

Gut

Leading article

Pioneer in the gluten free diet: Willem-Karel Dicke 1905–1962, over 50 years of gluten free diet

The purpose of this article is to commemorate the death of Willem-Karel Dicke 31 years ago and his first publication on gluten free diet in 1941 (Fig 1).

Dicke, born 1905 in Dordrecht, the Netherlands, became the medical director of the Juliana Children's Hospital in The Hague at the age of 31. J W Stoop, one of his disciples, wrote about him 'Dicke was an outstanding clinician, scientist and manager with exceptional personal qualities. His intuition, subtle approach, analytical capacities and broad clinical knowledge made him a brilliant clinician'.¹ We will provide evidence that these qualities and critical observations led him to the conclusion that wheat products contained the factor responsible for the severe clinical symptoms of coeliac disease, at that time also called Gee-Herter's disease, long before his thesis in 1950.

In the late '20s and early '30s it was generally agreed that the two main treatment principles in coeliac disease were rest and diet. The most important study in this period was published in 1924 when Sidney Haas² described his treatment of coeliac disease. He reported 10 cases, of whom he claimed that the eight treated by a banana diet were clinically cured whereas the two untreated had died. From that time there is hardly any form of diet that has not been recommended, such as carbohydrate diet (fruit, purée of potatoes or tomatoes), beef steak cure, milk diet (2–2.5 l/day). Even as early as 1887, however, in the former Dutch East Indies (at present Indonesia), VandeBurg advised a straight fruit diet. These principles of dietary treatment were frequently discussed during the meetings of paediatricians at that time. Visiting such a meeting in 1932 Dicke's attention was directed to a case report presented by Stheeman³ concerning relapses of diarrhoea after the consumption of bread and rusks; according to the testimony of one patient.

How do we know that Dicke was convinced of the beneficial effect of wheat free diet even before 1940? From the testimony of Dicke's wife in 1991, we learned that already between 1934 and 1936 he had started his experiments with wheat free diets. Also from Booth's conversation with Dicke it can be concluded that Dicke knew long before World War II that wheat products contained the offending agent; 'It was a young mother's statement of her coeliac child's rash improving rapidly if she removed bread from the diet, that alerted his interest, when he was a paediatrician in The Hague in 1936.'⁴

At the end of World War II, during the 1944/45 'Winter of starvation', the delivery of normal food such as bread to



Figure 1: Photograph of Dicke in his time as Director of the Wilhelmina Children's Hospital, Utrecht.

his young patients in his hospital was endangered. This period convinced him even more that merely eating less cereals and more uncommon food products, such as tulip bulbs, improved the clinical condition of his patients.

Dicke's first report about the wheat free diet was published in *Het Nederlands Tijdschrift voor Geneeskunde* in 1941 (W K Dicke: A simple diet for Gee-Herter's Syndrome).⁵ '... In recent literature it is stated that the diet of Haas (Banana-diet) and Fanconi (fruit and vegetables) gives the best results in the treatment of patients suffering from coeliac disease. At present (World War II) these items are not available.

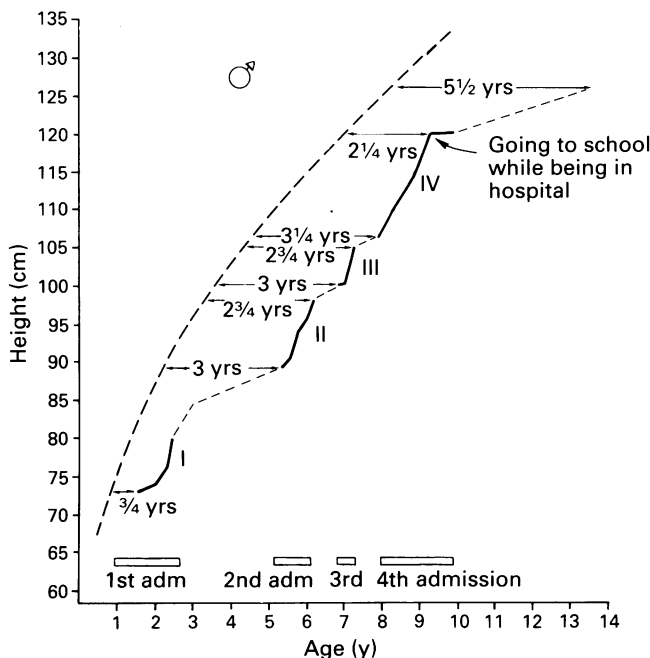


Figure 2: Growth curve of boy GH (born 10-06-35) (shown as interrupted straight line), by comparison with ideal growth curve (shown as dotted line). Hospital admissions are shown by roman numerals with the retardation in length in years. Under a strict wheat free diet in the hospital he is gaining weight and, with each admission, approaches his normal growth curve. (Reproduced and translated from Dicke's thesis with permission.)

