it possesses the only living species of *Mastotermes*, the most primitive living termite and one which is responsible for a large amount of economic loss. Mr. Hill gives the means of identifying all the termites, which he recognizes from Australia, New Guinea and the islands south of the Equator, between 140° E and 170° W, a region which is of very great interest to us in this country at the present time. Although the book is primarily a description of species in technical terms, it should not be forgotten that this is ultimately one of the major bases of our knowledge of any group of animals, and where an entire fauna is redescribed so far as possible by one man on a uniform basis, the value of all separate descriptions is enhanced.

The book, however, is more than that since it includes a summary of whatever is known, all too frequently very little, of the biology of the various forms. It may, therefore, be considered as a foundation upon which much future detailed work will be laid. It is not surprising, with such a rich fauna hitherto described in fragments by many investigators, that detailed experimental work on the physiology of the species has not been widely undertaken before. We may hope to see a great increase in such work in the near future, now that the solution of the always troublesome problems of identification of material has been rendered as simple as may be.

In spite of the fact that the book is lithoprinted, its appearance is very pleasing and the illustrations are excellently drawn and well reproduced. I noticed only a few typographical errors, of no particular consequence.

CHARLES H. BLAKE

SPECIAL ARTICLES

ON THE MECHANISM OF INSULIN ACTION: OBSERVATIONS WITH RADIOACTIVE PHOSPHORUS

IN spite of the large amount of study that the subject has received, the mechanisms by which insulin brings about increased deposition of glycogen and oxidation of glucose in striated muscle are little better understood than when these effects were first observed. The participation of the phosphorus compounds in carbohydrate metabolism offers the hope that tracer studies with the radioactive isotope of this element (P^{32}) would cast some light on the matter. In this preliminary report are described some effects of insulin on phosphate metabolism in resting muscle, as determined by this technique; possible correlations of the findings on the intact animal with those obtained on cell-free extracts are discussed.

The injection of insulin into cats under pentobarbital anesthesia was found to result in an increased turnover rate of phosphocreatine P and of the two labile phosphate groups of adenosine triphosphate during glucose absorption, and no change in the rate of turnover of glucose-6-phosphate beyond that resulting from the administration of glucose alone.

Three groups of experiments were performed on 24-hour fasted animals. In each case, the muscles were analyzed 4 hours after the subcutaneous action of Na_2HPO_4 containing P^{32}. In the control group (8 animals) only the phosphate was given. A second group (4 animals) was given 50 cem per kgm of 5 per cent. glucose solution intraperitoneally half an hour after the glucose. A third group (4 animals) was given glucose in the same way as the second group, followed half an hour later by a subcutaneous injection of 5 units of insulin per kgm.

The phosphorus compounds were separated from trichloroacetic acid filtrates and measurements of radioactivity made by methods similar to those previously described.[1] The data, given in Table 1, are expressed in terms of counts per minute per mgm of P, calculated to the basis of 10^6 counts per minute injected, per kgm body weight.

TABLE 1

TURNOVER RATES OF PHOSPHORUS COMPOUNDS IN RESTING MUSCLES OF CATS, AS INFLUENCED BY GLUCOSE AND INSULIN*

	Inorganic phosphate	Phospho-creatine	Adenosine triphosphate	Glucose-6-phosphate	Plasma inorganic phosphate
Group I Phosphate only	364 (224–470)	125 (64–182)	82 (50–117)	208 (114–298)	9460 (5300–14,400)
Group II Phosphate and Glucose	308 (212–460)	115 (67–210)	77 (64–106)	522 (402–665)	9475 (7150–14,200)
Group III Phosphate, glucose and insulin	325 (225–415)	317 (205–425)	189 (133–248)	482 (374–620)	6200 (4950–8800)

* All values are expressed in terms of counts per minute per mgm P, calculated to the basis of 10^6 counts per minute injected, per kgm body weight. Figures given are averages, with the range in parentheses.

It is apparent that the injection of insulin during glucose absorption results in an acceleration of the turnover rates of phosphocreatine and adenosine triphosphate, and that this effect is not seen in the absence of an external supply of insulin. It is quite

[1] Jacob Sacks and Charles H. Altshuler, *Am. Jour. Physiol.*, 137: 750, 1942.

possible that this increased turnover is associated with the increased oxidation of glucose that insulin produces in resting muscle. Such coupling of phosphorylation with oxidative reactions has been reported many times in cell-free extracts. In most of such studies the emphasis has been placed on adenosine triphosphate, but the present findings indicate that phosphocreatine is also involved. Obviously it is inadvisable to attempt any specific correlations between the observed changes in the intact animal and the vast number of reactions found in cell-free extracts. However, these results point strongly in the direction of such an association of phosphorylation with glucose oxidation in the resting metabolism of muscle, and indicate that insulin, by accelerating glucose oxidation, also accelerates these phosphorylation reactions.

