Mode of action of low atmospheric pressures on *Ephestia cautella* (Wlk.) pupae[1]

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Summary. The influence of low atmospheric pressures and low oxygen concentrations on 0-24-h-old *Ephestia cautella* (Wlk.) pupae at 26 °C was determined. Effects on respiration, insect mortality and loss in weight obtained, were due to low oxygen tension only, at both normal and low atmospheric pressure.

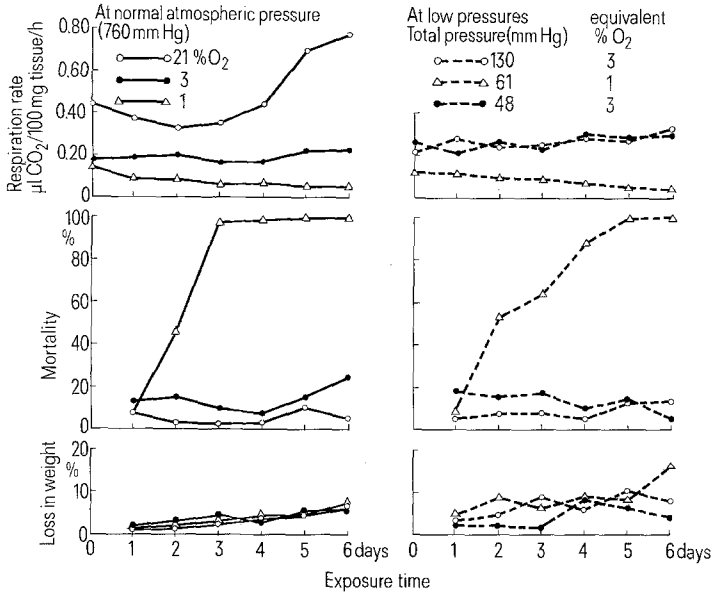
Previous work has shown the effect of low oxygen tensions[2] and of low atmospheric pressures[3] on stored product insects. It has been reported also that lowering the relative humidity in the exposure chambers enhances the effect of low atmospheric pressures on the mortality of insects[4]. In these cases, the effects of low relative humidities resulted in desiccation of insects, as expressed in loss of weight reaching the critical level to which insect mortality was attributed. At very low oxygen tensions, mortality occurred, even when desiccation was prevented by high relative humidity[5]. However, the effects of low pressures alone on insects have not yet been clearly demonstrated. Galun and Fraenkel[6], working with *Aedes aegypti* adults and housefly pupae, concluded that 'mortality of mosquitoes at very low pressures stems from at least 3 factors acting independently: a) dehydration; b) lack of oxygen; and c) low pressure as such.' It was deduced also that 'very low pressures have an adverse effect on respiration per se, independent of that of the concomitant low oxygen tension.' However, results obtained by Dumas et al.[7] do not indicate clearly whether the reduced pressures themselves had the effect of lowering respiration of *Tenebriodes mauritanicus* (L.) larvae.

In this work, the effect of low pressures and of low oxygen tensions was tested at a high relative humidity (93-99%) in order to eliminate the possible effect of desiccation of test insects due to exposure to lower relative humidites.

Ephestia cautella (Wlk.) pupae, 0-24-h-old, were taken from stock cultures reared at a constant temperature (26±1 °C) and relative humidity (70±5%), and on a mixture of wheat feed with 12% glycerine by weight[8]; the pupae were exposed to the treatments listed in the table.

The apparatus for maintenance of constant low pressures and gas concentrations, as described elsewhere[9], was connected to the test chamber. The entire system was kept in a constant temperature (26±1 °C) room.

Loss in weight and mortality tests, replicated 8 times, were



Response of *E. cautella* pupae exposed to different oxygen tensions at different atmospheric pressures.

Combinations of partial pressures of atmospheric gases and their equivalent concentrations at normal atmospheric pressures to which *E. cautella* pupae were exposed

Total pressure (mm Hg)	Partial pressure of each gas (mm Hg)			Concentration of gases (%) equivalent to the partial pressures at normal atmospheric pressure (760 mm Hg)		
	O_2	N_2	H_2O	$\%O_2$	$\%N_2$	Relative humidity
760	153.9	574.5	23.4	20.9	78	93
760	24.1	712.5	23.4	3.3	96.7	93
760	9.6	727.0	23.4	1.3	98.7	93
130	22.1	83.0	24.9	3.0	11.3	99
61	7.5	28.6	24.9	1.0	3.9	99
48	22.6	0.5	24.9	3.1	0.1	99

carried out on groups of 10 pupae confined in copper mesh exposed to the different treatments in 100-ml capacity exposure chambers. After treatment, the pupae were kept in the culture room until adult emergence was observed. Insects were considered dead when adults failed to emerge. Loss in weight was determined by weighing each group of pupae before and after treatments.