Therefore, I give a simple diet, which is helping these children at this time of rationing. The diet should not contain any bread or rusks. A hot meal twice a day is also well tolerated. The third meal can be sweet or sour porridge (without any wheat flour).⁷

Dicke's other written reference is his thesis.⁶ In this thesis he describes a meticulous dietary study over a period of several years at the Juliana Children's Hospital in a patient with coeliac disease, which started in 1936! A strict regimen of wheat free diet in the hospital had a favourable and normalising effect on his clinical symptoms, weight, and growth. Each time the boy went home and the wheat free diet could not be sustained he suffered a decline in his growth curve. During four longterm admissions each time the trend towards normal growth could be restored (Fig 2). In his other examples of the cases described, he has chosen on purpose older children with longstanding histories of suffering, thus removing any doubt about the correct diagnosis. He excluded patients with cystic fibrosis by performing duodenal drainage tests for the measurements of pancreatic enzyme concentrations in the duodenal juice. Faecal tests to exclude parasitic diseases and small intestinal roentgenograms were also performed.

In his thesis several growth curves of children treated with a wheat free diet are presented. In longterm studies over several years he shows that with a wheat free diet these children are gaining weight and are reaching a normal growth pattern by comparison with age matched controls. At the end of chapter 3 of his thesis he concludes that:

- 'if certain types of meal, such as wheat and rye are replaced in the daily diet, the patient improves;
- acute attacks of diarrhoea, do not occur, provided these types of meal are not given;
- after a latent period which can vary in length, deterioration and acute attacks of diarrhoea re-occur, if the objectionable types of meal are added to the diet too soon. . . .'

Considering Dicke's thesis⁴ as well as van de Kamer's publication in 1952⁷ it is quite clear that when Dicke went to

Utrecht to perform his classic dietary fat absorption experiments, he had already been convinced for years that wheat, rye, and oat meal products were the offending agents in the cause of coeliac disease.

It was fortunate that after World War II Dicke started a close co-operation with J H van de Kamer, a biochemist from the Netherlands Central Institute for Nutritional Research TNO in Utrecht, who was the first to develop an accurate and easily available method to measure faecal fat content in wet faeces.⁸ Also in collaboration with H A Weyers, a paediatrician from the Wilhelmina Children's Hospital in Utrecht, a method was developed that permitted the analysis of faecal fat excretion in children with coeliac disease by analysing the correct coefficient of fat absorption.^{6,9} These experiments are also described in chapter 4 of his thesis.

Dicke decided to take as the measure for fat digestion, the fat absorption coefficient, where the coefficient of fat absorption/24 hr is:

$$\frac{\text{total g fat in diet} - \text{total g fat excreted in faeces} \times 100\%}{\text{total g fat in diet}}$$

As the normal range they took values higher than 95%.

The crucial experiments with standardised diets, each time excluding or adding wheat or rye flour over long periods in these children with coeliac disease, were performed on four children in the Wilhelmina Children's Hospital in Utrecht and in one child in the Juliana Children's Hospital in The Hague. The children were challenged with different cereals under a strict dietary protocol with measurement of total faecal output, faecal fat content, and the fat absorption coefficient. Figure 3 shows one example of such a study. Based on these findings Dicke concluded in 1950 in his thesis that wheat flour but not well purified wheat starch (*amylum*) and also rye flour were the causes of the anorexia, the increased faecal output, and the streatorrhoea seen in these patients. The development of the gluten free diet was based on these discoveries. Together with van de Kamer and Weyers,¹⁰ he subsequently published that the alcohol soluble or the gliadin component of the water insoluble protein or gluten moiety of wheat, was responsible for the fat malabsorption in patients with coeliac disease. Although these findings were rapidly confirmed by investigators from Britain,¹¹ Scandinavia,¹² and Germany¹³ some doubt persisted, especially in American publications, concerning the efficacy of a gluten free diet.¹⁴⁻¹⁶ After the establishment of the intestinal biopsy technique for the diagnosis of coeliac disease, it became apparent that a wheat free diet should be maintained for long periods before an adequate response occurred, as Dicke had predicted.

Although it is well known nowadays that, in addition to the noxious cereal grains, genetic factors may predispose susceptible subjects to an immune response that damages the small intestinal mucosa and that other environmental factors may trigger this abnormal immune response,¹⁷ it was Dicke's astute observations some 50 years ago that first changed the prognosis and treatment of these patients dramatically.

It was to honour Dicke that the Dutch Society for Gastroenterology instituted the Dicke medal to reward pioneering work in hepatogastroenterology and, naturally, the first gold medal was awarded to Dicke himself.

