(haemolymph) by diffusion through follicular cell layer in collaboration with nucleolar extrusions[8-10]. In *C. rajadhari* nucleolar extrusion granules, which passed into oocyte cortex were believed to take part in yolk formation (Endogenous yolk)[11]. The oval, non-germinative, accessory, follicular cells were also helpful in the uptake of extraoocytic yolk protein because they were always found attatched themselves around the early vitellogenic oocytes (figure 3)[12]. The late vitellogenic oocytes of *C. rajadhari* also showed the presence of numerous yolk droplets in the oocortex as in the shrimp, *Chirocephalus bundyi*[8], prawns *Palaemon paucidens*[4] and *Macrobrachium lanchesteri*[5] (figure 5). As the oocytes grow in size, the yolk droplets became strongly eosinophilic and increased in size and number (figure 6). In large oocytes, oocortex showed a well defined thick membrane resembling an egg envelope. After maturation, the ova were ovulated and oviposited in the brood pouch of the female. In *C. rajadhari*, the process of resorbtion (oosorbtion) may be simultaneous with oocyte growth. The crowding and competition among the oocytes rendered some of them in such resorbing states. Oocyte resorbtion was also a normal phenomenon in the females of *P. paucidens*[4] and *M. lanchesteri*[5]. The presence of more than one size range of oocytes in the ovaries of *C. rajadhari* provided evidence for the continuous breeding pattern of these prawns.

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EFFECTS OF METHYL PARATHION ON THE RATE OF OXYGEN CONSUMPTION OF TADPOLES OF FROG, *RANA CYANOPHLICTIS*

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PESTICIDES, the economically useful poisons are known to spread through all segments of the environment, causing untold hazards to non-target organisms. Pesticides are generally responsible for a number of biochemical and physiological disturbances[1,2]. Pesticides have been shown to cause a sharp and substantial increase in the rate of oxygen consumption in insects[3] and fishes[4].

However the paucity of data on the toxic effects of methyl parathion on the rate of oxygen consumption during brain development, prompted us to carry out the present investigation. Oxygen consumption of the developing tadpoles of frog, *Rana cyanophlictis* has been studied on exposure to sublethal concentration of methyl parathion. Brain glucose levels were also determined to study the changes in the energy demands of the methyl parathion exposed animals, since it is known that the nervous tissue is the first to respond to any type of stress.

Approximately three-week-old tadpoles of frog, *Rana cyanophlictis* in the weight range of 0.5–2 g were obtained from local ponds and acclimated to laboratory conditions for a week, prior to experimentation. They were fed on Hydrilla. Sublethal concentrations of methyl parathion (0, 0-dimethyl-O-nitrophenyl-thiophosphate, EC 50 %$_0$ solution, Bayer Ltd., India), in tap water were prepared and the animals were exposed to this for 24 hr. The lethal concentration was experimentally determined. The LD_{50} value at 24 hr was estimated (LD_{50} = 8 ppm)[5]. The test animals were then kept in three different concentrations below LD_{50} (2.5 ppm (sublethal), 5 ppm and 7.5 ppm) for 24 hr. The rate of oxygen consumption was studied by Winkler's 'iodemetry' method[6]. The brain glucose levels were determined by the colorimetric procedure of Nelson-Somogyi[7].

The results presented in table 1 and figures 1 and 2 show an increase in the rate of oxygen uptake on 24 hr exposure to methyl parathion in the frog tadpoles. This indicates that the stress caused due to methyl parathion exposure requires greater energy, and hence an increase in the metabolic rate, which in turn indicates an enhanced oxidative process within the body on methyl parathion exposure.

It is plausible that the enhanced respiratory rate of tadpoles on methyl parathion exposure may be the direct consequence of the hyperactivity of the brain due to the action of the pesticide, since nervous system is known to be the first to respond to any stress condition. Supporting this, the brain glucose showed a significant decrease on methyl parathion exposure (see table 1) indicating that glucose is utilized at an enhanced rate to meet the augmented energy demand. Since methyl parathion is known to affect the cholinesterase system causing disturbance in neurotransmission[8] in poikilothermic vertebrates, it is presumed that the alterations occurring in the neuronal activity on methyl parathion exposure cause augmentation in the general metabolic rate.

