

( $28.8 \pm 5.2$   $\mu\text{g/l}$  and  $28.6 \pm 8.5$   $\mu\text{g/l}$ ) between the oxytocin and saline-treated pigs respectively. Thus, if PSE meat is the result of a stress-induced catecholamine release, this finding suggests that oxytocin modifies the action of the catecholamines rather than their release.

Malignant hyperthermia is a well-documented reaction which occurs in stress-susceptible pigs following exposure to halothane anaesthesia. It has been proposed that the mechanisms which produce malignant hyperthermia (irreversible lacticacidosis) are similar to those which bring about PSE meat<sup>11</sup>. In 2 pigs which were given oxytocin

during an established hyperthermic reaction (that is, the lacticacidosis was sufficient that the blood pH was less than 7.00) the reaction continued its usual fatal course. However, in 1 pig which was given oxytocin after halothane, but before the blood pH reached the critical level of 7.00, the reaction was halted (personal observations).

The physiological implications of these findings require further investigations of the effects of oxytocin and catecholamines on receptors in skeletal muscle, but may provide a useful tool for the investigation of the PSE condition in pig meat.

- 1 J. Ludvigsen, in: Recent points of view on the condition and meat quality of pigs for slaughter, p. 113. Ed. W. Sybesma, P. G. Van der Wal and P. Walstra. 1969.
- 2 B. A. Cross and G. W. Harris, *J. Endocr.* 8, 148 (1952).
- 3 D. W. Lincoln, A. Hill and J. B. Wakerley, *J. Endocr.* 57, 459 (1973).
- 4 M. Ginsburg and M. W. Smith, *Br. J. Pharmac. Chemother.* 14, 327 (1959).
- 5 L. E. McDonald, in: *Veterinary Pharmacology and Therapeutics*, IVth edn, p. 665. Ed. M. L. Jones, N. H. Booth and L. E. McDonald. Iowa State Univ. Press 1977.
- 6 N. G. Gregory and D. Lister, *Proc. Nutr. Soc.*, in press (1980).
- 7 C. A. Good, L. Kramer and M. Somogyi, *J. biol. Chem.* 100, 485 (1933).
- 8 I. Gutmann and A. W. Wahlefeld, in: *Methods of enzymatic analysis*, 2nd edn, p. 1464. Ed. H. U. Bergmeyer. Academic Press, New York 1974.
- 9 A. A. Taylor and S. J. Dant, *J. Fd. Technol.* 6, 131 (1971).
- 10 R. L. Robinson and D. T. Watts, *Clin. Chem.* 11, 986 (1965).
- 11 G. M. Hall, J. N. Lucke and D. Lister, *Br. J. Anaesth.* 52, 165 (1980).

## Effect of continuous light on the blood and urinary sugar levels in alloxan diabetic rats

N. M. Biswas, B. Paul and S. Banik<sup>1</sup>

*Department of Physiology, University College of Science and Technology, 92, Acharya Prafulla Chandra Road, Calcutta 700009 (India), 19 May 1980*

**Summary.** Continuous light decreases the rise in blood glucose and the excretion of urinary glucose, along with increased urine volume, in alloxan diabetic rats.

It is now well known that adrenocortical hypersecretion<sup>2-4</sup> and hyperglycemia<sup>5</sup> commonly accompany the onset of alloxan diabetes. Exton et al.<sup>6</sup> have noted that adrenalectomy reduces, and glucocorticoid treatment restores, glucose output from liver in alloxan diabetic rats. Since continuous exposure to light inhibits normal secretion of adrenocortical hormones<sup>7</sup> and appears to counteract the adrenocortical hyperactivity in the alloxan diabetic rat<sup>8,9</sup> the present experiment has been undertaken to determine whether constant illumination can prevent hyperglycemia in alloxan diabetes.

