Table 2. THE EFFECT ON PYRUVIC OXIDASE ACTIVITY OF ADDITION OF CO-CARBOXYLASE TO NORMAL AND VIRUS-INFECTED BRAIN PREPARATIONS

Brain preparation	No. of observations	Mean optical density*		Increase (per cent)	P
		No addition	+ Co-carboxylase		
Normal Hæm. A infection	9	0.3502	0.4220	17	0.03
	9	0.2139	0.3364	37	<0.001

* A lower enzyme activity was consistently recorded when pyrophosphate buffer was used in place of Sørensen's phosphate buffer.

present in the brain homogenate. The results of a typical experiment are summarized in Table 2.

It is perhaps relevant to note that it has long been recognized that many of the terminal signs of virus encephalitis resemble those of experimental vitamin B₁ deficiency, and although the depletion of co-carboxylase is not so profound in these as has been observed in experimental B₁ deficiency, it seems nevertheless possible that this observed depletion contributes to the terminal signs of virus encephalitis.

Spizizen³ has recently reported that in phage-infected *E. coli* the pyruvate metabolism falls to zero, but it can be restored by the addition of co-carboxylase.

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Ineffectiveness of Selenium in the Treatment of Nutritional Muscular Dystrophy in the Rabbit

It has been found recently that selenium is effective in the prevention of two experimental syndromes induced by feeding a vitamin E-deficient diet. These are exudative diathesis in the chick (Stokstad, E. L. R., private communication) and necrotic liver degeneration in the rat¹. Factor III, an unidentified substance in addition to vitamin E and cystine having protective activity against liver necrosis, has been identified as selenium. These reports pose the question whether selenium may have tocopherol-replacing properties with respect to muscular dystrophy, the classical symptom of vitamin E deficiency in the young of most species.

Eight New Zealand white rabbits were fed a tocopherol-deficient diet containing 35.4 per cent 'Cerelose', 37 per cent corn starch, 15 per cent casein, 5 per cent molecular distilled lard, 4 per cent salts, 3 per cent cellulose and a complete vitamin mixture except for vitamin E. Creatine and creatinine in the urine were determined at intervals according to a modification of the procedure of R. S. Hare².

After six weeks on the depletion diet the animals exhibited the well-known symptoms of muscular dystrophy and an increase in the creatine: creatinine ratio to 2-4:1. Six rabbits were treated intramuscularly with either 20 µgm. or 100 µgm. selenium per day as sodium selenite. Administration of this compound for periods up to 17 days elicited no response in the symptoms of dystrophy or in the level of creatine excretion. Administration of DL-α-tocopheryl acetate orally at a rate of 15 mgm. per

day to two controls induced a remission of symptoms and creatinuria in about five days.

These results indicate that nutritional muscular dystrophy is of more specific etiology than exudative diathesis or liver necrosis.

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Induction of Mouse Leukæmia with Purified Nucleic Acid Preparations

RECENT studies by Gross¹ and others^{2,3} have shown that leukæmia with immunogenetic characteristics of the recipient strain can be induced in certain low-leukæmia-incidence strains of mice by injecting into newborn animals cell-free extracts of tissues of certain high-leukæmia strains. Although the active leukæmogenic agent has been referred to as a virus, direct evidence on this point has been lacking. At present it can be said only that the agent is filtrable and thermolabile, and is transmitted in certain families of mice from one generation to another directly through the embryos. Under certain poorly defined conditions, cell-free extracts of leukæmic organs may also produce parotid gland tumours, sarcomas and perhaps other neoplasms, indicating that the active agent is either multipotent or that extracts contain several oncogenic agents.

It is evident, therefore, that while this phenomenon could be due to the transfer of discrete infective virus particles (here defining 'virus' in conventional terms which exclude the so-called provirus), it could also be visualized either as a process analogous to transduction by an agent resembling lysogenic bacteriophage (wherein the active nucleic acid would reside within a more complex deoxyribonuclease-resistant particle) or to bacterial transformation (wherein the active agent is free deoxyribonucleic acid).

The present report describes a series of experiments in which the incidence of leukæmia was apparently increased in a hybrid strain by injection into newborn mice of purified nucleic acid prepared from the lymphoid tissues of a high-leukæmia donor strain.

In a preliminary experiment, cell-free extracts were tested according to the method of Gross. 'Selas'-filtered extracts of the leukæmic tissues of AKR mice were injected into newborn mice of two low-leukæmia strains, C3H_f/Gs and C57BR/cd, and into C3H_f (female) × AKR (male) F₁ hybrids. The results shown in Table 1 essentially confirm the basic observations of Gross.

In the next experiment (Table 2), mice were injected under similar conditions with saline suspensions of deoxyribonucleic acid and ribonucleic acid that had been isolated from non-leukæmic and leukæmic organs of AKR mice. The nucleic acids were isolated by a general method reported to yield active transforming deoxyribonucleic acid from pneumococci⁴ and to disrupt tobacco mosaic virus⁵ and various *E. coli* bacteriophages⁶. Frozen, freshly collected lymphoid tissues (spleens and lymph nodes) were homogenized in 0.01 M versene at pH 7 and treated with 15 per cent sodium dodecyl sulphate