Table 1 *Changes occurring in the levels of brain glucose and oxygen consumption in control and methyl parathion exposed (5 ppm) tadpoles of R Cyanophlictis*

	Control	Methyl parathion exposed	% change
Glucose (mg/g ww)	4 76 +0.30	2.4 +0 91	−49 58 ($P > 0$ 05)
Unit metabolism (μl of O_2 consumed g/hr)	203 81 +8 1	305 24 +65 42	+49.77 ($P > 0.01$)

All the values are Mean + S.D of 6 observations

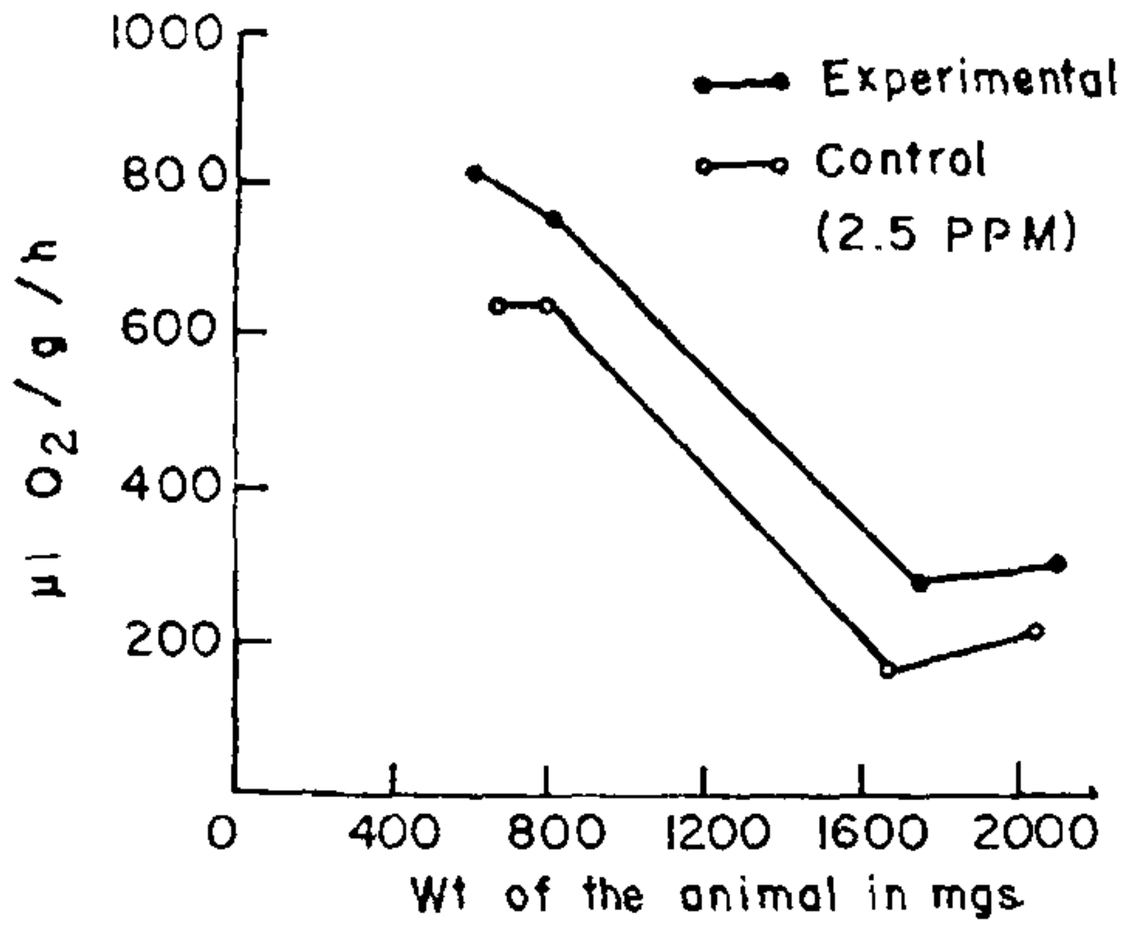

Figure 1. Oxygen consumption of normal (control) and methyl paration exposed (experimental, 2.5 ppm) tadpoles of *R. Cyanophlictis* as a function of size (six tadpoles were used in each body weight group).