**Materials and methods.** 24 adult female rats maintained at 34°C with 10 h illumination daily were used. The rats were fed a diet of 'Hindlever Rat Chow' and water ad libitum. Samples of urine were collected every 24 h in flasks from all the rats with 0.5 ml of a mixture containing 1.2% each of

penicillin and streptomycin, saturated with thymol to avoid bacterial contamination of the urine. Urine volumes were measured at the end of the 24-h period. 18 rats on 48 h fasting were given a single i.m. injection of alloxan (BDH, London) at a dose level of 7 mg/100 g b.wt, and the remaining 6 control rats received a measured amount of saline. After 3 days blood was obtained from all the rats by cardiac puncture under anaesthesia. Blood and urinary glucose content were estimated by the methods of Folin and Wu<sup>10</sup>, and Benedict<sup>11</sup> respectively. 12 alloxanized rats with exhibited a blood sugar level within the range of 450–500 mg/100 ml were divided equally into 2 groups. 6 alloxan treated rats were then exposed to continuous light. Illumination (40 W) was provided by an overhead fluorescent bulb (cool white). 6 other treated animals, and 6 controls, were kept in LD (light-darkness) cycles of 10:14 h. After 14

Table 1. Effect of continuous light on the blood glucose of alloxan diabetic rats

Treatment	Blood glucose after 3 days mg %	Blood glucose after 17 days mg %	Adrenal wt mg/100 g body weight	Initial body weight	Body weight after 17 days
Control	101.02 $\pm$ 1.61*	98.45 $\pm$ 0.67	15.81 $\pm$ 0.32	147.51 $\pm$ 0.80	169.04 $\pm$ 0.61
Diabetic	457.23 $\pm$ 1.68	664.18 $\pm$ 3.91	28.97 $\pm$ 0.58	147.42 $\pm$ 0.42	133.98 $\pm$ 0.51
Diabetic+ light	462.51 $\pm$ 2.10	349.30 $\pm$ 2.51	18.49 $\pm$ 0.36	147.33 $\pm$ 0.58	153.63 $\pm$ 0.35
p-value:					
Control vs diabetes	<0.001	<0.001	<0.001	NS	<0.001
Control vs diabetes+ light	<0.001	<0.001	<0.01	NS	<0.001
Diabetes vs diabetes+ light	NS	<0.001	<0.001	NS	<0.001

\* Mean  $\pm$  SE; NS, indicates statistically not significant.

Table 2. Effect of continuous light on the urine volume and urinary glucose of alloxan diabetic rats

Treatment	Urine volume ml/24 h after 3 days	Urinary glucose g/100 ml/24 h after 3 days	Urine volume ml/24 h after 17 days	Urinary glucose g/100 ml/24 h after 17 days
Control	14.36 ± 0.38*		14.98 ± 0.44	
Diabetic rats	46.38 ± 1.09	3.53 ± 0.09	145.01 ± 0.88	8.60 ± 0.11
Diabetic + light	47.17 ± 1.11	3.68 ± 0.08	105.3 ± 1.36	5.82 ± 0.09
p-value:				
Control vs diabetes	<0.001	-	<0.001	-
Control vs diabetes + light	<0.001	-	<0.001	-
Diabetes vs diabetes + light	NS	NS	<0.001	<0.001

\* Mean ± SE; NS, indicates statistically not significant.

days of light exposure urine samples were collected and the blood was obtained by cardiac puncture. Finally both the blood and urinary glucose content were estimated. All the animals were killed by decapitation. The adrenal glands were dissected out free of fat and weighed.

**Results.** Alloxan treatment in the female rats resulted in a significant rise of blood glucose and its excretion in the urine. The urinary volume and the weight of the adrenal glands were also increased markedly in alloxan diabetic rats as compared with controls (LD 10:14 h). The alloxanized rats showed a significant fall in their body weights compared with the controls. Continuous light exposure of diabetic animals appeared to decrease the rise of blood and urinary glucose along with the urinary volume. The weight of the adrenal glands showed a return to control levels in the light-exposed diabetic rats. Body-weight gain was found in diabetic rats shifted to continuous light (tables 1 and 2).

