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Inhibitory effect of galactose on hydrogen transfer in the hemolymph of *Vespa orientalis*

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Summary. The generation of pyruvate and lactate in *Vespa orientalis* hemolymph is prevented by the presence of galactose. The inhibitory effect is possibly produced by a competition for NAD and occurs in concentrations of 0.05 M.

Galactosemia is a well-known disease state brought about by an inborn metabolic error. In man and experimental animals such excess galactose may produce a variety of symptoms ranging from mild to fatal. Its clinical effect and the putative biochemical defects are discussed extensively by Isselbacher[1]. The disease is presumably due to an absence of the enzyme galactose-1-phosphate uridyl transferase, and results in an accumulation of galactose and galactose-1-phosphate, neither of which is toxic in the normal state. In galactosemia, unlike in other enzymatic disorders involving dysfunction of a single enzyme, a large number of other enzymes are inhibited[2].

In a previous investigation of monosaccharidase activity in the hemolymph of *Vespa orientalis*[3] we found that while glucose and fructose are freely utilized via both the aerobic and anaerobic pathways, galactose is not, and its presence interferes with the oxidation of these 2 monosaccharides, as measured in the enzymatic assay[4]. It was further found that hornet larvae die when fed on galactose[5].

Materials and methods. Hemolymph was obtained from the 'dark pupae' (i.e., pupae before eclosion) of *Vespa orientalis.* The reaction was carried out at pH 7.6 using 0.01 M phosphate buffer. Starting values were established prior to the incubation, and any changes in both the lactic and pyruvic acid levels were determined at 60 min post incubation, using Varley's method[4] for the enzymatic assay.

Final volume of the reaction mixture was 5 ml, consisting of 2.5 ml buffer, 2.0 ml of galactose solution prepared in 6 increasing concentrations from 0 to 0.1 mM/ml, and 0.5 ml hemolymph. Lactate and pyruvate levels in each sample prior to incubation were taken as zero and the monosaccharidase activity, measured as described in our previous work[3], was expressed in terms of μM of lactate and pyruvate/ml present after 60 min incubation at 37 °C. The inhibition effect of galactose on lactate and pyruvate production could thus be assessed.

Results. A total inhibition of lactate production was observed in galactose concentrations of 0.05 mM/ml and above, and a partial inhibition of pyruvic acid generation – at galactose concentrations of 0.5 mM/ml and above. The results are summarized in the table.

Discussion. In a previous study we demonstrated monosaccharidase activity in *Vespa orientalis*[3], encountering oxidation both by the aerobic and anaerobic pathways, but not in the presence of galactose. Apparently, galactose blocks the anaerobic (lactate) pathway of monosaccharide utilization even in low concentration whereas in higher concentration it also blocks the aerobic (pyruvate) pathway. These findings suggest several possibilities. First, that galactose not only cannot be utilized by *Vespa orientalis* larvae and pupae (and probably by various other insects) but may prove to be deadly poisonous for them. As such and because it is ordinarily absolutely harmless to all other organisms galactose should comprise an effective pesticide-insecticide, superseding ecologically dangerous DDT and the potentially lethal organo-phosphates. Its practicability, however, has yet to be proven by actual field trials.

Secondly, the results of our present study suggest also that, among the congenital metabolic defects (enzyme deficiencies), galactosemia causes the widest biological damage because it interferes with most of the enzymatic reactions dependent on the coenzyme NAD-NADH, that is, with most of the hydrogen transfer reactions. Whether this is due to competition or actual inhibition has yet to be established.

Inhibition of monosaccharidase activity in the hemolymph of *Vespa orientalis* larvae by the presence of galactose

Monosaccharides added	Galactose concentration (mM/ml) 0.00		0.01		0.05		0.20		0.50		1.00	
	L	P	L	P	L	P	L	P	L	P	L	P
Fructose 1 mM/ml	450	1900	110	1650	0	200	0	144	0	±	0	0
Glucose 1 mM/ml	140	950	20	200	0	25	0	±	0	0	0	0

L, μM lactate/ml, produced at 37 °C in 60 min; P, μM pyruvate/ml, produced at 37 °C in 60 min.

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