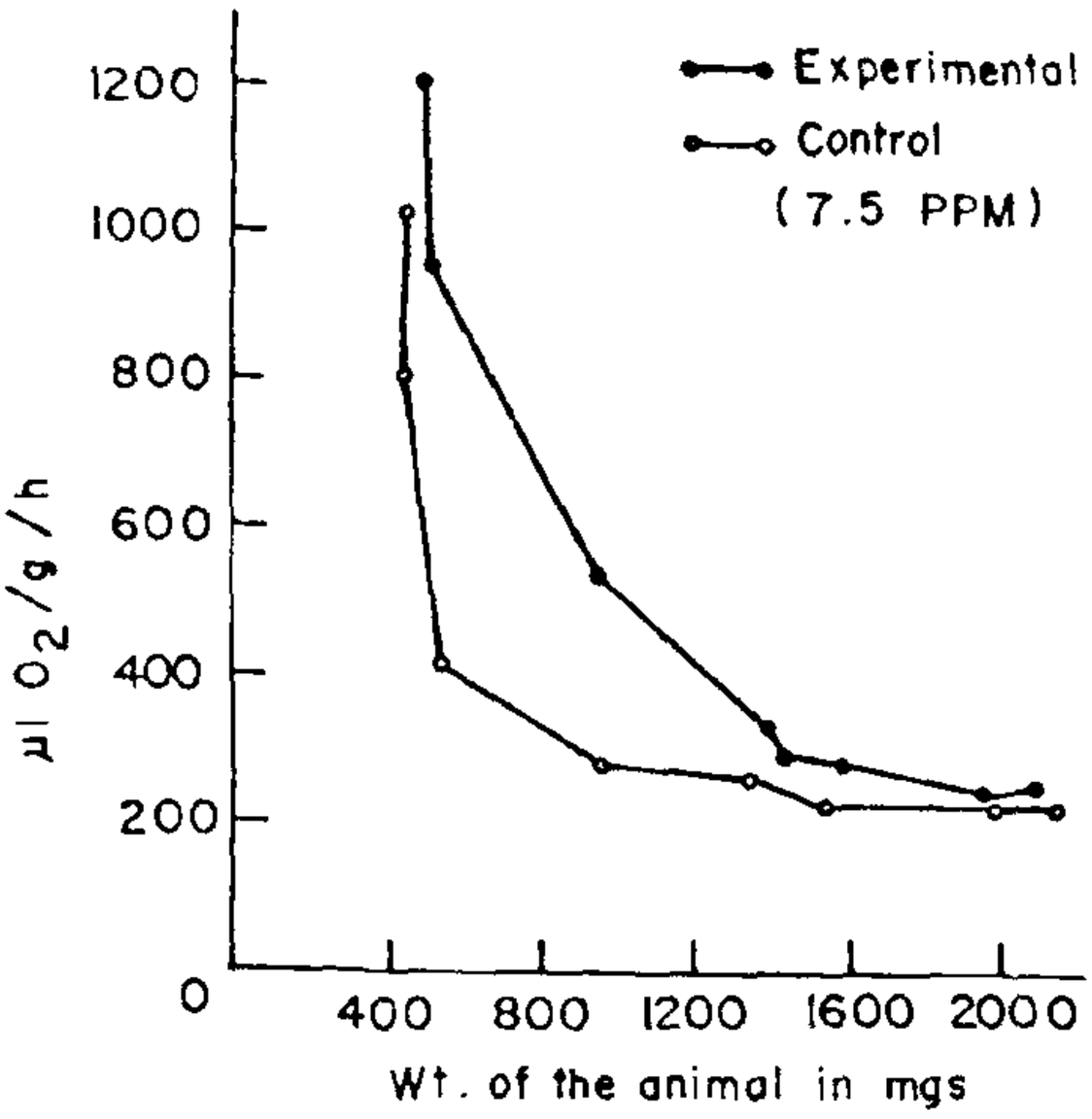

Figure 2. Oxygen consumption of normal (control) and methyl parathion exposed (experimental, 7.5 ppm) tadpoles of *R. Cyanophlictis* as a function of size (six tadpoles were used in each body weight group).

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EFFECT OF CAFFEINE ON PUFFING PATTERN IN THE POLYTENE CHROMOSOMES OF SALIVARY GLANDS OF *DROSOPHILA MELANOGASTER*

