laboratory bioassay. Results (table 1) suggest that female maturation[6] involves behavioural changes (increased receptivity) independent of any changes in the amount of sex pheromone, which is present in sufficient quantity on the day of emergence to elicit full copulatory responses from mature males.

1 We thank Dr A.M. Jordan for his helpful comments on the manuscript. The financial support of the Overseas Development Administration of the U.K. Government (Research Scheme R3450) and of the U.S. Department of Agriculture is gratefully acknowledged.

2 Address: U.S.D.A., S.E.A., A.R., Insects Affecting Man and Animals Research Laboratory, Gainesville (Florida 32604, USA).

3 G.A. Vale, Bull. ent. Res. *64*, 545 (1974).
4 G.A. Vale, Bull, ent. Res. *70*, 563 (1980).
5 J.P. Glasgow, The Distribution and Abundance of Tsetse. Pergamon Press, Oxford 1963.
6 T.G.T. Jaenson, Bull, ent. Res. *70*, 49 (1980).
7 P.A. Langley, R.W. Pimley and D.A. Carlson, Nature *254*, 51 (1975).
8 D.A. Carlson, P.A. Langley and P. Huyton, Science *201*, 750 (1978).
9 P.M. Huyton, P.A. Langley, D.A. Carlson and T.W. Coates, Physiol. Ent. *5*, 243 (1980).
10 P.M. Huyton, P.A. Langley, D.A. Carlson and M. Schwarz, Physiol. Ent. *5*, 253 (1980).
11 M.E. Leegwater van der Linden, Entomologia exp. appl. *29*, 169 (1981).
12 A.R. Mews, P.A. Langley, R.W. Pimley and M.E.T. Flood, Bull. ent. Res. *67*, 119 (1977).
13 E. Kovats, Adv. Chromatogr. *1*, 229 (1966).

Effects of D-glucose anomers on afferent discharge in the hepatic vagus nerve

T. Sakaguchi and M. Iwanaga

Departments of Physiology and Internal Medicine, Niigata University School of Medicine, Niigata City (Japan), 2 June 1981

Summary. Intraportal injections of α-D-glucose, optically equilibrated D-glucose and β-D-glucose reduced afferent discharges in the hepatic vagus nerve of anaesthesized rats. β-D-Glucose was most potent in decreasing the discharge.

Behavioral studies have presented good evidence for the existence of a neural glucosensitive mechanism in the hepatic portal vein[1]. Electrophysiologically, it has been shown that D-glucose injected into the portal vein affects afferent discharges taken from the hepatic vagus nerve in the isolated liver-vagus preparation[2]. D-glucose in the blood is known to exist as an optically equilibrated mixture of its 2 anomers; 36% α-D-glucose and 64% β-D-glucose[3], but no report has yet been published concerning the effect of D-glucose anomers on afferent discharges in the hepatic vagus nerve. Recently, it was observed that the vagus discharge was differentially changed by each of the glucose anomers injected into the portal vein in the rat.

Material and methods. 50 male rats weighing about 300 g were used. They were fasted for 22 h before each experiment, although allowed free access to water. The experiments were performed in the morning and animals, anaesthetized with pentobarbital sodium (45 mg/kg, i.p.), were adrenalectomized bilaterally 30 min before the beginning of nerve discharge recording to reduce variation in plasma levels of glucose in the blood[4]. Rectal temperature was maintained at about 36 °C by a heating lamp. Using elastic electrodes[5], nerve discharge was recorded from the distal cut end of the hepatic vagus nerve and expressed by the method described in the previous papers[2,6]. 5% glucose and 0.9% saline solutions were injected through a catheter inserted in the portal vein. The α or β form of crystallized D-glucose was dissolved in distilled water and kept at 36 °C immediately before use in order to eliminate mutarotation of the glucose. The purity of the 2 anomers was more than 98% and the rate of mutarotation of the 2 anomers in the water was negligible. Equilibrated D-glucose solutions consisting of 36% α-D-glucose and 64% β-D-glucose were obtained by keeping either α- or β-D-glucose solution for 22 h at room temperature (22 °C). The amount used for a portal injection was 0.2 ml and an injection was done for 15 sec via an infusion pump. Data were collected from the 1st response of each animal to a certain drug and differences were evaluated by Student's t-test.

