

VITAMIN A DEFICIENCY AND GASTROINTESTINAL STRUCTURE AND FUNCTION

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While epidemiological evidence suggests a causative link between vitamin A deficiency and diarrhoeal disease morbidity and mortality in countries where both conditions are prevalent (Glasziou and Mackerras 1993), the physiological mechanism for this is unknown. The aim of this study was to delineate the effect of clinical and subclinical vitamin A deficiency on intestinal structure and function.

Four groups of weanling male SPF Wistar rats were fed for 60-63 days (clinical deficiency) or 40-42 days (subclinical deficiency): (1) a vitamin A deficient diet; (2) vitamin A deficient diet, pair-fed to animals in group 1, vitamin A (250IU) added to drinking water; (3) ad libitum vitamin A deficient diet, vitamin A (250IU) added to drinking water; (4) normal rat chow. Vitamin A deficiency was verified by serum and liver retinol measured by HPLC. Jejunal mucosal homogenate was assayed for sucrase activity, protein and DNA. Villous height was measured using a calibrated micrometer. 3-O-Methyl-D-glucose transport was measured in unstripped jejunem under short circuited conditions in Ussing Chambers.

Clinical deficiency data is presented in the Table as mean \pm SEM.

	Group 1	Group 2	Group 3	Group 4
Serum retinol (μ mol/l)	$<0.02 \pm 0^1$	1.81 ± 0.07	1.72 ± 0.16	2.08 ± 0.07
Liver retinol (μ g/g)	$<0.5 \pm 0^1$	51.2 ± 4.2	34.4 ± 3.0	117.5 ± 9.1^1
Final weight (g)	217.7 ± 10.5^2	260.3 ± 8.7^3	314.4 ± 10.8	358.1 ± 8.2
Sucrase (U/g protein)	63.0 ± 6.2^4	89.3 ± 4.0	60.3 ± 4.7^4	72.7 ± 2.6
Villous height (μ)	420.4 ± 11.3	481.0 ± 18.8	417.8 ± 29.6	458.17 ± 16.2
Glucose transport (μ mol/cm ² /hr)	0.6 ± 0.1	0.5 ± 0.1	0.4 ± 0.1	0.4 ± 0.1

¹P<0.001 compared to all other groups, ²P<0.05 compared to all other groups, ³P<0.05 compared to all other groups, ⁴P<0.01 compared to pair fed group

Subclinical deficiency, despite negligible serum and liver retinol, caused no apparent changes in parameters measured. Clinical vitamin A deficiency causes reduced body weight and sucrase activity. Glucose transport is preserved. The effect of vitamin A deficiency on intestinal brush border enzymes may contribute to the increased severity of diarrhoeal disease. Serum and liver retinol depletion precede clinical features of vitamin A deficiency.

GLASZIOU, P.P. and MACKERRAS, D.E.M. (1993). *Lancet* 306: 366.