The forebrain of the animals was rapidly removed and put into a beaker with incubation medium 0-4 °C (Cl = 131.5 mM, PO_4H = 16.58; SO_4 = 1.22 mM; Na = 139.55 mM; K^+ = 4.79 mM; Ca^{++} = 2.73 mM; Mg^{++} = 1.21 mM and glucose = 9 mM). Cerebral slices were made by hand following the McIlwain technique (weight = 50 ± 10 mg). Oxygen consumption was measured, using a manometric technique[9]. Readings were made directly at 15, 30, 45 and 60 min. The glucose consumption was determined by the enzymatic method using glucose oxidase[10]. Statistical comparison of results was made by the Snedecor test[11].

Results. Results obtained are presented in the figure. In PCS rats O_2 consumption is significantly diminished (25-30%) throughout the experiment with respect to controls and sham-operated rats. Differences in O_2 consumption between control and sham-operated rats are not found. Glucose utilization diminishes (55%) with respect to controls and sham-operated rats (0.67 mg glucose/100 mg tissue) in PCS rats.

Discussion. The reduction of cerebral oxidative metabolism in PCS rats 7 days after operation is an index of early energetic alterations produced by PCS in the central nervous system. This decrease may be a consequence of a depletion of catecholamines[12] which could be caused by accumulation of false neurotransmitters. Based on these results, the Levodopa treatment of hepatic coma[6] which increases the catecholamine synthesis, may produce a displacement of false neurotransmitters[4] as well as a cerebral oxygen consumption increase and glucose utilization when Levodopa is administered in early PCS encephalopathy.

1 Acknowledgment. We thank Dr P. Garcia Partida for his assistance and Erik Lundin for his guidance in the preparation of the manuscript.
2 S. Sherlock, W.H.J. Summerskill, L.P. White and E.A. Phear, Lancet *2*, 453 (1954).
3 L. Zieve and D.M. Nicoloff, A. Rev. Med. *26*, 143 (1974).
4 B. Fischer, E.R. Fischer and S. Lee, Surg. Gynec. Obstetr. 1253 (1967).
5 A.T. Maiolo, G.B. Porro, C. Galli, M. Jessa and E.E. Polli, Exp. Biol. Med. *4*, 52 (1971).
6 M. Lunzer, I.M. James, J. Weinman and S. Sherlock, Gut *15*, 555 (1974).
7 J. Arias, F. Andres-Trelles and A. Alsasua, Arch. Pharmac. Toxic. *3*, 205 (1977).
8 H. McIlwain, in: Practical Neurochemistry, 2nd edn, p. 105. Churchill Livingstone, 1975.
9 W.W. Umbreil, R.H. Brunis and J.F. Stauffer, in: Manometric techniques, 5th edn, p. 70. Mineapolis Burges Co. 1972.
10 A. Sols and G. De La Fuente, Revta esp. Fisiol. *13*, 231 (1975).
11 G.W. Snedecor in: Statistical Methods, p. 534. Iowa State College Press Am., 1956.
12 A. Alsasua and J. Arias, Arch. Pharmac. Toxic. *V*, 97 (1979).

Intestinal glucose absorption in rats after secondary infections with *Nippostrongylus brasiliensis*

A.M. Scofield

Department of Biochemistry, Physiology and Soil Science, Wye College, University of London, Ashford, Kent TN25 5AH (England), 17 March 1980

Summary. A challenge infection of *Nippostrongylus brasiliensis* in immune rats resulted in an earlier onset of intestinal glucose malabsorption and increased glucose metabolism compared with rats receiving a primary infection. Intestinal absorption and metabolism recovered to control levels earlier during a secondary infection. The pattern of changes in absorption and metabolism was probably related to host immunological activity.

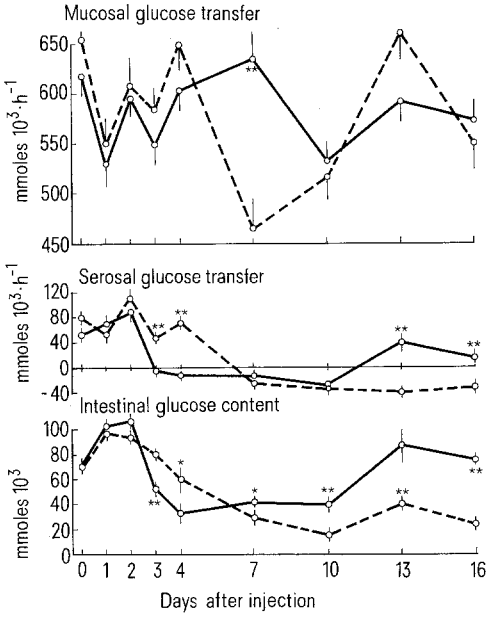
Studies of host intestinal glucose absorption after secondary infection with intestinal nematode parasites have produced differing results. In guinea-pigs receiving a secondary infection of *Trichinella spiralis* glucose malabsorption and the onset of pathological changes in the gut occurred earlier than after primary infection[1]. In contrast, mice failed to show any change in glucose absorption after secondary infection with *T. spiralis* although their mortality and worm

Glucose absorption by the small intestine was measured in vitro using a technique described in detail elsewhere[5]. The entire small intestine was removed from ether-anaesthetized rats, everted and divided into 3 segments of equal length to facilitate the subsequent procedures. Each segment was made into a sac and filled with test solution (Krebs-Ringer bicarbonate solution containing 10 mM D-glucose and gassed with oxygen containing 5% CO_2) and incubated for 1 h at 37 °C in 30 ml test solution in a 150-ml

results are shown in the figure. Means were considered significantly different when $p \leqslant 0.05$.

