Q_{10} values for the activity of salivary amylase of cold and warm adapted cockroaches

		Q_{10} values 15–25 °C	25–35 °C
Nymph	15 °C-adapted	2.7	1.4
	35 °C-adapted	2.3	1.6
Male	15 °C-adapted	1.5	1.2
	35 °C-adapted	3.0	1.3
Female	15 °C-adapted	1.3	1.2
	35 °C-adapted	2.1	1.3

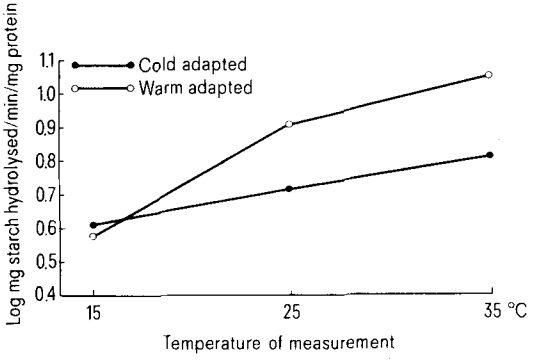

Fig. 3. Effect of temperature on the activity of salivary amylase of cold and warm adapted female cockroach.

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thermal characteristic remains unaltered. Warm adapted males exhibit a decrease (57%) of the Q_{10} value with an increase in temperature of measurement of the enzyme activity. An approximate rotational pattern (Prosser's type III) is observed for this rate process in males resulting in an alteration of the energy of activation ('μ') which signifies a qualitative change in the nature of enzymatic protein followed by a change in some cofactor, or a shift to an alternate pathway.

3. The warm adapted females exhibit an increased amylase activity -55% ($p < 0.01$) at 25 °C and 74% ($p < 0.001$) at 35 °C in comparison with cold adapted insects. In contrast, the situation is reversed when measurements are taken at 15 °C where the value for this rate process is 8% higher ($p < 0.05$) in cold adapted female roaches than in warm adapted ones. However, this minute difference may practically be insignificant. A decrease (38%) in the value of Q_{10} in the lower thermal range due to cold adaptation is observed in female roaches, but no such change is visible in the higher thermal range. A decreased Q_{10} value (38%) with an increase in temperature of measurement is evident in warm adapted females but remains stationary in cold adapted ones. Female roaches demonstrate 'Prosser's type IV-D' pattern of acclimation for the activity of its salivary amylase. This pattern is rarely met with and is non-compensatory in nature.

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Thiamine deficiency and protein secretion by pancreatic slices in vitro

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Summary. Pancreatic slices incubated in glucose medium take up oxygen and glucose and liberate pentose, pyruvate and proteins. Thiamine deficiency decreases oxygen consumption but increases liberation of pentose, pyruvate and proteins by pancreatic slices.

Among the vital organs of the body, the pancreas occupies an important position in view of its exocrine and endocrine functions. These functions involve active processes concerned with carbohydrate metabolism, protein biosynthesis and protein secretion. Pancreas has a high thiamine content[1], which gets depleted to a great extent (73%) at the peak of thiamine deficiency. The transketolase activity of this tissue is also decreased considerably (63%) despite increased tissue nitrogen content following thiamine deficiency[1].

Earlier studies from this laboratory have indicated that thiamine deficiency brings about various biochemical alterations such as a significant elevation in the blood levels of glucose, pyruvate and nonprotein nitrogen, and a marked decrease in the glucose tolerance[2]. Anorexia is one of the prominent symptoms of thiamine deficiency[3,4]. The behaviour of pancreatic slices during incubation in a glucose-saline medium was thought to be able to throw some light on pancreatic secretion of enzymes and other metabolites which could perhaps explain the anorexia in thiamine-deficient state.

Materials and methods. Male albino rats, 2 months of age and made thiamine-deficient[2], were sacrificed along with controls and 3 portions of pancreas, viz. duodenal (head), gastric (body) and splenic (tail) parts were cut out, and

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Oxygen consumption, glucose uptake, pentose release, pyruvate liberation and protein secretion by pancreatic slices of normal and thiamine-deficient rats

Determination done		Duodenal region	Gastric region	Splenic region
Oxygen consumption μmoles/g 2 h	Normal	29.4±3.0* (8)	31.2±3.1 (11)	36.0±2.9 (11)
	Thiamine-deficient	19.5±3.1 (8) p<0.05	17.5±1.9 (11) p<0.01	17.0±3.0 (11) p<0.001
Glucose uptake μmoles/g 2 h	Normal	18.8±2.8 (10)	17.0±1.1 (11)	13.3±1.3 (11)
	Thiamine-deficient	22.5±1.5 (10) p = n.s.	20.7±2.0 (12) p = n.s.	16.0±1.9 (12) p = n.s.
Pentose release μmoles/g 2 h	Normal	12.5±1.0 (9)	11.6±1.0 (10)	10.3±1.0 (10)
	Thiamine-deficient	20.1±1.7 (9) p<0.01	19.2±0.7 (10) p = 0.05	15.5±0.6 (10) p = 0.01
Pyruvate liberation μmoles/g 2 h	Normal	88±24 (5)	79±10 (5)	87±10 (5)
	Thiamine-deficient	449±74 (8) p<0.01	222±51 (8) p = 0.05	158±16 (8) p = 0.01
Protein secretion μmoles tyrosine/g 2 h	Normal	24.9±1.7 (4)	24.9±2.6 (5)	24.3±1.2 (5)
	Thiamine-deficient	50.2±9.5 (5) p = 0.05	32.7±1.4 (5) p<0.05	29.0±1.5 (5) p<0.05

*Standard error. Figures in parentheses indicate the number of rats.