Results and discussion. In a recording, reliable decreases in the afferent discharge of the hepatic vagus nerve were observed after the portal injections of 5% α-D-glucose, 5%

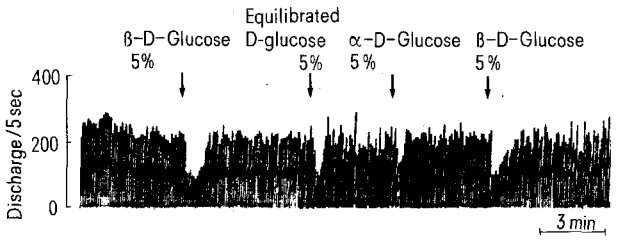

Figure 1. A recording showing the effects of D-glucose and its optical anomers on the afferent discharge of the hepatic vagus nerve. Arrows show the time of portal injection.

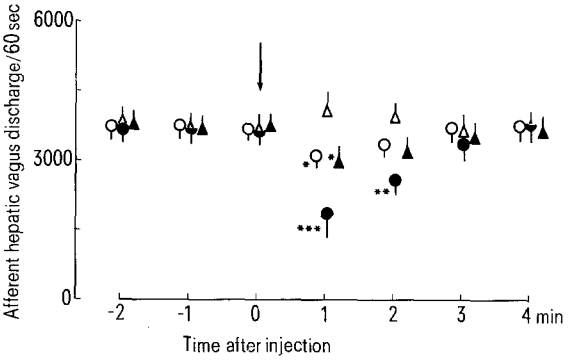

Figure 2. The effect of 5% α-D-glucose (○, n = 14); 5% equilibrated D-glucose (▲, n = 14); 5% β-D-glucose (●, n = 12); and 0.9% saline (△, n = 10) on afferent discharges of the hepatic vagus nerve. An arrow indicates the time of portal injection. Values are means ± SEM. *$p < 0.05$, **$p < 0.01$ and ***$p < 0.001$: significantly different from the value before injection.

optically equilibrated D-glucose and 5% β-D-glucose (fig. 1). No statistically significant differences between the spontaneous levels of discharge 0, 1 and 2 min before injection were observed in the α-D-glucose, equilibrated D-glucose, β-D-glucose and saline groups (fig. 2). In the saline group, the level of discharge did not change after the injections. In the α-D-glucose and equilibrated D-glucose groups, the levels of discharge 1 min after the injections decreased significantly ($p < 0.05$) to the levels before injection. The injection of β-D-glucose caused a further decrease in the discharge level; the levels of discharge were suppressed significantly 1 and 2 min ($p < 0.001$ and $p < 0.01$) after administration of the glucose and the decreases were significantly greater ($p < 0.05$–0.01) than those in the α-D-glucose and equilibrated D-glucose groups (fig. 2).

Afferent hepatic vagus discharge in the rat has been shown to decrease transiently after D-glucose injection into the portal vein (figs. 1 and 2). This gives support to the result of Niijima[2]. Regarding the physiological function of D-glucose anomers; several studies have disclosed that β-D-glucose is more rapidly taken up and metabolized by various cells and tissues than α-D-glucose[7–9]. Anatomically, hepatic afferent sensory nerve fibers have been identified as free nerve endings[10]. Since β-D-glucose was most potent in reducing the nerve discharge (fig. 2), the action of D-glucose on the hepatic nerve terminals seems to be predominantly due to an action of D-glucose on the intracellular glucose metabolism. These observations suggest that activation of hepatic glucosensitive mechanisms is dependent on the anomeric stereospecificity of D-glucose in the blood.

1 M. Russek, M.C. Lora-Vilchis and M. Islas-Chaires, Physiol. Behav. *24*, 157 (1980).
2 A. Niijima, Ann. N.Y. Acad. Sci. *157*, 690 (1969).
3 I. Miwa, K. Maeda and J. Okuda, Experientia *34*, 167 (1978).
4 T. Sakaguchi and K. Yamaguchi, J. Endocr. *85*, 131 (1980).
5 T. Sakaguchi, A. Warashina and A. Niijima, Pflügers Arch. *380*, 283 (1979).
6 T. Sakaguchi and K. Yamaguchi, Experientia *35*, 875 (1979).
7 R.G. Faust, J. cell. comp. Physiol. *56*, 103 (1960).
8 P.H. Fishman and J.M. Bailey, Am. J. Physiol. *226*, 1007 (1974).
9 I. Miwa, J. Okuda, H. Niki and A. Niki, J. Biochem. *78*, 1109 (1975).
10 T.L. Tsai, Acta Neuroveg. *17*, 354 (1958).