**Discussion.** The data reported here show that continuous illumination results in a considerable fall of blood and urinary glucose content as well as urine volume in diabetic rats. Similar depression of blood glucose level has been noted in the normal grass snake exposed to lighting for a prolonged period<sup>12</sup>. The mechanism through which light affects blood glucose level is yet to be determined. It has been reported previously that continuous illumination depresses the adrenocortical activity in alloxan diabetic rats<sup>8,9</sup>. Plasma corticosterone values appear to be higher in the female rats during the evening than in the day time<sup>13</sup>. On the other hand Cheiftz et al.<sup>7</sup> have reported a fall of plasma corticosterone levels due to reduced corticotrophin secretion in intact female rats exposed to constant light. A decrease in adrenal weight, as observed in alloxan diabetic

rats shifted to continuous illumination, might be due to a similar cause.

Since the adrenocortical hypersecretion increases the severity of diabetes<sup>2,3</sup> and the removal of both the adrenal glands reduces the glucose output in alloxan diabetic rats<sup>6</sup>, the present investigation suggests that the fall of blood and urinary glucose content in alloxan diabetic rats exposed to light is possibly the result of suppressed adrenocortical activity.

- 1 Acknowledgment. The authors' thanks are due to Prof. A.K. Maiti, Department of Physiology, Calcutta University for his constant encouragement.
- 2 G.C. Saba, *Acta endocr.* 40, 349 (1962).
- 3 J.J. Hoet, *Acta endocr.* 40, 358 (1962).
- 4 D.D. Fowler, C.M. Szego and S.H. Sloan, *Endocrinology* 72, 626 (1963).
- 5 F. Bahmar and S.H. Haraszati, *Z. Ges. exp. Med.* 119, 23 (1952).
- 6 J.H. Exton, S.C. Harper, A.L. Tueker, J.L. Jean and C.R. Park, *Biochim. biophys. Acta* 329, 41 (1973).
- 7 P. Cheiftz, N. Gaffud and J.F. Dingman, *Endocrinology* 82, 1117 (1968).
- 8 N.M. Biswas and B. Paul, *Gegenbaurs morph. J.* 123, 759 (1977).
- 9 B. Paul, N.M. Biswas and S. Banik, *Ind. J. exp. Biol.* 17, 940 (1980).
- 10 O. Folin and H. Wu, *J. biol. Chem.* 41, 367 (1920).
- 11 R.B.H. Gradwohl, *Clinical Laboratory Methods and Diagnosis*, 5th edn. vol. 1, p. 54, 1956.
- 12 R. Skoczylas and E. Sidorkiewicz, *Comp. Biochem. Physiol. A* 48, 439 (1974).
- 13 J.A. Ramaley, *Steroid* 20, 185 (1972).

### Stimulating effect of the divalent cation ionophore A 23187 on in vitro neuroblast differentiation; comparative studies with myoblasts

A.M. Duprat and P. Kan<sup>1</sup>

Laboratoire de Biologie générale, Université Paul-Sabatier, ERA-CNRS No 327, 118, route de Narbonne, F-31062 Toulouse Cedex (France), 27 May 1980

**Summary.** The effects of the divalent cation ionophore A 23187 and papaverine on the in vitro differentiation of isolated embryonic cells from young neurulae (*Pleurodeles waltlii*) were observed. These experiments suggest that an intracellular increase or decrease of the divalent cation concentration ( $\text{Ca}^{++}$ ) stimulates or disturbs the morphological differentiation of already determined embryonic cells from neurulae but does not change their developmental pathway.

The involvement of  $\text{Ca}^{++}$ ,  $\text{Mg}^{++}$ ,  $\text{Na}^+$  and  $\text{K}^+$  in fundamental biological processes is now well established<sup>2-5</sup>. The important role of these cations in, for example, the early steps of embryonic development<sup>6-8</sup>, in the regulation of embryonic induction<sup>9-13</sup> and in morphogenetic movements such as neurulation<sup>14</sup>, is also now evident.

The primary effect of the ionophore A 23187 is to promote the transport of divalent cations, mainly  $\text{Ca}^{++}$ , either across an artificial lipid bilayer<sup>15,16</sup> or through the plasma membrane of cells<sup>17,18</sup> and also to release  $\text{Ca}^{++}$  ion from the intracellular organelles<sup>8,13,19</sup>. It is because of this that ionophore A 23187 is a very good tool for the in vivo study