In recognition of Dicke's outstanding clinical and scientific capacities he was appointed to the Chair of Professor of Pediatrics at the University of Utrecht and he became Medical Director of the Wilhelmina Children's Hospital in 1957.

He sadly passed away prematurely in 1962 because of severe cerebrovascular disease.

It is fortunate that his widow Mrs A Dicke-Schouten, surviving him until February 1992, could witness the revival of a broad international recognition of his pioneering work.

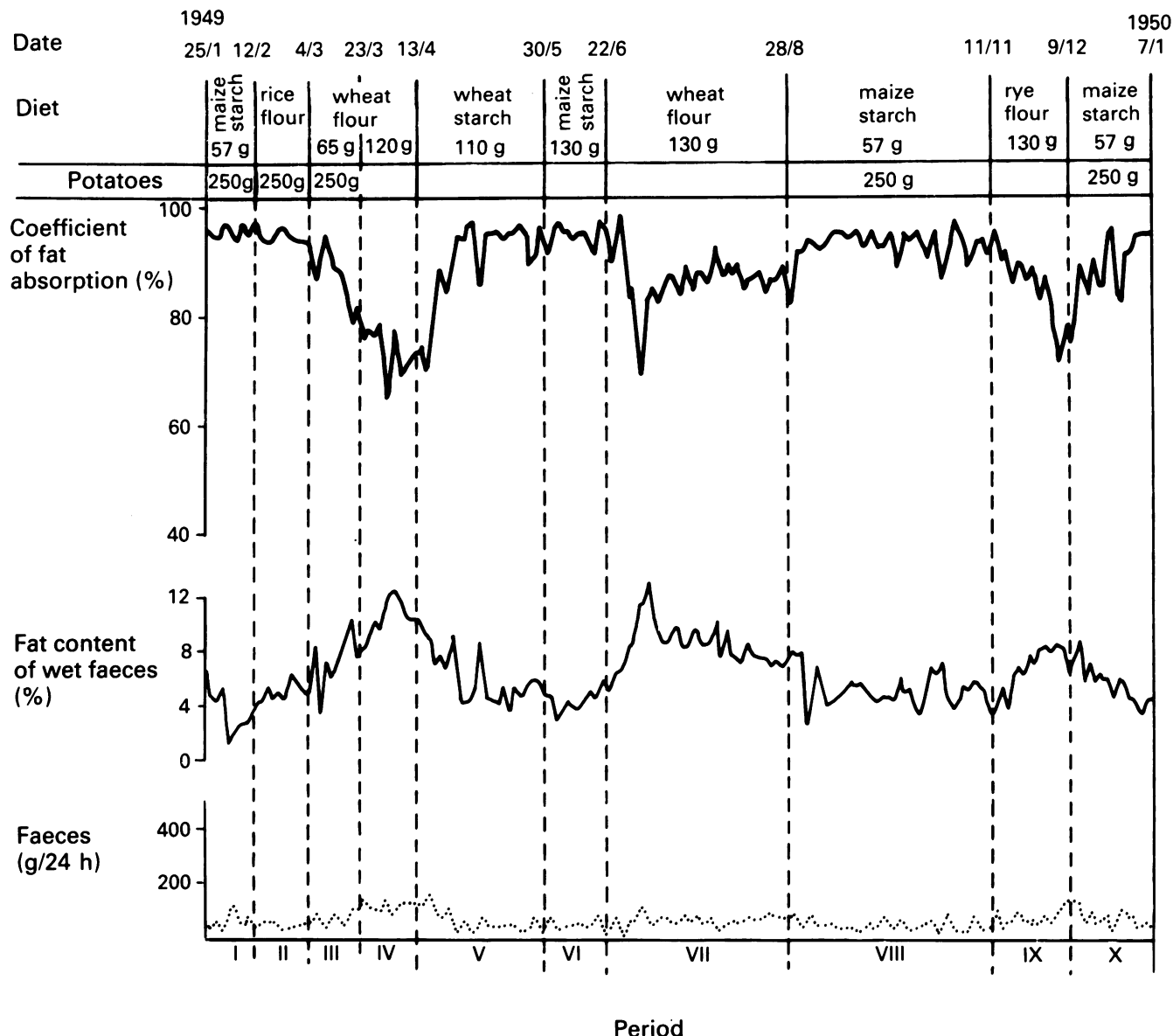


Figure 3: Patient CK is one of five classic examples of dietary fat balance studies in coeliac disease patients presented in Dicke’s thesis in 1950. Note the deleterious effect on fat absorption and faecal weight of wheat and rye flour. By contrast wheat starch is not causing such an effect. (Reproduced and translated from Dicke’s thesis with permission.)

She also provided additional information concerning the development of Dicke’s dietary hypothesis.

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