Three decades of gastroenterology in Soweto South Africa: from descriptive to scientific observations
Segal, I.

Citation for published version (APA):
Segal, I. (2002). Three decades of gastroenterology in Soweto South Africa: from descriptive to scientific observations

General rights
It is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), other than for strictly personal, individual use, unless the work is under an open content license (like Creative Commons).

Disclaimer/Complaints regulations
If you believe that digital publication of certain material infringes any of your rights or (privacy) interests, please let the Library know, stating your reasons. In case of a legitimate complaint, the Library will make the material inaccessible and/or remove it from the website. Please Ask the Library: http://uba.uva.nl/en/contact, or a letter to: Library of the University of Amsterdam, Secretariat, Singel 425, 1012 WP Amsterdam, The Netherlands. You will be contacted as soon as possible.
Chapter 5

Does sucrase deficiency in Black South Africans protect against colonic disease?

Veitch AM, Kelly P, Segal I, Spies SK, Farthing MJG

Lancet 1998;351:183
Research letter

The spectrum of non-infectious gastrointestinal disease in black Africans is different from that amongst whites living in Africa or elsewhere. Colon cancer, colonic polyps, appendicitis, and inflammatory bowel disease are rare in black Africans.\(^1\) In the Soweto township of Johannesburg in South Africa, black Africans have been living in an urban environment for one or two generations. A more westernized lifestyle has been adopted compared with rural Africans, including the adoption of a relatively low-fibre diet, although differences in non-infectious gastrointestinal disease between blacks and whites have persisted. Increased faecal short chain fatty acids (SCFAs) have previously been shown in healthy black people in Soweto.\(^2\) We investigated whether there is a subclinical enteropathy in this population which may possibly result in malabsorption of disaccharides with subsequent colonic conversion to SCFAs.

We studied 23 black and 23 white patients attending Baragwanath and Johannesburg General Hospitals, Johannesburg, and HF Verwoerd Hospital, Pretoria, for routine upper gastrointestinal endoscopy for dyspepsia; those with diarrhoea, weight loss, systemic illness, HIV-1 infection, or gastric or duodenal ulceration were excluded. Duodenal villus height and crypt depth were measured by computerised morphometry. Lactase, sucrase and maltase activities in snap-frozen duodenal biopsy specimens were measured as described by Dahlqvist\(^3\) and Trinder\(^4\) modified for use in microtitre plates.

Mucosal lactase-specific activity (nmol glucose/min per mg protein) was lower in black patients (mean 0.2 [range 0-8.8] vs 25.5 [0-85.5], \(p=0.0002\)), as was sucrase (22.8 [0-73] vs 43.5 [1.7-122.91], \(p=0.008\)), than in white patients, but there was no difference in maltase activity (203.1 [0-285.3] vs 280 [13-619.3]) (figure).

\[\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure.png}
\caption{Duodenal disaccharidase activities in black (hatched bars) and white (open bars) South Africans. Results expressed as median (interquartile range). Specific activity: nmol glucose/min per mg protein.}
\end{figure}\]
There was no significant difference in body-mass index, haemoglobin mean cell volume, serum vitamin B\textsubscript{12}, red-cell folate, serum calcium, or albumin between black and white patients, and there was no difference in villus height (400 [IQR 235-456] vs 377 [317-449] μm), crypt depth (154 [119-188] vs 144 [133-170] μm), epithelial height (28 [22-36] vs 30 [24-310] μm), or density of lamina propria inflammatory cell infiltrate (185 [157-211] vs 180 [153-207] 0-25 mm\textsuperscript{2})

We have shown sucrase deficiency in black Africans. Lactase deficiency in black races in the small intestine is well recognised. The mechanism for relative sucrase deficiency in black people is unclear. There was no gross mucosal abnormality and maltase activity was preserved. In the absence of mucosal injury, failure of expression of disaccharidases may be regulated at the genetic or molecular level. The human sucrase-isomaltase gene has been cloned, and its expression found to be regulated at the mRNA level.\textsuperscript{5}

We suggest that sucrase malabsorption resulting in increased colonic SCFAs in the black South African population might be one of a number of protective mechanisms against non-infectious colonic disease in this population.