The eosin test, originally employed by BURGOS and DI PALO[7] to differentiate between dead and living mammalian spermatozoa, was used to determine the survival characteristics of both eupyrene and oligopyrene spermatozoa in vitro. This test is based upon the fact that eosin in aqueous solution stains dead cells only, leaving living cells unstained. The eosin test works well on gastropod spermatozoa and stains the dead spermatozoa – both oligopyrene and eupyrene types.

The testes were teased in Ringer solution (NaCl 0.65 g; KCl 0.042 g; $CaCl_2$ 0.025 g; H_2O 100 ml) and testicular squashes studied at room temperature (30 ± 2 °C) employing phase-contrast microscope. Percentage of living and dead spermatozoa of both categories was determined after every 15 min. These observations were supplemented and confirmed when such squash preparations were made in 0.5% eosin in Ringer solution and percentage of stained (eosinophilic) and unstained (eosinophobic) spermatozoa carefully counted. The data has been recorded in the Table.

Survival characteristics of the two types of spermatozoa vary a good deal. Percentage motility at 0 h in case of eupyrene spermatozoa was only 53.33 ± 2.149%, whereas for oligopyrene spermatozoa it was high, viz. 95.38 ± 0.856%. The percentage of motility for both categories of spermatozoa sharply declined in the first 15 min, whereafter the fall in percentage motility became gradual; whereas all eupyrene spermatozoa died within 60 min, a good percentage of oligopyrene spermatozoa (over 21%) showed motility up till 75 min. However, after 90 min, all oligopyrene spermatozoa were also killed.

All this shows that eupyrene spermatozoa in vitro have a shorter survival span as compared with the oligopyrene ones which survived much longer.

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Neurochemical Correlates of Alloxan Diabetes: Protein and Ribonucleic Acid Levels in the Different Regions of the Rat Brain

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Summary. The levels of protein and ribonucleic acid in the cerebrum, cerebellum, optic lobes and medulla oblongata of normal and alloxan-diabetic rats were measured. In general, the protein content and levels of ribonucleic acid in the broad compartments of the brain of rat decreased during diabetes.

Abnormalities of protein metabolism in various tissues of mammals during diabetes have been extensively studied[2–9]. BUCK et al.[4] demonstrated a decrease in the rate of nuclear protein biosynthesis in rat liver; while hepatic ribosomal protein synthesis in rats has been shown to decrease during experimental diabetes[5]. SAYUK[8] reported a decrease in protein and nucleic acid content in tissues from rats with diabetes. However, no information is available on the regional distribution and changes in the levels of protein and nucleic acids in the brain of rat during alloxan diabetes. The paper presents information on the changes in the distribution of protein and ribonucleic acid in different regions of rat brain during diabetes.

Materials and methods. Immature albino rats (Wistar strain) of both sexes, weighing 60–85 g were used. They were maintained in cages at room temperature (25 ± 2 °C) on a commercial diet (Hindustan Lever Ltd., Bombay, India). Water was available ad libitum.

Diabetes was induced by i.v. injection of alloxan monohydrate, as described earlier[10].

Rats were decapitated and the brain was removed from the ventral side. The adhering blood vessels were removed and different regions of the brain, viz., cerebrum, cerebellum, optic lobes and medulla oblongata, were separated with a sterilized scalpel by keeping the brain in mammalian Ringer[11] at 0 °C. The tissues were weighed in an electric balance with Ringer and immediately used for analysis.

Proteins were estimated by the method of LOWRY et al.[12] The levels of ribonucleic acid were measured following the method described by SCHMIDT-THANNHAUSER and SCHNEIDER[13]. Blood glucose was measured by the method of Folin[14].

Results. The Table summarizes the results obtained. Blood glucose levels showed 108% increase over controls during diabetes. In general, the protein content decreased

Changes in the levels of protein and ribonucleic acid in different regions of the brain of alloxan-diabetic rats

	Cerebrum		Cerebellum		Optic lobes		Medulla	
	Control	Test	Control	Test	Control	Test	Control	Test
Protein[a]	125.34 ± 6.8	99.5 ± 4.1	96.3 ± 4.0	79.5 ± 2.3	84.0 ± 4.0	68.2 ± 1.8	87.6 ± 5.2	66.2 ± 3.0
	– 20.65[c] $p > 0.01$		– 17.5[c] $p > 0.01$		– 18.8[c] $p > 0.01$		– 24.5[c] $p > 0.01$	
RNA[b]	6.7 ± 0.9	4.7 ± 0.8	5.9 ± 0.6	4.2 ± 0.3	6.3 ± 0.4	4.6 ± 0.7	7.3 ± 0.2	5.04 ± 0.6
	– 30.1[c] $p > 0.05$		– 29.4[c] $p > 0.01$		– 27.7[c] $p > 0.01$		– 31.3[c] $p > 0.01$	

