University of Nebraska - Lincoln

DigitalCommons@University of Nebraska - Lincoln

Faculty Publications from the Harold W. Manter
Laboratory of Parasitology

Parasitology, Harold W. Manter Laboratory of

8-1982

Intestinal Absorption of β -methyl-D-glucoside in Rats Infected with *Eimeria nieschulzi*

Donald W. Duszynski

University of New Mexico, eimeria@unm.edu

K. Ramaswamy

University of South Carolina School of Medicine

Gilbert A. Castro

University of Texas Medical School

Follow this and additional works at: <https://digitalcommons.unl.edu/parasitologyfacpubs>

Part of the [Parasitology Commons](#)

Duszynski, Donald W.; Ramaswamy, K.; and Castro, Gilbert A., "Intestinal Absorption of β -methyl-D-glucoside in Rats Infected with *Eimeria nieschulzi*" (1982). *Faculty Publications from the Harold W. Manter Laboratory of Parasitology*. 179.

<https://digitalcommons.unl.edu/parasitologyfacpubs/179>

This Article is brought to you for free and open access by the Parasitology, Harold W. Manter Laboratory of at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in Faculty Publications from the Harold W. Manter Laboratory of Parasitology by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.

RESEARCH NOTES

J. Parasitol., 68(4), 1982, pp. 727-729
© American Society of Parasitologists 1982

Intestinal Absorption of β -methyl-D-glucoside in Rats Infected with *Eimeria nieschulzi*

Donald W. Duszynski,* K. Ramaswamy,† and Gilbert A. Castro, Department of Physiology and Internal Medicine, University of Texas Medical School, Houston, Texas 77030. *Present address: Department of Biology, University of New Mexico, Albuquerque, New Mexico 87131. †Present address: Gastroenterology Division, Department of Medicine, University of South Carolina School of Medicine, Veterans Hospital Enclave, Columbia, South Carolina 29201

Reduced weight gain or weight loss is one of the most obvious symptoms of coccidiosis in vertebrate animals. Studies with pair-fed animals (e.g., Michael and Hodges, 1971, *Vet. Rec.* **89**: 329-333) generally show that weight depression is not strictly a matter of anorexia. Some of the physiological mechanisms known to contribute to weight depression during coccidiosis include morphological changes in the architecture of the gut (Whitlock and Ruff, 1977, *J. Parasitol.* **63**: 193-199), ultrastructural changes in both infected and uninfected epithelial cells (Sheppard, 1974, *J. Parasitol.* **60**: 369-391), systemic effects of the parasite on blood parameters (Ruff et al., 1978, *J. Parasitol.* **64**: 23-26), the malabsorption of various nutrients (Turk, 1974, *Am. Soc. Exp. Biol.* **33**: 106-111), and others. We know from earlier work with *Eimeria nieschulzi* in rats that these mechanisms are regulated by the area of the gut parasitized, the stage of the parasite's life cycle, and the severity of the infection (Duszynski et al., 1978a, *J. Parasitol.* **64**: 83-88; 1978b, *J. Protozool.* **25**: 226-231; 1978c, *J. Protozool.* **25**: 370-374). Another mechanism that may contribute to the weight depression seen in mammals infected with coccidia could be an altered rate at which nutrients influx into the mucosa, but previous studies by other authors have not dealt with uptake uncomplicated by metabolism. Stein and Marquardt (1973, *Exp. Parasitol.* **34**: 262-267) examined glucose absorption by tracing the passage of ^3H -glucose through the mucosa of the intestine of rats infected with *E. nieschulzi*. They also injected ^3H -glucose into the duodenum and measured radioactivity in blood samples collected from the hepatic por-

tal vein. Their in vitro results showed impaired absorption of ^3H -glucose only on day 8 postinfection (p.i.); their in vivo data indirectly indicated that rats had no compensatory absorption by other parts of the small intestine. Although their data are reasonable and support similar studies with chickens, an inherent problem of interpretation occurs because ^3H -glucose can be metabolized in its passage through the mucosa. Thus, although they assumed that all the label measured was glucose, a part of it may have been converted to some tissue metabolite (e.g., CO_2 and lactate) which was also counted as ^3H -glucose. Here we measured the uptake rates of β -methyl-D-glucoside (a non-metabolizable analog of glucose that is transported by the same monosaccharide-transport carrier as glucose) by mucosal cells in uninfected rats and in rats infected with *E. nieschulzi*. Our studies were designed to: (1) record the effect of the parasite on mucosal uptake of the glucose analog along the length of the gut; (2) determine whether or not there were any compensatory increases in uptake in uninfected parts of the gut; and (3) measure the extent of recovery of uptake rates.

