

Food colourings and preservatives—allergy and hyperactivity

Hyperactivity affects about 10% of children. The most frequent cause is attention-deficit hyperactivity disorder (ADHD). Several factors have been suggested to aggravate the disorder, including food components such as phosphates, glucose, lactose, preservatives, and dyes. A study in the 1970s led to the introduction of the Feingold diet, named after the investigator.¹ Since then, several conflicting findings have been reported.²⁻⁴

Belinda Bateman and co-workers⁵ recently observed a reduction of hyperactivity in 3-year-old children on a diet without artificial colourings or benzoate preservative, and an increase in hyperactivity in these children after active and also placebo challenge. The investigation included a cohort of children randomly divided into four groups, depending on the presence or absence of either hyperactivity or atopy. The four groups were assessed over a 4-week period: week 1=avoidance diet, week 2=placebo or active challenge, week 3=washout, week 4=active or placebo challenge. Assessment was done double-blind with questionnaires (mean daily parental behaviour rating) and with weekly tests done by a psychologist.

In Bateman and colleagues' study,⁵ the term hyperactivity seems to be used as synonymous to ADHD. A diagnosis of ADHD is tentative in a child aged 3 years. Diagnostic criteria and psychological tests are being validated for children older than 6 years.⁶ ADHD and hyperactivity due to other causes vary from day to day and from hour to hour depending on several factors. Therefore a 1-week observation period seems short.

Although this study⁵ initially included a general population sample of 2878 families, it ended up—after exclusion of non-responders, those who refused behavioural screening, those who refused skin-prick tests, non-selected patients, and dropouts—with a sample of only 277 children, less than 10% of the initial figure. What were the exclusion criteria? That 40% of these children were hyperactive (in a general population, this group does not exceed 10%) suggests a marked selection bias: families interested in hyperactivity seem to be over-represented. Thus results from this study should not lead to recommendations for the general population.

The results of Bateman and colleagues' study⁵ are surprising. Indeed, hyperactivity decreased in children on a diet without artificial food colourings and benzoate preservatives, and increased following re-introduction. The effect was reported only by the parents, whereas no difference was seen based on objective psychological testing. Although the authors claim that their results are sufficiently strong to suggest a benefit from the diet, we question this statement. Parents reported improvement during both placebo and active challenge, independently of their order, and heightened hyperactivity during the

washout week. However, the parents' observations can be easily explained by their expectations, which could lead to a biased assessment. The small difference between the active and placebo weeks, in favour of active challenge, is, in our opinion, not sufficient to recommend dietary treatment. Bateman and colleagues' study was not designed to explore a possible food allergy as a cause of hyperactivity. As the authors state, the results only show that atopic children are not over-represented in the group of children benefiting from the diet.

Most child neurologists will still not recommend the Feingold diet for their patients, but will not object to parents willing to try the diet. Allergologists will caution the fact that avoidance diets might also lead to disruption in daily life.⁷

In practice, children suspected of hyperactivity should undergo a careful initial clinical assessment including standard tests. We do not believe that routine testing should include screening for food colouring and preservatives as causative factors for hyperactivity. If the parents raise the question, a diet limited in time—eg, for 3 or 4 weeks—might be instituted, and its effect carefully assessed by the managing physician. Parents should also be questioned about the disruption of the diet on the child's daily life. If parents and health professionals decide to continue the diet after the trial period, repeated assessments—eg, every 6 months—should be done as no data for the duration of such a diet are available so far. We

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strongly believe that unnecessary diets should not be instituted for hyperactivity.

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Cancer and psychosocial distress: frequent companions

Recently, L Carlson and colleagues¹ reported on the high occurrence of psychosocial distress in cancer patients. They did a cross-sectional study with questionnaires in a tertiary hospital setting. Although this study was done in Canada, similar results would probably be obtained in most countries in the western world. All cancer patients older than 18 years who visited the hospital over a 1-month period for any reason were eligible for study. 90% of these patients (n=2776) completed the questionnaires. Most of these patients had breast, prostate, colorectal, or lung cancer. Based on the brief symptom inventory-18 (BSI-18) scores, more than 37% of patients were judged to be in serious psychological distress. Overall, somatisation was most common, followed by depression and anxiety. Psychological distress was more typical in younger patients, women, and those from ethnic minorities, from households with lower income, and those with a longer duration of illness. About 30% of patients were unaware of the possibilities for

psychosocial support; 44% of patients who refused psychosocial support did not feel the need for any help.

What can be learnt from these results? The percentage of patients who met the criteria for general distress was much higher than expected. Most previous studies have focused on one of the four periods of the illness trajectory—ie, the diagnostic and pretreatment phase, the treatment phase, the post-treatment phase, and the terminal phase. However, because the study was cross-sectional, one should conclude that psychosocial distress was imminent throughout the whole disease trajectory. Why do oncologists miss the diagnosis of psychological distress so often? Is it lack of interest, of knowledge, or of time during busy daily practice? The answer to these questions cannot be easily given. All three reasons probably have a role.

What can be done to overcome the underestimation of psychological distress in cancer patients? An easy method to screen for distress is the BSI-18 questionnaire. Patients could be asked to fill in this questionnaire at appropriate times during the disease process and follow-up. The BSI-18 is designed to measure psychological distress and is highly reliable.² The checklist yields three subscale scores: somatisation, depression, and anxiety, with an internal consistency ranging from 0.78 to 0.89. The questionnaire takes 1–3 min to complete and a nurse could collect the data.

Another striking finding of Carlson and colleagues' study was the fact that almost half of all patients who met the distress criteria had not sought professional psychosocial support, nor did they intend to do so in the future. The patients who did not use the psychosocial service were asked why they did not use it. The main reasons were the perception of not needing help (44%), unfamiliarity with the services provided (19%), and doubt whether the services would be of benefit (8%). In our opinion, another doctor-related reason should also be mentioned in addition to the lack of trust in the effectiveness of such strategies: unawareness of the psychosocial treatment strategies available.

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