[a] Mean ± SD of 5 observations expressed as mg protein per g wet tissue.
[b] Mean ± SD of 5 observations expressed as mg RNA/g wet tissue.
[c] Percentage change; sign (–) indicates a decrease in the levels of protein and RNA, over controls.

in the cerebrum, cerebellum, optic lobes and medulla oblongata on in vivo administration of alloxan (Table). Likewise, the levels of ribonucleic acid in different regions of rat brain studied also decreased considerably on alloxanization (Table). The protein content was higher in the cerebral region compared with other regions of the brain in both normal and diabetic rats. However, decrease in the protein content was highest in the brain stem of diabetic rats (Table). On the other hand, the level of ribonucleic acid was higher in the medulla compared to the other regions of the brain. Paralleling the change in protein content, medulla showed marked response for the changes in the levels of ribonucleic acid during diabetes (Table). This differential response of the different regions of the brain is in relation to the differential functional status of the broad compartements of the brain.

Discussion. The decrease in the levels of protein and ribonucleic acid in different regions of the brain of alloxan-diabetic rats appears to be the direct effect of insulin deficiency caused by alloxanization. It has also been shown earlier that insulin deficiency during diabetes inhibits protein synthesis and accelerates the catabolism of amino acids[15]. It has been suggested that the deceleration in the incorporation of amino acids into proteins is one of the major reasons for the decreased protein synthesis observed during diabetes[15]. Supporting this, the free amino acid content in the brain of diabetic rats has been found to be considerably high (JAYASHREE, unpublished observations). However, PETERSON et al.[7] report an increase in the rate of protein synthesis during diabetes in rats.

The decrease in the levels of ribonucleic acid in different regions of the brain of rat during alloxan-diabetes is in accordance with the decrease in total protein content of the different regions on alloxanization. This, therefore, indicates that the entire protein synthetic machinery is disrupted during diabetes.

[1] Acknowledgment. The authors thank Dr. A. R. KASTURI BAI, Head of the Department of Zoology, Bangalore University, for providing facilities during the study.

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Cardiac Pressoreceptors and Peripheral Resistance

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Summary. The results obtained show that the pressoreceptors, probably ubicated in the left ventricle of the rat, respond to the distention with vasodilatation. The afferent tract of this reflex is in the vagus nerve and the efferent one is in the sympathetic nervous system. The probable function of this reflex is discussed.

Previously we have described the effect of the cardiac pressoreceptors on the regulation of the cardiac rate in the rat[1]. To produce the activation of the pressoreceptors, the ascending aorta was occluded and this produces cardiac distention. We have also observed that it is necessary to excise the aortic and carotid pressoreceptor nerves to obtain the reflex.

Effects of the ascending aortic occlusion on the aortic perfusion pressure

	Control pressure (mm Hg)	δ (mm Hg)
Control rats ($n = 11$)	73±10	+0.7±0.4
Rats without aortic and carotid nerves ($n = 14$)	123±12	− 8.2±2.4[a]
Atropine treated rats ($n = 10$)	83±10	− 5.3±1.4[b]
Vagotomized rats ($n = 9$)	127±13	0 ±0.2
Phentolamine treated rats ($n = 14$)	48± 6	+0.1±0.1

n, number of experiments. [a] $p < 0.005$. [b] $p < 0.001$.

The purpose of this work was to study the effect of the cardiac pressoreceptors on the peripheral resistance and to describe the different pathway of the reflex.

Methods. Wistar rats weighing about 200 g were used. The animals were anesthetized with 1 g/kg urethane and kept with controlled breathing by means of a Harvard breathing pump (Model 680). The abdominal aorta was exposed by a midline incission and cannulated in a peripheral direction below the renal arteries, and the carotid artery also was cannulated in a central direction. Blood was pumped from the carotid cannula to the hind quarters by a pump with a flow of 0.7 ml/min. Blood obtained from donor animals was used to fill the tubing. Perfusion pressure was recorded from T tube on a poligraph via a Statham P23AA pressure transducer. The modifications in the perfusion pressure were considered representative of changes in the peripheral resistance.

The ascending aorta was identified and occluded about 10 sec and the perfusion pressure was recorded continuously. 5 groups of rats were used: a) control rats; b) rats with carotid and aortic nerves excised; c) vagotomized

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