# Xylose absorption in Papua New Guineans with leprosy

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# Xylose absorption in Papua New Guineans with leprosy

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## Summary

Blood xylose concentrations were measured at 60, 90 and 120 min after 25 xylose was given orally in 33 well-nourished Papua New Guinean in-patients at Port Moresby; 12 had lepromatous (group A) and seven non-lepromatous (group B) leprosy, and 14 were controls (group C). Differences between mean xylose concentrations were not significant at any time interval. Three patients (two in group A and one in group B) had 90 min xylose concentrations $<2.0$ mmol $l^{-1}$. There was no association between xylose concentration and current therapy. Leprosy does not impair small-intestinal absorptive function; it therefore differs from other chronic infections, in which there is xylose malabsorption.

**Key words:** leprosy; absorption; xylose absorption; small intestine.

## Introduction

Acute and chronic systemic infections significantly impair xylose absorption (Cook, 1980, 1981). Leprosy is a systemic infection: *Mycobacterium leprae* can be detected in many organs including the liver and spleen; however, in man deep viscera are not usually affected morphologically.

In the present investigation, blood xylose concentrations have been estimated after oral xylose in well-nourished Papua New Guinean (PNG) adults with leprosy; all were undergoing treatment. It was felt possible that one or more of the therapeutic agents, apart from the disease, might interfere with absorption.

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Table 1. Details of the patients with leprosy, and the controls*

| Group | No. | Age | Sex | | Body-weight (kg) | Haemoglobin (g $dl^{-1}$) | Serum protein (g $l^{-1}$) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | M | F | | | albumin | globulin | | | |
| | | | | | | | | $\alpha_1$ | $\alpha_2$ | $\beta$ | $\gamma$ |
| Lepromatous leprosy (A) ..... | 12 | 29 (12–50) | 7 | 5 | 52 (33–76) | 10.7 (8.2–13.9) (n = 8) | 31 (25–37) | 3 (1–6) | 8 (4–12) | 8 (6–11) | 26 (22–34) |
| Dimorphous or 'burnt out' leprosy (B) ..... | 7 | 38 (25–54) | 4 | 3 | 57 (42–69) | 10.5 (9.2–13.0) (n = 4) | 35 (27–42) | 3 (2–6) | 6 (3–9) | 8 (4–10) | 25 (18–30) |
| Control (C) ..... | 14 | 27 (16–52) | 9 | 5 | 53 (39–88) | 10.7 (5.7–14.6) (n = 7) | 37 (31–46) | 3 (2–4) | 6 (4–9) | 7 (6–10) | 22 (15–30) |

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

Table 2. Details of therapeutic agents at time of test in groups A and B

| Therapeutic regime | Group | |
|---|---|---|
| | A | B |
| Dapsone | 1 | 4 |
| Dapsone + clofazimine | 4* | 3** |
| Dapsone + clofazimine + chloroquine | 5** | – |
| Dapsone + clofazimine + thalidomide | 1 | – |
| Clofazimine + thalidomide | 1 | – |

* One patient was also receiving isonicotinic acid hydrazide and streptomycin for a *M. tuberculosis* infection.

** One patient was also receiving prednisone.

**Patients and Methods**

Table 1 summarizes the diagnosis, age, body-weight and sex of 33 PNG in-patients at the Port Moresby General Hospital; they came from many provinces of PNG. All were well-nourished; none had clinical evidence of gastro-intestinal disease, although some with leprosy did have abnormal hepatic structure and function (vide infra). Three groups (A–C) were delineated. Six in group A also had *erythema nodosum leprosum* (ENL) and two had Jopling's type 1 reactions. One with ENL was mildly jaundiced and two had hepato-splenomegaly (one had secondary hepatic amyloidosis and the other a *Mycobacterium tuberculosis* infection of the tarsus); three others had mild splenomegaly. In group B, two had hepato-splenomegaly (one was subsequently shown to have a hepato-cellular carcinoma), and one had osteomyelitis of the foot. Of those admitted with trivial symptoms only, and no evidence of an infection (group C), results for seven have already been reported (Cook, 1981); one had mild hepatomegaly, one splenomegaly, and two were anaemic. Table 2 gives details of the therapeutic regimens at the time of investigation.

Nine patients in group A had *M. leprae* in skin smears: mean bacterial index (Ridley, 1958) in eight was +3 (1–6); morphologic index (Shepard and McRae, 1965) ranged from 2 to 94% in five of them. In one, a nerve biopsy revealed numerous *M. leprae*, and in another skin smears were negative. In group B, two had evidence of a *M. leprae* infection: mean bacterial index was +3 (3–4) and morphologic indices were 4 and 35%. In three, skin smears were negative.

