Zeitschrift: Acta Tropica
Herausgeber: Schweizerisches Tropeninstitut (Basel)
Band: 38 (1981)
Heft: 2

Artikel: Influence of systemic infections on xylose absorption
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DOI: https://doi.org/10.5169/seals-312817

Acta Tropica *38*, 173–178 (1981)

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Influence of systemic infections on xylose absorption

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Summary

Serial blood-xylose concentrations have been determined in 28 Papua New Guineans after 25 g oral xylose; 9 had an acute and 8 a chronic systemic infection, 4 the tropical splenomegaly syndrome and 7 were free of infection. Mean xylose was significantly depressed at 30, 60, 90 and 120 min in those with acute and chronic infections. Correlations between xylose and serum albumin, α_1, α_2, β- and γ-globulin concentrations were not significant. When the xylose test is used in tropical countries the effect of systemic infections on the result must be careful evaluated, otherwise tropical malabsorption ('tropical sprue') might be diagnosed erroneously.

Key words: systemic infection; infection; blood xylose; absorption.

Introduction

A significant reduction in urinary excretion of xylose after an oral load has previously been demonstrated in the presence of acute and chronic systemic infections (Cook, 1972, 1974a, 1974b; Lancet, 1980). In the present investigation, blood-xylose concentrations have been estimated after oral xylose in Papua New Guineans with and without such infections.

Patients and methods

Table 1 gives details of 28 inpatients at the Port Moresby General Hospital; all were well-nourished and none had clinical evidence of malabsorption or diabetes mellitus. Four groups were

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Table 1. Details of four groups of patients investigated*

Group	No. studied	Age (yr)	Sex		Body-weight (kg)	Haemo-globin (g dl^{-1})	Serum protein (g l^{-1})				
			M	F			albumin	globulin			
								α_1	α_2	β	γ
Acute infection (A)	9	24 (14–46)	7	2	55 (45–74)	11.2 (6.9–15.4)	27.4 (16–34)	4.9 (3–7)	8.2 (6–11)	7.0 (7–11)	27.3 (16–37)
Chronic infection (B) ..	8	27 (15–50)	7	1	45 (37–60)	11.2 (9.2–13.6)	25.6 (8–39)	3.9 (2–6)	8.0 (3–12)	6.5 (3–10)	26.4 (16–43)
Tropical splenomegaly syndrome (C)	4	27 (23–30)	3	1	54 (49–60)	8.3 (7.8–9.1)	30.2 (28–35)	2.7 (2–4)	4.0 (3–5)	4.2 (4–5)	40.0 (27–47)
Control (D)	7	22 (12–38)	5	2	53 (40–88)	10.4 (5.7–14.6)	35.6 (31–39)	3.0 (2–4)	6.6 (4–8)	7.3 (6–9)	22.1 (19–28)

* Mean (and range) are given for each index.

delineated on the basis of clinical and laboratory criteria. Group A consisted of 9 patients with acute infections. Diagnoses were: bacterial lobar pneumonia or another acute chest infection (5), septic arthropathy (2), and acute *Plasmodium falciparum* malaria (2); 5 were pyrexial at investigation. Chest infections were confirmed by radiography (in 3 by sputum bacteriology also); arthropathy was confirmed by joint aspiration; *P. falciparum* was demonstrated in the peripheral blood of those with malaria. Four with chest infections had started penicillin treatment. Group B consisted of 8 patients with chronic infections. Diagnoses were: pulmonary tuberculosis (7), and tuberculous peritonitis (1); 2 were pyrexial at investigation. Diagnosis was confirmed by radiography (in 4 *Mycobacterium tuberculosis* was isolated from the sputum also) or peritoneal biopsy. Two with pulmonary tuberculosis had started streptomycin, thiacetazone and isoniazid treatment. Group C consisted of 4 patients with the tropical splenomegaly syndrome (Cook, 1980) which is thought to be a result of an aberrant immune response to the malaria parasite. In all, the spleen was palpable at least 15 cm below the costal margin. Thick blood-films did not show evidence of a current malaria infection. One was receiving penicillin for a mild respiratory infection which had already resolved. Group D consisted of 7 patients who were used as controls; none had evidence of infection. All had trivial symptoms, only, and none was pyrexial. The 2 anaemic patients were receiving oral iron and folic acid.