With regard to glucose-6-phosphate, on the other hand, it is evident that the administration of glucose alone to the 24-hour fasted animal does produce an increased turnover which is not affected by an external supply of insulin. It remains for future study to determine whether the increased turnover evoked by glucose depends on the normal secretion of insulin.

Caution is necessary in evaluating the data on the glucose-6-phosphate in relation to the question of phosphorylation during glucose absorption. The presence of P^{32} in higher concentration in this substance than in the other organic phosphorus compounds shows that some phosphorylation of glucose does take place. However, in relation to the total probable glucose absorption, the amount of phosphorylation observed is rather small. This becomes evident on comparison of the P^{32} levels of glucose-6-phosphate and plasma inorganic phosphate. The average glucose-6-phosphate content of resting muscle does not exceed 10 mgm per cent., calculated as P, and there is no interchange of phosphate groups between this substance and phosphocreatine or adenosine triphosphate.[1] Therefore the transfer of 1 mgm per cent. of P across the cell membrane in the form of glucose-6-phosphate should raise the P^{32} level of this substance to at least 1,000 counts per minute per mgm P when the P^{32} of the plasma P is of the order of 10,000 counts per minute per mgm P. But the highest values obtained are only about half of this amount, indicating the transfer of about 3 mgm per cent. of glucose into the muscle in the form of glucose-6-phosphate. This holds even in the insulin experiments, when it would be anticipated that glycogen deposition and glucose oxidation are taking place at relatively high rates. If the glucose phosphate were dephosphorylated in the formation of glycogen, not only the glucose-6-phosphate but also the inorganic phosphate of the muscle should show higher P^{32} levels in the insulin experiments than in the other two groups. Two possible interpretations of the present data are: (1) that the absorption of glucose by the muscle fiber does not involve the entrance of a phosphate group into the cell, or (2) that glucose-6-phosphate is not involved in the principal mechanism of glucose absorption by resting muscle.

Summary. Insulin causes a marked increase in the turnover rates of phosphocreatine and adenosine triphosphate in resting muscle during glucose absorption, as determined with radioactive phosphorus, but does not cause any increase in turnover of glucose-6-phosphate beyond that produced by glucose itself.

The radioactive phosphorus used was supplied by the Department of Physics of the University of Michigan.

This work was supported by a grant from the John and Mary R. Markle Foundation.

JACOB SACKS

DEPARTMENT OF PHARMACOLOGY,
UNIVERSITY OF MICHIGAN MEDICAL SCHOOL

SULFONAMIDE DEPRESSION OF INORGANIC CATALYTIC ACTION[1]

AN anti-catalytic action has been urged as the fundamental mechanism of the antiseptic action of the sulfonamides, although it would probably be weaker than that of many other antiseptics. The evidence for this has been limited to catalysts in living microorganisms. This theory does not necessarily conflict with the competition-interference theory in which the utilization of para-aminobenzoic acid and other agents essential to bacterial growth is affected by sulfonamides, because metabolic functions are basically mediated by catalytic action. Depression of all catalysts by the sulfonamides would be expected to decrease metabolism and growth of microorganisms. Johnson[2] has reported that the activity of the catalyst luciferase is reduced by sulfonamides and a number of other chemically unrelated agents, which, however, possess in common a depressant pharmacological action, especially narcosis. Certain narcotics, alkaloids, antiseptics and toxic ions are known for their depressant actions on inorganic catalysts, which are wholly foreign to living microorganisms. Thus far, however, no test has been made for similar possible effects of the sulfonamides. If positive, this would help to establish a general anticatalytic action for these chemotherapeutic agents. This report presents positive results, demonstrated by us with three inorganic catalysts, namely, fullers' earth, platinum black and colloidal silver.

Methods: Santesson's[3] procedure for testing cata-

[1] From the Department of Pharmacology and Therapeutics, Stanford University School of Medicine, San Francisco, Calif.

[2] Johnson, SCIENCE, 95: 104, 1942.

[3] Santesson, *Skand. Arch. f. Physiol.*, 42: 129, 1922.