Table 1 also gives details of mean laboratory indices. Mean total white-blood-cell counts in groups A, B and C were 10.7 (8.2–12.1) (n = 7), 7.0 (4.0–11.1) (n = 4) and 7.2 (4.0–10.4) (n = 12) $\times 10^9\ l^{-1}$, respectively. Serum urea concentration was normal in them all. Three in group A had a mild increase ($<34\ \mu mol\ l^{-1}$) in serum bilirubin concentration; four had an elevated serum alanine transferase (three had ENL and the other hepatic amyloidosis), and four had an elevated alkaline phosphatase concentration. In group B, three had a mildly raised serum alanine transferase concentration. Aspiration liver biopsy-specimens were obtained from five in group A and four in group B. Three in group A demonstrated non-specific inflammation and focal accumulations of foamy phagocytes (which did not contain bacilli); in one, amyloid largely replaced the hepatic parenchyma; in another (who had tuberculosis), non-specific inflammation, a tuberculoid granuloma, and a few acid-fast fragments of bacilli were seen. In group B, three showed mild inflammation, a few small histiocytic foci and vacuolated phagocytes, but no bacilli; in another an hepato-cellular carcinoma was demonstrated.

Following an approximately 10 h overnight fast, 25 g xylose (Koch-Light Ltd., England or BDH Chemicals, England) dissolved in 500 ml water at room temperature was ingested. The patients sat throughout the tests. Venous blood samples taken at 60, 90 and 120 min after the mid-

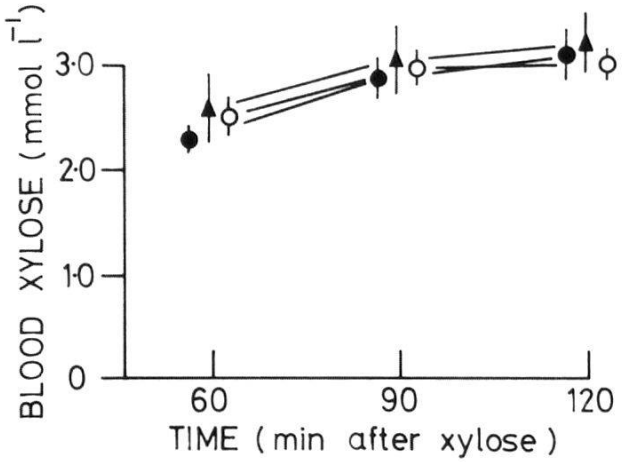


Fig. 1. Blood xylose concentrations after 25 g oral xylose. Means ± 1 SEM are shown for groups A (●), B (▲) and C (○). None of the differences between means is significant.

point of xylose ingestion were immediately added to and mixed with 2 vol zinc sulphate (18 g $l^{-1}$) +2 vol barium hydroxide (18 g $l^{-1}$). The supernatant, subsequent to centrifugation was frozen at −20° C until xylose estimation (Roe and Rice, 1948). All determinations were made in duplicate; separate standard curves were constructed at the beginning and end of each set of estimations. Serum total protein was estimated by the biuret method; electrophoresis utilized cellulose polyacetate membranes (Gelman Instrument Co., Michigan, USA).

## Results

Fig. 1 summarizes mean blood xylose concentrations at 60, 90 and 120 min in groups A, B and C; differences are not significant. Two patients in group A (one, who had ENL and amyloidosis, was treated with dapsone, clofazimine and chloroquine, and the other with dapsone and clofazimine) and one in group B (treated with dapsone) had a 90 min blood xylose concentration <2.0 mmol $l^{-1}$ (1.4, 1.8 and 1.9 mmol $l^{-1}$, respectively). Serum $\gamma$-globulin concentrations were 34, 34 and 30 g $l^{-1}$. There were no associations between xylose concentrations and therapeutic agents.

## Discussion

Functional integrity of the jejunum is not significantly compromised by leprosy in well-nourished PNG patients; although the control group was not altogether ideal, mean blood xylose concentration at 90 min was comparable with that in other controls in whom there was definitely no evidence of systemic infection (Cook, 1979). Involvement of the gastro-intestinal tract has been recorded in the armadillo (*Dasypus novemcinctus*) infected with *M. leprae* (Kirchheimer et al., 1972), but evidence of morphological change and impairment of absorptive function in the human jejunum is less impressive (Mitsuda and Ogawa, 1937; Kean and Childress, 1942; Powell and Swan, 1955; Desikan and Job, 1968; Bernard and Vazquez, 1973; Kasili et al., 1979), despite the fact

that leprosy affects many other organs. In 25 Indians with leprosy, an impairment of fat absorption in two (8%), and a reduction in 5 h urinary xylose excretion (after a 25 g oral load) associated with an abnormal Schilling test in three (12%) was reported (Kaur et al., 1978); seven (five of whom had no evidence of malabsorption) also had 'partial villous atrophy'. However, those abnormalities might have been within the normal range for residents of a tropical country. A high incidence of abnormal xylose and Schilling tests has been demonstrated in *malnourished* patients with leprosy (Noronha, 1971).

Impaired xylose absorption associated with acute and chronic systemic bacterial infections, in the absence of jejunal morphological change (as shown by light microscopy), has previously been demonstrated (Cook, 1972, 1974, 1980); that might be mediated by bacterial toxins, but that has not been proved. The explanation for *normal* xylose absorption in groups A and B in the present study is probably that *M. leprae* infections are too 'low grade' and insidious to produce functional changes of the enterocyte.

The 90 min blood xylose concentration is of proven value in assessing absorption of that monosaccharide in an environment where accurate collection of a 5 h urine sample is difficult or impossible (Cook, 1979).

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