The role of volume expansion, of prostaglandins and catecholamines in the development of acute renal failure

N. Papanicolaou, G. Skoutelis, P. Papanicolaou, P. Theodorakopoulos, M. Paris, A. Dontas, J. Bariety and P. Milliez[1]

Laboratoire de Recherches sur les Hormones du Groupe de Recherches sur la Pathologie Rénale et Vasculaire, U. 28 de l'INSERM, Hôpital Broussais, F-75014 Paris (France), 13 avril 1981

Summary. A single injection either of isotonic or hypertonic saline solutions protected rats against acute renal failure (ARF) induced with glycerol. This protection was accompanied by increased urinary prostaglandin E (PGE) concentration. On the contrary, a single s.c. injection either of hypotonic saline or isotonic glucose solution, which did not increase urinary PGE concentration, or depletion of the endogenous catecholamines, using reserpine, did not protect the animals against acute renal failure.

In previous studies it has been observed that infusion of PGE_1 and PGE_2 partially protected the rats against ARF induced with glycerol[2,3]. On the other hand Wilson et al.[4] found out that volume expansion protected the rats against ARF. Since it had been observed that 1. volume expansion either of the intravascular or the extracellular space stimulated renal prostaglandin (PG) synthesis in rats[5,6] and dogs[7] 2. the increased release of PGs following volume expansion diminished in binephrectomized rats 3. the concentration of PGs in rat, rabbit an dog kidney (rather the capacity of renal tissue to synthetize PGs) is high[8–10], it has been suggested that 1. the kidney is the major source of PG synthesis and 2. renal PGs must play important rôle in the development of ARF. These suggestions have been strengthened by the observation that a PG synthesis inhibitor (indomethacin) enhanced ARF in rabbits[11] and prevented the protective effect of saline against ARF in rats[12] and moreover the addition of PGE_2 to a saline infusion restored its protective effect[12].

In these studies we investigated 1. whether the protective effect of saline against ARF is accompanied with an increased urinary PGE concentration, 2. whether the injection of isotonic glucose solution (which decreases PG synthesis)[13] protects the animals against ARF, and 3. whether the depletion of catecholamines, using reserpine, protects the animals against ARF. Our results are suggestive of a) an involvement of renal prostaglandins in the protection afforded by saline, b) an insignificant role of catecholamines in the development of this model of ARF.

Material and methods. Male Wistar rats 200–250 g (mean 221 ± 3 g) were used in this study. The animals were randomly allocated to 8 groups, each one consisting of 10 animals. ARF was induced by single s.c. injection in the anterior abdominal wall of 10 ml kg^{-1} of 50% (v/v) glycerol solution. The animals were subsequently deprived of food and water throughout the investigation. 24 h collections of urine were made, using metabolic cases.

Group 1 (G 50%): The rats were injected only with glycerol (50% 10 ml kg^{-1}). Group 2 (GG): The animals were injected with glycerol and isotonic glucose solution (75 ml kg^{-1}). Group 3 (GS 4.5 g l^{-1}): The animals were injected with glycerol and hypotonic saline (4.5 g l^{-1}, 75 ml kg^{-1}). Group 4 (GS 9 g l^{-1}): The animals were injected with glycerol and isotonic saline solution (9 g l^{-1}, 75 ml kg^{-1}). Group 5 (GS 18 g l^{-1}): The animals were injected with glycerol and hypertonic saline solution (18 g l^{-1}, 75 ml kg^{-1}). Group 6 (GS 150 g l^{-1}): The animals were injected with glycerol and hypertonic saline solution (150 g l^{-1}, 5 ml kg^{-1}). Group 7 (GR): The animals were pretreated with reserpine 4 days before they had been injected with glycerol in order to deplete catecholamines (2×0.5 mg kg^{-1} day^{-1} for 4 days). Group 8 (GRG): The animals were treated as those of the 7th group and injected with isotonic glucose solution (75 ml kg^{-1}) before they were injected with glycerol.