Table 1 gives details of mean haemoglobin and serum protein concentrations. Seven and 3 in groups A and B had a polymorphonuclear leucocytosis. Two in group C had a leucopenia; they also had a reticulocytosis. Those in group D had a normal blood leucocyte count, and 2 had an iron-deficient anaemia of undetermined origin, which was confirmed by bone-marrow examination. Serum urea was elevated in 4 of group A (10.3, 10.5, 22.0 and 27.2 mmol l^{-1}) due to dehydration; all others were normal. In 2 in group A (lobar pneumonia and malaria), serum bilirubin (51 and 375 μmol l^{-1}) and glutamic oxalo-acetic transaminase (76 and 100 iu l^{-1}) concentrations were elevated. Serum bilirubin was also elevated in 2 in group C (27 and 20 μmol l^{-1}). In 1 patient in group B (extensive cavitating tuberculosis), alkaline phosphatase was 102 iu l^{-1}. Aspiration liver biopsies were obtained in 3 patients in group C; all showed marked sinusoidal lymphocytosis.

Following an approximately 10-h overnight fast, 25 g xylose (Koch-Light Ltd, England) dissolved in 500 ml water at room temperature, was ingested. The patients were all sitting throughout the tests. In groups A, B and D, blood samples were obtained at 0, 15, 30, 60, 90, 120 and 150 min after the mid-point of xylose ingestion, and were immediately added to 2 vol. zinc sulphate (18 g l^{-1}) + 2 vol. barium hydroxide (18 g l^{-1}). In the patients in group C, samples were obtained at 60, 90 and 120 min only, but processed similarly. Following mixing and centrifugation, the supernatant was separated and frozen at –20° C until xylose estimation by a colourimetric method (Roe and Rice, 1948). All determinations were made in duplicate, and separate standard curves were constructed at the beginning and end of each set of estimations. Serum total protein was estimated by the biuret method, and electrophoresis using cellulose polyacetate membranes (Gelman instrument Co., Michigan, USA).

Results

Fig. 1 summarizes serial blood-xylose concentrations in groups A, B and D; mean concentration was significantly reduced at 30, 60, 90 and 120 min in groups A and B compared with D. Peak concentrations in group D were at 90 and 120 min. In 2 of group A, blood-xylose concentration did not rise above 1.0 mmol l^{-1}; one had lobar pneumonia and the other acute malaria. There was no evidence that any of the therapeutic agents had influenced blood-xylose concentrations in those individuals receiving them. Table 2 summarizes mean blood-xylose concentrations in group C. Correlations between xylose, and serum albumin, α_1, α_2, β- and γ-globulin concentrations were not significant.

