be representative for the general inflammatory state. Since NO in gas will be more evenly distributed, this method may correlate better with disease, i.e. the area of inflamed mucosa together with the degree of inflammation in affected areas. The lower levels of NO in Crohn's disease may be an example of this, since normally a larger area of the epithelial lining is affected in the ulcerative colitis compared to Crohn's disease. Both direct measurements of NO in the intact colonic lumen and indirect measurements of NO-synthase activity in whole mucosa (Middleton et al., Lancet 341 (1993) 465–66; and Boughton-Smith et al., Lancet 342 (1993) 338–40) indicate that NO production is much larger in patients with inflammatory conditions in the intestine than in controls.

However, the difference between NO production in the two ulcerative colitis patients appeared much larger when the direct luminal method was used (100 times compared to 4–8 times). Furthermore, no increased NO-synthase activity was seen by the indirect method in patients with Crohn's disease, whereas our direct method showed approximately 20 times increased levels of luminal NO. When measuring NO production in whole mucosa using the indirect method, normal NO production from all layers of the mucosa will contribute, thus increasing background levels and reducing the differences between the groups. Luminal NO measurements, on the other hand may reflect NO production only in the most superficial parts of the mucosa, as NO produced in the deeper mucosal structures will be trapped by e.g. haemoglobin in mucosal blood vessels and therefore will not reach the lumen. Consequently, this suggests that the NO production detected in luminal gas in inflammatory conditions is mainly located to very superficial mucosal layers. Thus NO gas measurements in the intestines may be a sensitive measure of inflammation, since NO production should not take place at all in the normal epithelium.

Active ulcerative colitis is associated with an increase in the activity of inducible NO synthase. This apparently does not fit with the classical view of inducible NO synthase as being steroid sensitive (Moncada et al., Pharmacol. Rev. 43 (1991) 109–141), since in our study and in an earlier study (Boughton-Smith et al., Lancet 342 (1993) 338–40), patients treated with corticosteroids did not differ from untreated as far as NO production was concerned. Whether this reflects an incomplete penetration of steroids to NO-producing cells in the mucosa or the presence of a steroid resistant inducible NO synthase remains to be studied. In our study, salicylates did not seem to interfere with NO production either. However, it should be pointed out that all patients on treatment with salicylates suffered from an acute exacerbation of ulcerative colitis despite ongoing treatment. Thus, we cannot tell whether salicylates interfere with NO synthesis in other ulcerative colitis patients where treatment is more successful and no activation of colitis occurs. Since the treatment have apparently failed in these patients with acute symptoms and high NO levels, measurements of intestinal NO may be useful in the testing of new drugs.

It still remains unclear whether an increased NO production seen in patients with an inflammatory condition in the intestine is beneficial or harmful for the tissue. NO or subsequent reactive products may have cytotoxic actions against host cells when produced in excess (Tepperman et al., Am. J. Physiol. 265 (1993) G214–G218). On the other hand, large concentrations of luminal NO are normally present in e.g. the nasal airways (Lundberg et al., Eur. J. Resp. 7 (1994) 1501–1504) and the stomach (Lundberg et al., Gut 35 (1994) 1543–1546) without causing local tissue damage. Moreover, NO has been suggested to play an important role in host defence mechanisms, e.g. by its bacteriostatic properties (Moncada et al., Pharmacol. Rev. 43 (1991) 109–141). This is supported by the previous finding that patients with active ulcerative colitis exhibit a reduced number of bacteria in the rectal mucosa compared to patients with inactive disease and to controls (Hartley et al., J. Medical. Microbiol. 3.6 (1992) 96–103.

What is claimed is:

1. A method for diagnosing food intolerance in a human, comprising the steps of:

obtaining a gas sample from the lumen of the intestines of the human;

measuring the level of nitric oxide in the gas sample;

comparing the measured level with the expected level for a healthy human or with a prior level measured in the human; and

diagnosing the presence or absence of food intolerance using the results of said comparison.

2. The method according to claim **1**, wherein the sample is taken from the lumen of the rectum.

3. The method according to claim **1** or **2**, wherein an increased level of nitric oxide indicates coeliac disease.

4. The method according to claim **1** or **2**, wherein an increased level of nitric oxide indicates food allergy.

5. The method according to claim **1** or **2**, wherein an increased level of nitric oxide indicates lactose intolerance.

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