Respiration rates, as expressed by carbon dioxide output (4 replicates), were determined on groups of 50 insects weighed and exposed to treatments in 50-ml test chambers. Daily carbon dioxide output was determined by stopping the gas flow for 2 h and measuring the gas composition.

The results on respiration (figure) show that, contrary to the typical U-shaped curve obtained at normal atmospheric conditions (21% oxygen)[10], respiration curves obtained at 3% oxygen or corresponding tension at low pressures (130 and 48 mm Hg) demonstrate a suppressed respiration rate. The 1% oxygen curves, on the other hand, showed the tendency of decreasing carbon dioxide output which leads to insect death. Results obtained at normal atmospheric pressure and those obtained at the corresponding low oxygen tensions, at the low atmospheric pressures tested, were very similar.

This similarity in the response of the treated insects is also apparent in the curves of weight loss and mortality. Accordingly, 100% mortality was obtained at 1% oxygen (normal pressure) as well as in the corresponding oxygen tension at 61 mm Hg. However, at 48 and 130 mm Hg and 3% oxygen, this effect on mortality was not obtained. Weight loss curves, on the other hand, did not indicate a critical upset of water balance in insects. It may be concluded that the effects on mortality and respiration of insects was due to the low oxygen tension only, at both normal and low atmospheric pressure.

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Effects of insulin on plasma fibrinogen levels in rats submitted to tissue injury or ACTH administration

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Summary. Insulin is necessary to produce an increase of plasma fibrinogen in rats submitted to tissue injury or ACTH administration. This increase is more significant when endogenous or exogenous excess of insulin is present, while in uninjured rats the absence or excess of insulin does not modify plasma fibrinogen.

It is known that tissue injury produces an increase in plasma fibrinogen levels in the rat[1,2]. Also during the tissue injury the liberation of ACTH is increased. The administration of ACTH increases the fibrinogen[3] due to an extra-adrenal mechanism[4] and to an increment of hepatic synthesis[5,6]. ACTH also increases the pancreatic synthesis and liberation of insulin[7-10]. As insulin contributes to the improvement of the nitrogeo balance after surgical injury[11], and is also important in regulating the activity of protein metabolizing enzimes[12], it was of interest to study the role of insulin on the increment of fibrinogen following tissue injury or the administration of ACTH.

Material and methods. 205 rats of both sexes, weighing between 140 and 200 g each, were used. The experiments were performed in 48-h fasted rats. Uninjured rats were used as control. Tissue injury was performed by laparotomy[13] wiuh careful manipulation of liver and visceras. The modifications of plasma fibrinogen by absence of endogenous insulin were studied in alloxan-diabetic rats. Aqueous alloxan (Sigma) in doses of 150 mg/kg was administered by i.p. injection. 48 h after the injection of alloxan or when the rats had finished the period of fasting, they were submitted to laparotomy or drug injection. Crystaline (Lilly) insulin was administrated in doses of 0.4 IU/100 g s.c., twice daily. Soluble ACTH (Elea), 1 IU twice daily, was given s.c. in 0.9% ClNa solution. Tolbutamide (150 mg/kg daily) was administrated s.c. in saline solution. 96 h after laparatomy or administration of drugs, blood was extracted over a mixture of ammonium oxalate and potassium oxalate in a 2:1 proportion. The methods used to extract the blood and to determine the plasma fibrinogen have been described elsewhere[14]. Glucose levels were determined by o-toluidine meuhod[15-17]. Rats were considered diabetic when plasma glucose was over 300 mg/100 ml. As the rats injected with alloxan presented a significant hemoconcentration (with a mean hematocrit of 53.4%), and control rats had a mean hematocrit of 44%, all the results were corrected according to normal values of hematocrit by Boas and Peterman equation[18]. Statistical treatment was made by the t-test. Significant differences were taken as $p < 0.05$.

Results. Solvent of drugs (distilled water or 0.9% ClNa solution) does not modify fibrinogen, compared with intact rats.

The results obtained in groups of rats submitted to tissue injury (laparotomy) and rats injected with alloxan or insulin are presented in table 1. Laparotomy produces an increase of fibrinogen compared with control. This increase was not observed in laparotomized rats injected with alloxan (Lap+A), in which fibrinogen levels are similar to those observed in controls. On the other hand, rats injected with alloxan or with insulin did not present variation of fibrinogen in comparison with control rats or rats injected only with solvent. On the contrary, the laparotomized rats injected with alloxan and insulin (Lap+A+I) increase the fibrinogen to levels similar to those observed in laparotomized uninjected rats. In laparotomized rats injected with