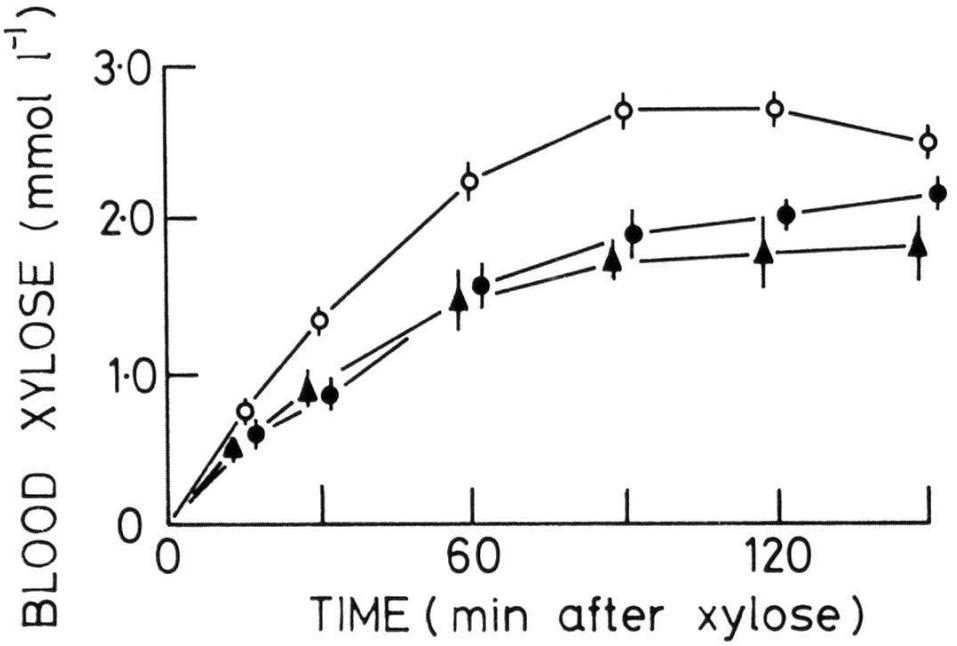

Fig. 1. Blood-xylose concentrations after 25 g oral xylose. Means ± 1 S.E.M. are shown for groups A (▲), B (●) and D (○). Means for those with infections (groups A and B) were significantly depressed at 30 ($t = 2.89$; $p < 0.01$), 60 ($t = 3.07$; $p < 0.01$), 90 ($t = 6.07$; $p < 0.001$) and 120 ($t = 3.18$; $p < 0.01$) min.

Table 2. Blood xylose concentrations in group C

No. studied	Blood xylose concentration (mmol l^{-1})*		
	60 min	90 min	120 min
4	2.40 (0.44)	2.31 (0.27)	2.24 (0.24)

* Mean (and 1 S.E.M.) are given for each index.

Discussion

In most patients with systemic infections the blood-xylose curve after oral xylose is relatively flat; that should be taken into account in interpretation of the xylose test in 'third-world' countries, where such infections are very common; otherwise tropical malabsorption ('tropical sprue') (Lancet, 1980) might be suspected. Although xylose is partly metabolized by the liver (Wyngaaden et al., 1957; Segal and Foley, 1959), it seems likely that most of the depression was due to impaired small-intestinal absorption. There was not a significant correlation between xylose and serum γ-globulin concentrations; a significant *inverse* correlation has previously been demonstrated for xylose excretion (Cook, 1974c) and glucose absorption rate (Cook, 1973), and serum γ-globulin concentration. Although jejunal histology was not examined in the present study it seems extremely unlikely that the illnesses in group A were of long enough

duration to have affected that significantly; such infections have previously been shown not to alter morphology (Cook, 1971, 1972, 1974a, 1974b). Xylose concentrations at 120 min were closely approximated in all of the groups studied; that is presumably explained by the fact that in the rapid absorbers the bulk of the xylose load had already left the jejunum, while in the slow absorbers some xylose was still present in the distal small-intestine. The role of gastric emptying was not assessed in the present investigation.

Although only limited data were obtained, mean blood-xylose concentration seemed to reach a high level earlier in the group with the tropical splenomegaly syndrome; that is probably a result of the gross increase in perfusion within the portal system which is associated with a high plasma volume.

Although xylose and glucose might share the same transfer mechanism (Salomon et al., 1961; Alvarado, 1966), xylose is absorbed much more slowly (Cook, 1977). Glucose absorption rate from the jejunum has been shown to be depressed by systemic infections (Cook, 1971); plasma insulin concentrations, in response to oral glucose, are also likely therefore to be depressed. Because insulin is atherogenic (Lancet, 1977), that might explain in part why rural people in 'third-world' countries have a greatly reduced incidence of coronary atherosclerosis; they live largely on carbohydrate, but most have at least one systemic infection. It seems probable that blood concentrations of some therapeutic agents are depressed in patients with systemic infections; that might have important therapeutic implications.

The present study indicates that 90 min after xylose ingestion is the best time for blood xylose measurement (Sladen and Kumar, 1973); the greatest difference in means between those with and without infections occurred at that point. Using a 5-g oral load, the 60-min sample gave the most reliable result (Haeney et al., 1978). In a 'third-world' country where accurate 5-h urine collections are difficult to obtain, blood concentration at 90 min probably gives the most reliable index of xylose absorption from the upper small-intestine.

Acknowledgments. I am grateful to Miss M. Johnson for the liver function tests and serum urea concentrations, and to Dr. J. C. White for the haematological indices.

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