

H₂ BREATH TESTS DURING DIARRHEA

N. W. SOLOMONS, R. GARCÍA, R. SCHNEIDER, F. E. VITERI and
V. ARGUETA von KAENEL

*From the Division of Human Nutrition and Biology, Institute of Nutrition of Central America and Panama,
and the Department of Paediatrics, San Juan de Dios General Hospital,
Guatemala City, Guatemala*

ABSTRACT. Solomons, N. W., Garcia, R., Schneider, R., Viteri, F. and Argueta von Kaenel, V. (Division of Human Nutrition and Biology, Institute of Nutrition of Central America and Panama and Department of Pediatrics, San Juan de Dios General Hospital, Guatemala City, Guatemala). H₂ breath test during diarrhea. *Acta Paediatr Scand*, 68: 171, 1979.—The peak rise in breath hydrogen and the volume of excess pulmonary excretion of hydrogen in response to a 10 g dose of the non-absorbable disaccharide, lactulose, was significantly lower in children with active gastroenteritis and diarrhea than in non-diarrheal controls. Thus, despite the fact that the H₂ breath test is a convenient, non-invasive technology for use in children, it cannot be recommended for measuring carbohydrate malabsorption in individuals with active, on-going episodes of diarrhea.

KEY WORDS: Hydrogen breath test, carbohydrate malabsorption, infantile diarrhea, lactulose

When ingested carbohydrate is exposed to certain colonic bacteria, fermentation results in the intrainestinal production of hydrogen (H₂); some of this H₂ is excreted by the lungs. Since a linear and relatively constant relationship between the amount of non-absorbed carbohydrate and the rate of H₂ excretion has been demonstrated (1), clinical carbohydrate malabsorption tests based on breath analysis have been developed. The innocuous, non-invasive nature of the collection procedure as compared with other techniques for quantifying carbohydrate malabsorption has encouraged application of the H₂ methodology in young children (2, 4, 6).

We have been interested in determining the amount of nutrient loss which can occur as the result of acute infantile gastroenteritis, and felt that the H₂ breath test would be useful in the evaluation of this problem. However, our initial experience with administering a 15 g oral glucose dose to infants with intractable diarrhea and clinical monosaccharide intolerance or with giving 1.75 g of lactose per kg to children with acute enteritis failed to show the expected rise in breath H₂ where

large amounts of fecal reducing substances could be detected in the stools (Solomons, García and Viteri, unpublished). Moreover, in some institutionalized children followed prospectively, a drastic reduction in the usual rate of pulmonary H₂ elimination on a normal diet accompanied episodes of diarrhea (Schneider, unpublished). These curious results led us to examine the question of whether or not the presence of diarrhea, itself, was affecting the pulmonary excretion of H₂.

PATIENTS AND METHODS

Five pre-school aged children without diarrhea in the Clinical Research Center of the Division of Human Nutrition and Biology, Institute of Nutrition of Central America and Panama served as controls. Ten children aged 6 months to 3 years selected at random from among patients admitted to the Rehydration Unit of the San Juan de Dios General Hospital with acute gastroenteritis, had ongoing, active diarrhea. Only 3 of the latter subjects had received antibiotics prior to study. All subjects received 10 g of the non-absorbable disaccharide, lactulose. Samples of expired air were collected before and at 30-min intervals following the administration of the lactulose dose for a total of 4 hours and the breath H₂ concentration was measured by gas chromatography as previously described (5). The pulmonary excretion of

