indicating that both the de-novo and the transmethylation pathways are active in the swine arteries.

The patterns of incorporation in the aorta and pulmonary arteries were not significantly different from those of the coronary arteries; the degrees and distribution of radioactivity incorporation were similar to those found on incubation of the same precursors with rabbit aortas[4] or human peripheral arteries[6]. There is, therefore, no evidence from these experiments to suggest that the different susceptibilities to atherosclerosis of the coronary arteries and aorta vs. the pulmonary artery are attributable to differences in phospholipid metabolism of these arteries[9].

*Zusammenfassung.* Der Grad des Einbaus von $^{14}$C-markierten Prekursoren in die Arterienphospholipide des Schweins war folgender: Cholin > Aethanolin > Serin > Glycerin > Format > Azetat. Bei Bebrütung von Arterien mit $^{14}$C-Cholin oder $^{14}$C-Aethanolamin trat ein deutlich erhöhter Einbau von $^{14}$C in das Phosphatidylcholin auf.

R. J. Morin

*Departments of Pathology,*
*Los Angeles County Harbor General Hospital,*
*Torrance (California 90509, USA), and the*
*U.C.L.A. School of Medicine,*
*Los Angeles (California, USA), 28 April 1970.*

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## Intestinal Absorption of D-Xylose by Germfree Rats

It is now recognized that the morphology of the intestine is determined in important ways by the presence of the normal intestinal microflora. Recent understanding of these effects has come from studies of germfree animals which were shown to have less mucosal surface area[1] and reduced mucosal cell turnover rate[2–4] when compared to conventional control animals. Differences also have been observed in the morphology of the reticuloendothelial elements of the intestinal mucosa[2,5]. Despite these morphological observations, a surprisingly small amount of information is available concerning intestinal function in germfree animals. We have begun a series of investigations[6] to determine the effects of microorganisms on intestinal absorption and this report summarizes observations on the absorption of D-xylose by germfree and conventionalized rats.

*Materials and methods.* Fischer strain rats were obtained germfree from the Charles River Breeding Laboratories, Willmington, Massachusetts immediately after weaning. They were divided randomly into 2 groups of equal numbers of male and female rats and were transferred to separate Trexler-type, flexible plastic isolators[7]. One group was 'conventionalized' by a procedure previously described[6] and served as the control group. The other group of rats remained germfree. Both groups received sterilized diet L-356[8], sterilized distilled water ad libitum, and a vitamin supplement. They were otherwise maintained under identical environmental conditions, the single exception being the presence of the microflora in the conventional rats. Germfree status was determined biweekly with standard microbiologic techniques[6].

At the age of 6.5 months, 250 mg of D-xylose in 0.5 ml of water was administered to each rat by gastric tube. 6 h following administration of the test solution, the rats were killed with chloroform and the gastrointestinal tracts removed. The contents of the stomach, small intestine, and large intestine were collected separately by washing with isotonic saline solution. The total amount of D-xylose remaining in each section of the gastrointestinal tract was determined using the method of Roe and Rice[9]. The percentage of D-xylose absorbed was calculated by the procedure of Makela et al.[10] which accounts for differences in gastric emptying.

*Results and discussion.* The results are presented in the Table. In germfree rats, 11.3% of the D-xylose remained in the stomach at the end of the test period compared to 3.8% in the stomachs of conventionalized rats. This difference was not significant at the 0.05 level but was similar to observations made previously which indicate a decreased rate of gastric emptying in germfree rats[6,11]. There was no difference between germfree and conventionalized groups in the amount of D-xylose which remained in the small intestine but significantly more was recovered in the cecum and colon of germfree rats ($P < 0.025$). The net absorption was 74.3% in the germfree rats and 87.8% in the conventionalized rats, a significant difference ($P < 0.025$).

These observations are different from those previously reported by Heneghan[12] in which it was concluded that xylose is absorbed more rapidly in germfree rats. Our data are not comparable to his because he measured absorption during the first hour following administration, a time when both germfree and control animals had absorbed less than half of the administered dose. There may have been other differences which are not immediately apparent, e.g., in the composition of the intestinal microflora of the control animals.