80–100 mg of slices each incubated in 3.3 ml of phosphate buffer[5] pH 7.4 containing glucose (5 mM) in an oxygen atmosphere and at 37 °C in Warburg flasks which were shaken at 80 oscillations per min for 2 h. The centre well contained 0.2 ml of 20% KOH for absorption of CO_2. The oxygen consumption was measured for a period of 2 h. Glucose was estimated in the medium by the method of Hugget and Nixon[6] using glucose oxidase-peroxidase reagent. The pentose[7], pyruvate[8] and protein[9] liberated into the medium were also determined after 2 h period of incubation. In the preparation and incubation of slices, all details described by Hokin and Hokin[10–12] were adopted. Blood was collected in oxalated tubes from rats at the time of sacrifice and the pentoses determined by the orcinol method[7].

Results. The oxygen consumption of pancreatic slices was greatest in the splenic and least in the duodenal region (table). In thiamine deficiency, oxygen consumption was decreased significantly in all regions of pancreas, the greatest (52.8%) decrease being in the splenic region. Pancreatic slices took up glucose efficiently from the incubation medium, those from the splenic region showing the least uptake. No change in glucose uptake was observed with pancreatic slices from thiamine-deficient rats. Pancreatic slices from normal rats released pentose into the medium during incubation which was significantly increased ($p < 0.001$) in the duodenal and gastric region, but not in the splenic region in the case of slices from thiamine-deficient rats. The slices also liberated pyruvate into the medium during incubation, which was in the same amount in all regions of pancreas. The slices from thiamine-deficient rats liberated much more pyruvate than those of normal controls, this being 5fold in the duodenal region, 3fold in the gastric region and nearly 2fold in the splenic region ($p < 0.001$, $p < 0.05$ and $p = 0.01$) respectively. During incubation, pancreatic slices efficiently secreted proteins into the incubation medium. In normal rats, all the 3 regions secreted nearly the same amount of proteins, while in thiamine-deficient rats, the protein secreted was in increased amounts, being greatest in the duodenal region and least in the splenic region.

Discussion. A decrease in oxygen consumption by slices from thiamine-deficient rats may indicate decreased breakdown of glucose. If the pentose release is considered in terms of percentage of glucose uptake, it is found to be 66, 68 and 78 in the duodenal, gastric and splenic regions respectively in normal pancreas, which is raised to 89, 91 and 96 in the corresponding regions of pancreas of thiamine-deficient rats. As pentoses are released to an equal extent by all regions of normal pancreas, it appears that pentose phosphate pathway may be operating uniformly throughout this tissue. Pentose production appears to be a very important process in normal pancreas, a glandular tissue which has an increased need for RNA required for the biosynthesis of secretory proteins. It is not clear why pentoses which are intermediates in the HMP shunt pathway are released into the medium rather than being re-utilized for hexose formation by normal pancreas. The low transketolase activity[1] in pancreas, as compared with brain, liver and kidney, may be a possible reason for this decreased utilization of pentose. The increase in $\frac{\text{pentose release}}{\text{glucose uptake}}$ ratio in the case of thiamine-deficient rats may be attributed to the further lowering of the transketolase activity in that condition[1], as observed earlier.

In many tissues, pyruvate is converted to acetyl CoA which is oxidized to CO_2 by citric acid cycle, or is diverted to other pathways. Busch and Baltrush[13] have observed that pyruvate-^{14}C and acetate-^{14}C are rapidly incorporated into pools equilibrating with citric acid intermediates, in vivo in pancreatic tissue. But the fact that pyruvate is liberated into the medium by pancreatic slices during incubation may indicate that pancreas is one of the tissues that normally contribute pyruvate to the blood. The pancreatic slices of thiamine-deficient rats liberate more pyruvate to the medium than those of normals, indicating a decrease in the pyruvate utilization resulting from thiamine-lack, although glucose uptake is unaffected. Perhaps the glucose metabolism goes on well upto the pyruvate and pentose stages in pancreas and the thiamine deficiency impairs only the further metabolism of these intermediates.

It is interesting to find that protein secretion by pancreatic slices is enhanced in thiamine deficiency. Thus the anorexia of thiamine deficient rats cannot be due to a defect in the secretion of digestive enzymes, but it may be due to other reasons. Further study is needed in this direction, to find out whether it is due to defective metabolism of glucose, resulting from thiamine lack.

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