,000		25,000†
16	20,000	44,000†		no survivors
17	no survivors	no survivors		no survivors
Control mice	1100 ± 300 (F1)‡	1200 ± 260 (C57 BL/6)§	1100 ± 300 (F1)‡	1100 ± 300 (F1)‡

* Average of two individual determinations. † One mouse only. ‡ Average of determinations on 23 mice. § Average of determinations on 21 mice.

Leukemia 8174 arose spontaneously in an F1 (C58 by BALB) ex-breeder in the laboratory of Joseph Burchenal. It was converted to ascitic form and is passed in the same strain of mice by the intraperitoneal inoculation of ascitic fluid containing approximately 20 million cells. Survival time is 12 to 16 days. Leukemia 82B also originated in an F1 (C58 by BALB) ex-breeder in Burchenal's laboratory. It is transferred into the same strain of mice by the intraperitoneal inoculation of approximately 10 million cells from minced tumor of leukemic donors. Survival time is 12 to 16 days.

Leukemia C1498 arose as a spontaneous myelocytic leukemia in C57 BL/6 mice. At the present time, however, it is not considered histologically typical of that type of leukemia. This strain was received from Jackson Laboratory, Bar Harbor, Maine. It is transferred in mice of C57 BL/6 strain by inoculation of approximately 10 million cells from minced tumor of leukemic donors. Survival time is 10 to 16 days.

A designated number of mice were inoculated with each type of leukemia. An equal number of untreated control mice of C57 BL/6 and F1 mice were set aside. At daily intervals, two mice from each leukemic group and two from each control group were bled from the branchial artery with syringes that had been moistened with saline before use. The serum was separated from each sample, and individual analysis of SLD was made. The average of each two determinations was recorded.

Serum lactic dehydrogenase activity was measured by the method previously described (*1*). The reaction was followed in a Beckman model DU spectrophotometer using a tungsten light source. The activity is expressed as units per milliliter of serum, per minute. One unit equals a decrease in optical density of 0.001 per minute, per milliliter under the conditions described. The mean value of SLD, based on individual determinations on the serum of 23 normal F1 (C58 by BALB) mice, was 1100 ± 320. The mean value, based on the determination of the serum of 21 normal C57 BL/6 mice, was 1200 ± 260.

In mice inoculated with line-I leukemia, which is associated with the shortest survival time of those in this study, an increase in SLD was apparent within 24 hours after injection. Terminally, SLD rose to 24,000 units. Those mice inoculated with the cells of leukemia 8174 showed a slower but progressive rise in SLD activity, reaching a maximum terminally. Inoculation with C1498 and with 82B leukemia resulted in similar increments of SLD activity (Table 1).

The mechanism of the elevation of SLD during experimental mouse leukemia and human leukemia has yet to

Table 2. Comparison of lactic dehydrogenase activity of selected tissue homogenates from control and leukemic mice.

Tissue	Lactic dehydrogenase activity (units/gram wet tissue)	
	Untreated control*	Leukemic†
Serum	760	60,000
Lymph node	80,000	280,000
Liver	130,000	280,000
Spleen	100,000	230,000

* F1 mouse. † F1 mouse inoculated 16 days previously with leukemia 82B.

be explained. Lactic dehydrogenase activity was measured in homogenates of various tissues from an untreated control F1 mouse and an F1 mouse that had been inoculated with leukemia 82B 16 days previously. The SLD of the control mouse was 760 units and that of the leukemic mouse 60,000 units. Table 2 compares the LD activity per gram of wet tissue in these two mice. The serum levels present the most striking differences between the leukemic and nonleukemic mouse. A comparison of the LD activity of the other tissues reveals higher values in the homogenates of the viscera of the leukemic mouse, although the differences are not as impressive as those observed in the serum.

The observations on various mouse leukemias reported here indicate that mouse leukemias, like some human leukemias, result in an increase in SLD. The mechanism of the increased serum activity is unknown.

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References and Notes

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Evolution of Respiratory Enzyme System during Germination of Bacillus subtilis

Church and Halvorson (*1*) reported that intact spores of *Bacillus cereus,* following activation by heat, oxidize glucose, gluconate, 2-ketogluconate, and pyruvate, but fail to oxidize glucose-6-phosphate, fructose-6-phosphate, hexose diphosphate, acetate, ribose, or fructose. On the other hand, cell-free extracts of spores of the same species which have been prepared by sonic oscillation or by grinding oxidize glucose and pyruvate but not gluconate, 2-ketogluconate, glucose-6-phosphate, fructose-6-phosphate, or hexose diphosphate. Hardwick and Foster (*2*), after working with cell-free extracts of spores from several aerobic bacteria, reported an absence of any measurable respiratory enzymes.

In a previous report (*3*), we showed that spores of *Bacillus subtilis* (PCI 219) develop into vegetative forms after passing through four separate phases—namely, resting spores, appearance of translucent area in spores, formation of germ pores, and emergence of a new bacillus. In this report, it is observed that respiratory-enzyme systems are formed according to the phase of development of spore germination and that respiratory-enzyme systems of germinating spores differ from those present in vegetative forms.