Our observations suggest that normal intestinal microorganisms have a significant effect on the intestinal absorption of D-xylose but the explanation is not completely clear. We do not believe the decreased absorption in germfree rats was related to intestinal motility. Abrams

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| | Number | Body weight (g) | Dose remaining 6 h following intragastric administration (%) Stomach | Small intestine | Cecum and colon | Absorption[b] (%) |
|---|---|---|---|---|---|---|
| Germfree | 8 | 218.0 ± 16.0[a] | 11.3 ± 4.9[a] | 6.0 ± 1.5[a] | 17.7 ± 4.6[a] | 74.3 ± 4.6[a] |
| Conventionalized | 8 | 222.1 ± 19.4 | 3.8 ± 1.8 | 6.0 ± 1.3 | 5.7 ± 2.0 | 87.8 ± 2.5 |
| *P* value[c] | | | $<0.2->0.1$ | $>0.9$ | $<0.025$ | $<0.025$ |

[a] Mean ± S.E. of mean. [b] Corrections were made for differences in gastric emptying by considering only the amount of D-xylose which actually left the stomach was available for absorption[9]. [c] Student's *t*-test.

and BISHOP[11] have shown that the time necessary for intestinal transit is acutally longer in germfree mice, and a similar observation has been made in germfree rats[6]. Degradation of D-xylose by intestinal bacteria is negligible under the condition of our studies[12,13] and could not have been responsible for the differences observed. We believe that the differences in D-xylose absorption are most likely related to differences in mucosal absorption per se. This interpretation suggests a functional correlation with previously reported morphologic studies in which germfree rats were shown to have significantly less mucosal surface area than conventional controls[1].

*Résumé.* Les observations faites sur les rats gnotobiotiques et les rats normaux montrent que l'absorption de D-xylose est beaucoup plus elevée chez les rats normaux que chez les rats gnotobiotiques. Cette différence est attribuée au changement de transport muqueux produit par la présence de microrganismes intestinaux.

B. TENNANT, M. REINA-GUERRA and D. HARROLD

*Department of Clinical Sciences, School of Veterinary Medicine, University of California Davis, Davis (California 95616, USA), 19 May 1970.*

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## Kallikrein-Like Activity in the Urine of Renal Hypertensive Rats

Since evidence has been given that kinins produce changes in the renal blood flow and sodium excretion[1-3], it was considered of interest to study the enzymatic systems which might be involved in the production or in the release of kinins in the kidneys. This communication describes the results obtained in measurements of the kallikrein-like activity (KA) found in the urine of rats in which different reductions in renal functional mass were produced. Interest was focused on groups of rats which after manipulation of the kidneys developed renal hypertension. The study was undertaken in 114 Wistar and 30 Sprague Dawley male rats distributed in different groups: a) 42 normal; b) 35 uninephrectomized; c) 17 figure-of-8-ligature in one kidney; d) 50 uninephrectomized and with figure-of-8-ligature in the remnant kidney. In the latter group the operation was carried out according to the GROLLMAN[4] procedure to induce the development of renal hypertension. Blood pressure was measured in the rat tail at weekly intervals beginning 10 days after kidney removal. The microphone technique described by FRIEDMAN and FREED[5] was used. During blood pressure determinations the animals were kept quiet with light ether anesthesia. Every week or fortnight, the urine was collected by placing the animals in metabolic cages in the morning for 3–9 h. No food was given but they had free access to drinking water during the collection period. In many experiments only the freshly voided urine was used for testing, but in some of the cases the urine was collected for several hours in order to carry out purification procedures. Toluene, as antiseptic, was added (2%) when the collection period lasted more than 4 h. Each sample of urine was measured, centrifuged and kept frozen in the refrigerator when not immediately assayed.

The KA was tested either in dialyzed or non-dialyzed urine using 2 bioassays: the contraction of an isolated rat uterus and the depressor effect on the blood pressure of an anesthetized rat. According to BERALDO et al.[6] and CROXATTO et al.[7] urinary kallikrein induces a direct oxytocic effect upon isolated rat uterus and its effect is dose dependant. This effect is inhibited by Trasylol (Bayer) and carboxypeptidase B. The urine keeps its kallikrein activity after prolonged dialysis against distilled water in the cold room. The responsible substance releases kinins very rapidly in the presence of kininogen. A standard bradykinin solution was used to quantificate the urine effect upon the isolated rat uterus and blood pressure. The KA was expressed in ng of bradykinin which produces equivalent oxytocic effect induced by 1 ml of urine. No significant differences were found between a dialyzed or non-dialyzed fraction of the same urine sample.

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