Results and discussion. In both experimental groups the rates of serosal glucose transfer and intestinal glucose content fell significantly during infection, the fall occurring earlier after secondary than after primary infection so that by 3 days after secondary infection the parameters were significantly lower than after primary infection. A return to normal values had occurred by 13 days after secondary infection whereas the values were still significantly depressed 16 days after primary infection and were significantly lower than the secondary infection group. The rate of mucosal glucose transfer varied with time after infection but in no regular manner. Significant reductions occurred 1 day after secondary infection, which is difficult to explain, and at 7, 10 and 16 days after primary infection and 10 days after secondary infection, which corresponded to significant reductions in the other parameters measured. The difference in the variation of these parameters with time may be related to the fact that mucosal transfer rate is largely a measure of the transfer capacity of the mucosa whereas serosal transfer rate and intestinal glucose content depend on transfer rate and the rate of glucose metabolism. Intestinal glucose metabolism increases during infection[5] and leads to a greater degree of change in serosal transfer rate and intestinal glucose content than in mucosal glucose transfer.

The present work demonstrates that significant reductions in the rate of accumulation and metabolism of glucose by the small intestine and its transfer across the wall of the gut occurred sooner after secondary infection with *N. brasiliensis* than after primary infection and that these parameters returned to normal more rapidly after secondary than primary infection. The pattern of these changes is probably related to more rapid stimulation of immunological activity following secondary than primary infection[7,8], and to less pronounced and shorter lasting tissue changes in the immune than non-immune host[9].



Glucose transfer rates and content in vitro of entire small intestine of female rats at various times after primary (dotted line) or secondary infection (continuous line) with 5000 larvae of *N. brasiliensis*. n = 15 for controls (day 0), 12 for 2-day group, 9 for 1- and 3-day groups and 6 for the rest. Vertical bars are SEM. *, ** Means of the primary and secondary infections significantly different at 5% or 1% levels respectively.

1 T.M. Lin and L.J. Olson, J. Parasit. *56*, 529 (1970).
2 L.J. Olson and J.A. Richardson, J. Parasit. *54*, 445 (1968).
3 A.M. Scofield, J. Parasit. *61*, 159 (1975).
4 P.A.G. Wilson and J.M. Dick, J. Helminth. *38*, 399 (1964).
5 A.M. Scofield, Int. J. Parasit. *7*, 159 (1977).
6 G. Morley, A. Dawson and V. Marks, Proc. Ass. clin. Biochem. *5*, 42 (1968).
7 E.E.E. Jarrett, W.F.H. Jarrett and G.M. Urquhart, Parasitology *58*, 625 (1968).
8 E.E.E. Jarrett, D.M. Haig and H. Bazin, Clin. exp. Immun. *24*, 346 (1976).
9 W.H. Taliaferro and M.P. Sarles, J. infect. Dis. *64*, 157 (1939).

Correlation between brain blood flow and catecholamine levels in rat brain areas under hypobaric hypoxia

M. Le Poncin-Lafitte, P.C. Pesquies and J.R. Rapin

Department of Physiology, CHU St-Antoine, 27, rue Chaligny, F-75012 Paris, and CRMAS, F-75731 Paris air cedex 15 (France), 13 February 1980

Summary. Hypobaric hypoxia induces an important increase in the cerebral blood flow in all areas and more particularly in the bulb and hypothalamus; the increase in the cerebral blood flow allows for an oxygen intake sufficient to maintain the norepinephrine level in these structures.

Most effects of hypoxia on brain catecholamines have been studied under normobaric hypoxia. When the amount of inspired oxygen was reduced from 21% to 8–6%, the activities of tyrosine hydroxylase[1,2], dopamine hydroxylase and monoamine oxidase were reduced[3]. At a simulated altitude of 7.000 m[4] or 5.200 m[5], a decrease in the brain catecholamine level was observed. The decrease is related to a decrease in synthesis, since the turnover of dopamine and of norepinephrine were reduced[6]. However, in a few brain areas, the norepinephrine turnover was not changed, even at an altitude of 7.000 m[7].

These findings can be explained by 2 hypotheses; either there is variable sensitivity, according to the brain areas, of oxygen-dependent enzymes necessary for catecholamine synthesis, or a sufficient oxygen intake is ensured by changes leading to a regional blood flow redistribution. This paper deals with the verification of the latter hypothesis.

Materials and methods. All experiments were performed in Long Evans male rats weighing between 200 and 260 g. They were carried out in a decompression chamber (CRMAS) at a simulated altitude of 7.000 m corresponding to a barometric pressure of 300 Torr and to a piO_2 of 53 Torr. Decompression was performed within 5 min, the temperature being maintained at 21 ± 1 °C. The ex-