TISSUE INCUBATED FOR 2 HR. IN BICARBONATE-RINGER (N_2/5% CO_2).

Tissue	mgm. dry weight	Substrate	Q Aceto-acet.	$Q\beta$-Hydr-oxybutyr.
Rat brain	15·29 19·92	acetoacet. (0·002 M.) ,, + oxaloacet. (0·0067 M.)	—0·49 —0·55	0·23 0·37
Rat testis	33·62 44·39	acetoacet. (0·002 M.) ,, + oxaloacet. (0·0067 M.)	—0·98 —0·69	0·38 0·35
Rat kidney	14·39 9·76	acetoacet. (0·002 M.) ,, + oxaloacet. (0·0067 M.)	—1·57 —1·64	0·81 0·85

The reaction acetoacetic acid + oxaloacetic acid → citric acid + H_2O does not require oxygen. According to Breusch[1], the enzyme is insensitive to cyanide. One would have expected, therefore, that addition of oxaloacetic acid would raise the anaerobic rate of acetoacetic acid disappearance to the level of the aerobic rate (Q-value of about 6 for kidney and about 2 for brain). The addition of oxaloacetic acid was, however, without effect.

Moreover, it was found in these experiments that the ratio of acetoacetic acid disappearing to β-hydroxybutyric acid formed was roughly 2 : 1, a result typical for a dismutation, and it was concluded that the first step of acetoacetic acid metabolism is oxidative, or at least reversibly linked with an oxidative step. This, too, was not in agreement with a mechanism where the first step would have been a non-oxidative condensation. Subsequent investigations[5,6] gave no evidence for oxidation at the α- or the γ-position or for an oxidative cleavage into two C_2-compounds.

Though the limited value of negative evidence is fully realized, it remains to be explained why the expected results were not observed under the conditions employed.

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Effect of Temperature on the Reducing Activity of Leucocytes in Milk

SOME investigations were carried out as to the possibility of milk from cows infected with mastitis having an increased reducing activity due to the number of leucocytes contained in it, and in consequence failing the Standard Routine Resazurin Test adopted by the Ministry of Agriculture for the grading of milk under the National Milk Testing and Advisory Scheme. During the course of this work, milk samples with a high leucocyte content were submitted to ten-minute and one-hour resazurin tests at 37° C. immediately after production, and then again after holding the milk overnight at different temperatures, so as to attempt to reproduce the varying atmospheric temperatures at which milk is held before testing.

As will be seen from the accompanying results, milk held at 55° F. overnight did not reduce resazurin so rapidly as when tested immediately after production. This is no doubt due to the fact that leucocytic metabolism in milk is entirely catabolic and not anabolic ;

VARIATION IN THE RATE OF REDUCTION OF RESAZURIN DUE TO HOLDING THE MILK AT DIFFERENT TEMPERATURES.

	Resazurin disk reading at the end of		Complete reduction of resazurin
	10 min.	1 hour	
Tested immediately after production	4	0	1 hr.
Tested after holding at 55° F. overnight	4½	1½	1½ hr.
Tested after holding at 40° F. overnight	0	0	10 min.
Tested after holding at 32° F. overnight	2½	1	1½ hr.

for this reason the life of the leucocytes in milk is probably dependent upon the temperature at which the milk is held, and the nearer the temperature is to body temperature the shorter the life of the leucocyte, and the lower the temperature (providing that it is now low enough actually to damage the cells physically) the longer the life of the leucocytes.

If this is so, milk with a high leucocyte content should not reduce resazurin so rapidly after it has been held at temperatures approaching body temperature, and milk held at lower temperatures should reduce at approximately the same rate before and after holding. This has been found to be so, except in the case of milk held overnight at 40° F., when the reducing activity of the cells appear to be definitely increased for no apparent reason.

In all cases a bacterial plate count test was carried out to ensure that any increase in reducing activity was not due to the growth of cryophilic bacteria in the milk during the holding period.

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The Soil as a Source of Infection of Dry Rot of Potato

IT has long been suspected that the fungus causing dry rot (*Fusarium caeruleum*) is present in field soils[1,2] and in soil adhering to seed tubers[3], but, so far as I am aware, no direct proof, based on experimental evidence, has been published.

In 1942, inoculation of susceptible tubers with unsterilized field soil, previously sprayed with a spore suspension of *F. caeruleum*, caused dry rot to develop. The result suggested that this direct inoculation method with soil might be useful to prove the presence of the fungus in field soils and in soil adhering to seed potatoes.

In 1942 and 1943, soil samples were obtained from several farms in Cheshire ; each sample consisted of soil scraped from thirty tubers with a sterile knife and collected in a new envelope in the field at digging time, before the tubers came into contact with any possible source of infection such as hampers, seed boxes or sacks. All the samples proved to be infected. Suitable controls remained sound. Similar results were obtained with many samples collected at random from healthy seed tubers stored in seed boxes in lofts during the winter 1942–43.

During the present winter soil samples were collected at wholesale merchants' stores from seed potatoes imported from Scotland and from Northern Ireland. From each consignment ten of the top-