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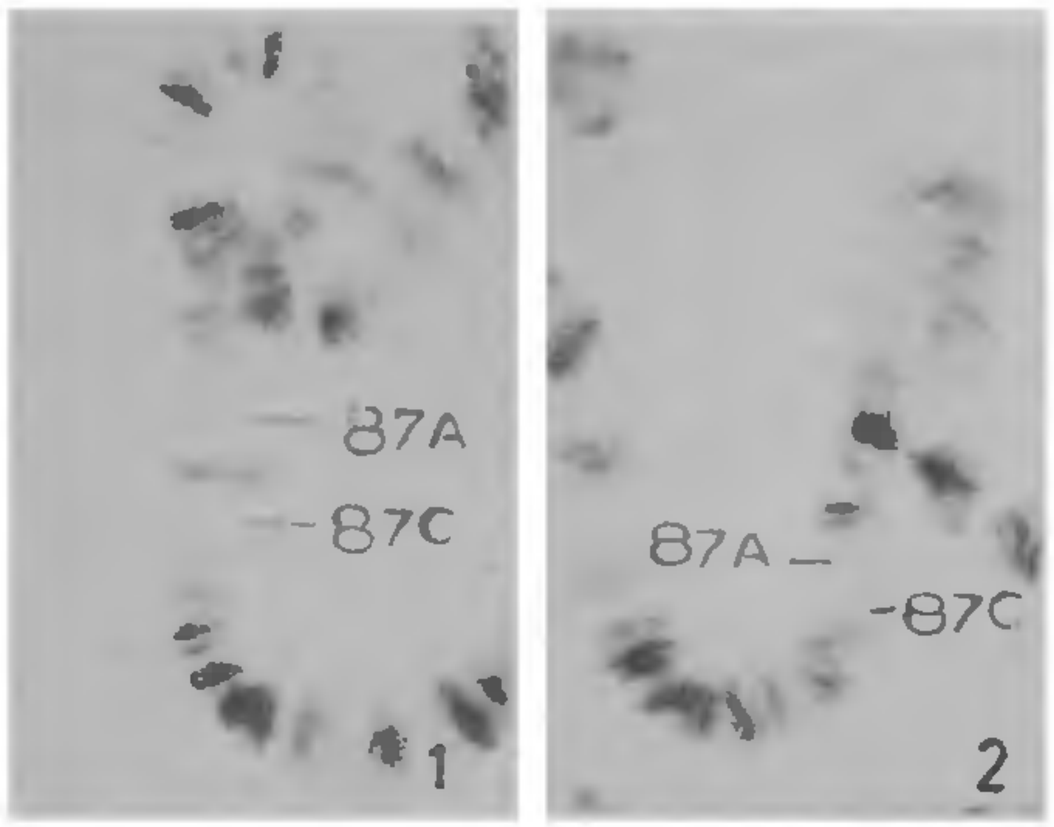
CAFFEINE (1,3,7-trimethylxanthine), a plant product and a member of methylxanthine group is widely used in general beverages and is being consumed by a large section of the population. It has also therapeutic use as central nervous system stimulant. Caffeine has some antimitotic activity and causes chromosomal abnormalities in both plant and animal cells. A wide range of other cellular activities are also affected by caffeine[1]. But no information is available on the effect of caffeine on puffing activity. Larval salivary glands of *Drosophila* when treated with heat shock or uncouplers of oxidative, phosphorylation result in the induction of a certain group of puffs[2–4]. These puffs are also induced by antibiotics, valinomycin and dinactin[5]. It was suggested that the cellular level of ATP plays an important role in the induction of heat shock puffs[4]. Since the group of methylxanthines, to which caffeine belongs, elevates the cyclic AMP level in the tissues[6], its effect on the puffing activity is studied.

Salivary glands of late third instar larvae of *D. melanogaster* Oregan K inbred stock reared at 24° C on a corn meal food enriched with extra yeast, were used in this experiment. The sister glands were dissected out and incubated in Ringer solution[4]. One of the glands was treated with caffeine, freshly dissolved in Ringer solution, while the other gland was kept in nomal Ringer solution as control. After the incubation time (40–90 min) the glands were fixed in 1:3 mixture of acetic acid and methanol, stained with 2% aceto-orcein for 15 min, squashed in 50% acetic acid and sealed with DPX. Puffs were scored according to the chromosomal maps of Bridges[7]. Six sets of glands were used in this experiment.

Incubation of the salivary glands in Ringer solution containing caffeine (10^{-2}M) at 24 C for 1 hr induces the heat shock puffs. The intensity of puffing was greater in 87A and 87C while it was relatively small in 63C, 67B, 93D and 95D puffs (figure 1). Other heat shock puffs were ineffective. The puffs are induced within 30–40 min after incubation with caffeine and they attain their maximum size after 60 min. When incubated for 90 min there was no change in the size of the puffs. None of the heat shock puffs is stimulated in the control glands (figure 2).



Figures 1, 2. **1.** Puffs 87A and 87C induced in salivary gland chromosome (3R) of *D. melanogaster* by *in vitro* treatment with caffeine (10^{-2}M, for 60 min), **2.** Control.

Caffeine, a known inhibitor of certain forms of cyclic nucleotide phosphodiesterases (the enzymes catalysing the conversion of cyclic AMP to 5′-AMP) elevate the concentration of cyclic AMP[6]. In the present experiment it is assumed that caffeine elevates the cyclic AMP level in the salivary gland cells, which activate a specific group (heat shock) of puffs.

Oligomycin, a metabolic inhibitor, lowers the ATP level and did not induce any heat shock puffs. It was therefore suggested that cyclic AMP level elevation is involved in the induction of this special group of puffs[8]. Behnel[9,10] reported that chloramphenicol induced the heat shock puffs in *D. melanogaster* due probably to the suppression of cellular respiration and inhibition of protein synthesis. It is therefore likely that caffeine which increases the cyclic AMP level, also inhibits the respiratory chain reactions. The cumulative effect of both these processes seems to be involved in activating a specific group of puffs.

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