Male Sprague-Dawley rats (Sprague-Dawley, Madison, WI), weighed 200 to 250 g and were inoculated with either 10^4 or 10^6 sporulated oocysts (3-4 mo old) of *E. nieschulzi*. Infected rats were maintained as in earlier studies (Duszynski et al., 1978a, loc. cit.). On each of days 2 and 4 (early merogony), 7 and 9 (peak parasitemia and gamogony), and 16 (host recovery) p.i., respectively, one pair of uninfected control rats and one pair infected with each dose of oocysts were killed by a

FIGURE 1. Rate (mean $\pm$ SE) of β -methyl-D-glucoside uptake by the mucosa of proximal, mid, and distal segments of small intestine of pairs of rats infected with 10^4 and 10^6 sporulated oocysts of *E. nieschulzi*. Asterisk indicates significance ($P < 0.05$). Data for days 2 and 4 (early merogony) and 7 and 9 (peak parasitemia) are combined ($n = 4$ for each bar).

blow on the head. The abdomen of each rat was opened and the small intestine was cut at the duodenum and the ileo-cecal valve. A

glass cannula was inserted into the duodenum and the intestine was perfused with 50 ml of cold (4 C), 0.85% (w/v) aqueous NaCl. The

intestine was measured, divided into thirds, and the middle 15 cm were removed from each third to determine the uptake rate of β -methyl-D-glucoside.

Each 15 cm segment was weighed and intestinal sacs were prepared so that the serosal side of each segment was rendered inaccessible to the substrate (for details on the preparation of these sacs see Malathi et al., 1973, *Biochim. Biophys. Acta* **307**: 613–626). Briefly, sacs were made by everting the intestine onto polyethylene tubing (PE280), tying off small segments to the tubing and then cutting between ties. Sacs were incubated in 10 ml of Krebs-Ringer phosphate buffer containing ^{14}C - β -methyl-D-glucoside. Extracellular monosaccharide concentration was determined by the use of ^3H -mannitol. The concentration of β -methyl-D-glucoside used here (30 mM), which was found to saturate the transport system, and an incubation time of 5 min (when the uptake was linear) were found to be the optimal conditions to compare initial rates of uptake between uninfected and infected rats. Two hundred to 300 mg of tissue was used for each incubation flask. Sacs were processed at the end of incubation as reported by Crane and Mandelstam (1960, *Biochim. Biophys. Acta* **45**: 460–476). Radioactivity was measured by a Beckman Liquid Scintillation Spectrometer, using Beckman Ready-Solv fluid. Results are expressed as concentration of monosaccharide in tissue water, assuming a water content of 80% of the wet tissue weight.

Infection with 10^4 oocysts did not effect weight gain of rats used in this study whereas infection with 10^6 oocysts produced weight loss by days 7 and 9 p.i., and this loss was still evident at day 16 p.i.

Early in the infection (days 2 and 4 p.i.), uptake rates were depressed in the proximal two-thirds at both dose levels, but these reductions were not statistically significant. The rates of uptake of β -methyl-D-glucoside were significantly depressed in the first two-thirds of the small intestine on days 7 and 9 p.i. at both infection levels. By the time patency

ended (16 p.i.) most rates measured were lower than in corresponding controls, but not statistically so. Throughout patency, uptake rates in control and infected animals were similar in the distal one-third of the small intestine (Fig. 1).

The reduction in uptake rates seen here can be correlated with an increase in the total weight of the mucosa accompanied by hypertrophy of the crypts (Duszynski et al., 1978b, loc. cit.). Electron microscopy of rat midgut infected with *E. nieschulzi* has shown significant ultrastructural changes in villus epithelial cells and also that cells at villus tips resembled undifferentiated crypt cells (Shepard, 1974, loc. cit.). Hence, these reductions in uptake could be explained on this basis alone because it is well known that undifferentiated crypt cells are not absorbing cells (Symons et al., 1971, *Int. J. Parasitol.* **1**: 179–187).

We do not know whether the reduced uptake rates we measured play any significant role in total assimilation of dietary nutrients. In fact, some effects of the parasite may compensate for others. We know that *E. nieschulzi* will slow the rate of transit of intestinal contents during peak patency of heavy infections (Duszynski et al., 1978c, loc. cit.). This decreased movement of gut contents would permit its prolonged contact with the intestinal mucosa and thus allow more time for uptake of nutrients to occur across a mucosal surface in which the rate of uptake has been impaired. Finally, the mammalian intestine has a high reserve capacity for absorption (Crane and Mandelstam, 1960, loc. cit.; Dowling and Riecken, 1974, *Intestinal adaptation*, F. K. Schattauer Verlag, Stuttgart-New York, 271 p.); thus even when uptake is reduced in infected areas of the small intestine, absorption by unaffected regions might be sufficient for adequate nutrition. However, the loss of weight might argue against this.

This study was supported by Grant AM-11361 from the NIAID, NIH. Dr. Castro is the recipient of Research Career Development Award AI-00087.