Acta Paediatr Scand 69: 687–688, 1980

LETTER TO THE EDITOR

Sir,

We read with interest (and some dismay) the paper by Solomons and associates—*Acta Paediatr Scand, 78:* 171, 1979. The authors present data to show that excess pulmonary excretion of hydrogen (H_2) in response to a 10 gram dose of the non-absorbable disaccharide lactulose, was significantly lowered in children with active gastroenteritis and diarrhoea when compared with non-diarrhoeal controls. They go on to state (fairly categorically) that the breath H_2 test cannot be recommended for measuring carbohydrate malabsorption in individuals with active, ongoing diarrhoea. Whilst we agree with their findings—and indeed have found a similar effect in our laboratory, we cannot allow their conclusions to go unchallenged for the following reasons:

1. On the basis of their own results: it should be easily possible to differentiate between carbohydrate malabsorption and normal absorption in the majority of cases. The H_2 rise, although lower in diarrhoeal patients, is still significant when carbohydrate is malabsorbed (the gas chromatography method of H_2 detection should measure down to 5 parts per million (p.p.m.)).

2. Three patients in the diarrhoeal group had been on antibiotics which could have been a factor in the reduced H_2 production of the total group. Were their H_2 rises the lowest?

3. Statistical analysis of the difference between means where $n=5$ for controls and $n=10$ for the diarrhoeal group may be misleading. Unpublished data from our laboratory using larger numbers of children verifies the lowering of the mean, but not median H_2 production in the diarrhoeal group, but the large standard deviation (S.D.) renders this difference in mean statistically non significant. (See table below.) The H_2 response was obtained following ingestion of 6.68 g lactulose in 22 healthy children and 13 children with diarrhoea in whom no antibiotics had been used.

	Normal group	Diarrhoea group
Mean H_2 ± S.D.	39.8 ± 44.8 p.p.m.	26.9 ± 24.9 p.p.m.
Range	0–190 p.p.m.	0–98
Median	26	23

We wish to stress that in our experience most patients with ongoing diarrhoea produced a substantial (albeit lowered) rise in breath H_2 when carbohydrate is malabsorbed. Possible false negatives are easily confirmed by using lactulose to test hydrogen production. The breath H_2 test is therefore *not* rendered inapplicable to the study of carbohydrate malabsorption in diarrhoeal disease—that group where this kind of testing is important.

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The Editor has asked Drs Solomons and Vitesi to comment on this letter.

Sir,

We are heartened that our publication (7) has stimulated interest in the evaluation of the H_2 breath test in active diarrheal disease and that Drs Robb and Davidson have substantially confirmed our observations. A research team in Bangladesh recently published the results of H_2 breath tests of lactose absorption in 331 rural villagers (4): they also noted a reduction of breath H_2 excretion during active diarrhea (K. H. Brown, personal communication). The phenomenon seems adequately confirmed.

It is refreshing to have data from other parts of the world, as our results reflect the reality of typical gastroenteritis as seen in Central America. Here, the patients usually present to

the hospital in advanced states of dehydration after having received a series of home and proprietary remedies, often including antibiotics. A more prompt presentation to the hospital of diarrheal cases with lesser alterations of bacterial floral mass, milder intestinal secretion rates, and less self-medication in Australia might possibly better explain the textural differences between the findings for the two regions than sample size, per se.

Clearly, if, as originally described (2), the H_2 breath test is applied in each individual, expressing the response to a test carbohydrate as a percentage of the response to a nonabsorbable carbohydrate, such as lactulose, i.e. using the patient as his or her own control, application of the H_2 breath test, even in active diarrhea might be valid. However, this would require the diarrheal patient to be in a "steady-state" condition during both of the H_2 breath tests! Usually, however, standard diagnostic criteria of a "normal" or "abnormal" H_2 response have been employed. The difference in response to lactulose between groups in the data of Drs Robb and Davison confirm our assertion (7) that other than the established diagnostic criteria would be required for evaluation of individuals with gastroenteritis.

Most importantly, however, this controversy invokes newer concepts of clinical decision-making theory; it is important to consider the *predictive accuracy* of a test, embodying the concepts of "specificity" and "sensitivity" or the ability of a diagnostic tool to avoid false-positive and false-negative diagnoses, respectively (5). Dr Ronald Barr, in a soon-to-be-published chapter (1), has applied the concepts of specificity and sensitivity to the use of the H_2 method as a screening procedure for carbohydrate malabsorption at the population level. In normal adults, the H_2 breath test has been found to be virtually 100% specific and sensitive for diagnosing lactase deficiency (3, 6). Dr Barr correctly points out, however, that any condition that systematically reduces the production of H_2 would decrease the sensitivity of the H_2 breath test, increase the number of false negatives, and lead to an *under*estimation of the prevalence of carbohydrate malabsorption; if the rate of malabsorption is high (as might be expected in a group of children with gastroenteritis), even small numbers of underproducers in a survey sample would greatly reduce the predictive accuracy of the breath test results and distort the estimation of malabsorbers for that population.

Considering all of these factors, we must reiterate our caution regarding the unreliability of the H_2 breath test for carbohydrate malabsorption in a population of individuals with active, ongoing, diarrheal episodes.

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