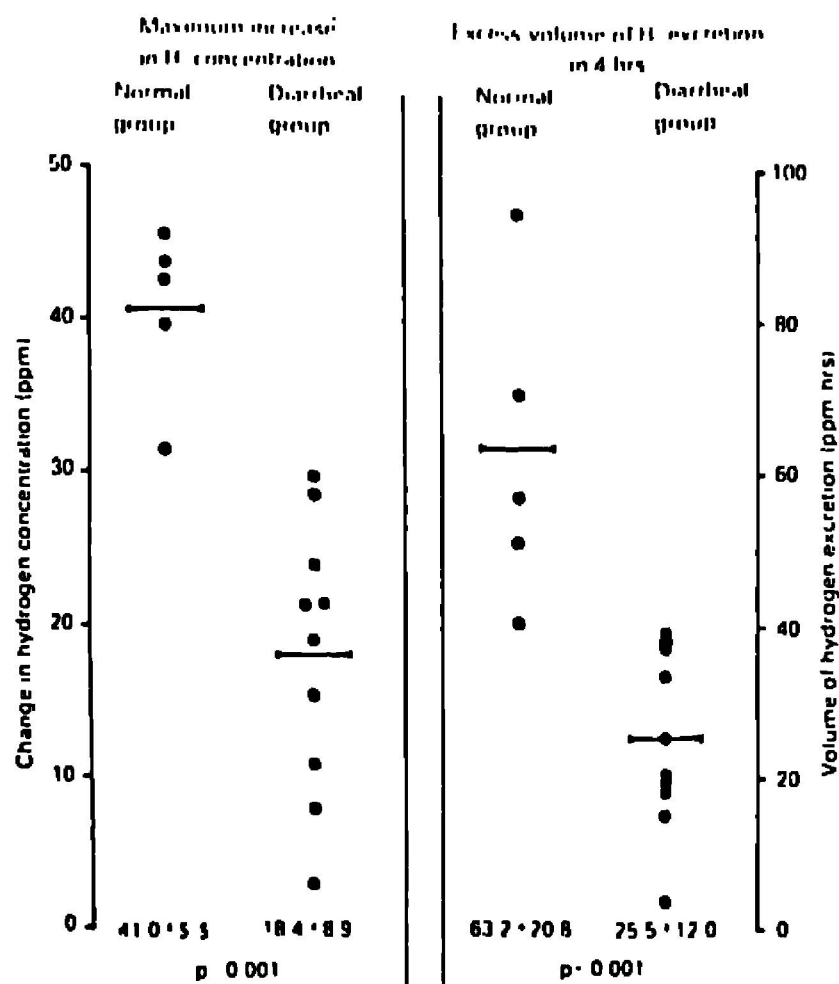


Fig. 1. Scattergram illustrating the breath hydrogen response to ingestion of 10 g of lactulose in healthy children and in children with diarrhea. Hydrogen excretion has been quantified both as the maximum increase in H₂ concentration in ppm, and as the excess volume of H₂ in ppm·hrs over 4 hours.

H₂ was compared in two ways: 1) as the maximum increase in H₂ concentration in ppm, and 2) as the volume of H₂ eliminated during the 4-hour period in ppm·hrs.

RESULTS AND DISCUSSION

As shown in Fig. 1, a significant reduction ($p < 0.001$) in H₂ excretion to the same 10 g dose of lactulose was seen among children with acute diarrhea as compared with normal control children. The explanation is clear but several mechanisms could be involved. Firstly, quantitative or qualitative changes in fecal flora during diarrhea could reduce the critical mass of appropriate bacteria necessary for fermentation of the carbohydrate substrate. Alternatively, changes in colonic motility during active diarrhea could change the partition of colonic H₂ excretion between breath and flatus with a proportionately greater amount of intestinal H₂ being excreted by rectum. We and others previously shown that oral antibiotics can interfere with the bacterial metabolism and H₂ excretion from carbo-

hydrates (3, 5), but in only 3 of the 10 subjects was prior use of antibiotics a factor.

The H₂ breath test can be a valuable and reliable technology for the study of carbohydrate malabsorption. Its non-invasive nature makes it particularly suited to studies in young children. The important conclusion of the present study is that standard H₂ breath test criteria for carbohydrate malabsorption based on normal subjects cannot be reliably applied to individuals with active gastroenteritis. This would seem to render the H₂ breath-analysis techniques inapplicable to the study of carbohydrate absorption in diarrheal disease.

ACKNOWLEDGEMENTS

The authors would like to thank the nursing staff of the Clinical Research Center of INCAP and of the Pediatric Emergency Service of the San Juan de Dios General Hospital. Dr Solomons was a recipient of the Josiah Macy Jr Foundation Faculty Fellowship and of the Future Leaders Award of the Nutrition Foundation.

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Submitted June 21, 1978

Accepted Aug. 28, 1978

(N. W. S.) Division of Human Nutrition and Biology
INCAP
Carretera Roosevelt, Zona 11
Guatemala, C.A. AP 11-88
INCAP publication I-1010