Spores of *B. subtilis* (PCI 219) were harvested from a 5-day culture grown on a meat-extract agar medium at 37°C, heated at 80°C for 20 minutes, and then washed in distilled water three times and resuspended in distilled water. The spores of this suspension were used as resting spores for the experiment. Oxygen uptake by spores was measured in a Warburg manometer at 37°C.

In the presence of L-asparagine, the resting spores showed an increased uptake of oxygen when caramelized glucose was used as the substrate instead of the unheated glucose. It was previously reported (*4*) that a significant number of spores of *B. subtilis* (PCI 219) will change to the translucent spore stage from the resting stage if the phosphate-buffer solution contains a caramelized sugar (glucose, fructose, maltose, lactose, or sucrose) instead of unheated sugar with one of the amino acids (L-asparagine, DL-isoleucine, DL-serine, or DL-valine).

It is apparent that the factors which favor translucent-spore formation as previously reported (*4*) also result in an increased oxygen uptake by the resting spores. In other words, resting spores change to the translucent spores in a phosphate buffer that contains caramelized glucose and L-asparagine; they also show an oxygen uptake. On the other hand, almost none of the resting spores in a phosphate buffer that contains unheated glucose and L-asparagine change to the translucent forms or show a measurable oxygen uptake. An examination to determine whether translucent spores are able to oxidize unheated glucose was therefore conducted.

Translucent spores were prepared by incubating the resting spores in the phosphate solution containing caramelized glucose (1 percent) and L-asparagine (1.5 mg/ml) for 1 hour at 37°C. These translucent spores were washed in distilled water three times and resuspended in distilled water. Manometric data are given in Fig. 1, where results clearly indicate that the translucent spores rapidly oxidize unheated glucose.

To determine the pathways of oxidation of glucose by the translucent spores, two experiments were performed. In one, some intermediates of glycolysis and of the tricarboxylic acid cycle were used as substrates, and, in the other, some inhibitors were used. Manometric data from these experiments are give in Table 1. These data indicate that the translucent spores do not oxidize fructose-1, 6-diphosphate, pyruvate, citrate, succinate, or malate and that the oxidation of glucose is not inhibited by HCN or NaF. These facts show that glucose oxidation by the translucent spores may not proceed on the pathways of Embden-Meyerhof and the tricarboxylic acid cycle.

Table 1. Oxygen uptake by the translucent spores in the presence of various intermediates of the tricarboxylic acid cycle and the Embden-Meyerhof pathway. The Warburg vessels contained 0.5 ml of translucent spore suspension, 0.2 ml of substrate, 0.1 ml of inhibitor, and 1.5 ml of 0.1*M* phosphate buffer at *p*H 7.2 and 37°C in a fluid volume of 3 ml.

Substrate	Inhibitor	QO_2 (*N*)
0.1*M* Glucose		138.84
0.1*M* Malate		42.65
0.1*M* Pyruvate		44.47
0.1*M* Succinate		42.22
0.1*M* Citrate		38.12
0.1*M* Fructose-1, 6-diphosphate		61.41
Endogenous		39.53
0.1*M* Glucose	0.01*M* HCN	139.14
0.1*M* Glucose	0.01*M* NaF	151.80

Vegetative forms of *B. subtilis* are able to oxidize glucose-6-phosphate, fructose-1,6-diphosphate, pyruvate, malate, or succinate. Moreover, these oxidations are inhibited by HCN and NaF. It seems, therefore, that glucose oxidation in translucent spores differs from that in vegetative forms.

To determine the pathways of glucose oxidation by the translucent spore, we used sodium gluconate as a substrate first. The results are shown in Fig. 1. It is clear

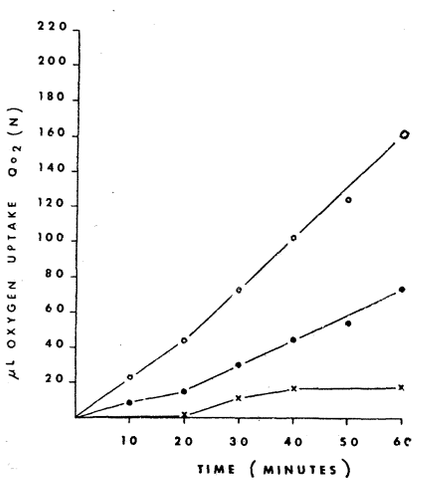

Fig. 1. Unheated glucose and sodium gluconate oxidation by translucent spores: ○—○, glucose; ●—●, gluconate; ×—×, endogenous. The Warburg vessels contained 0.5 ml of translucent spore suspension, 0.2 ml of substrate, and 1.5 ml of 0.1*M* phosphate buffer at *p*H 7.2 and 37°C in a fluid volume of 3 ml.