

Annotations

Diet and behaviour

Behavioural toxicology has proved to be a difficult science. There are major methodological obstacles in the path of those trying to discover whether environmental substances such as lead or constituents of the diet such as food additives can damage the course of normal psychological development. These obstacles together with the effects of intense public interest, account for the persistence of uncertainty about the best advice to give the parents of children with disorders of conduct and learning.

Effects of eliminating food additives

Three recent, full reviews of the scientific studies have come to different conclusions about Feingold's hypothesis that hyperactivity in childhood results from an idiosyncratic reaction to artificial colourings (and some preservatives and salicylates) in food.¹ One review concludes that the hypothesis is essentially correct,² one that it is unsupported,³ while a Consensus development conference of the National Institutes of Health⁴ concluded that the evidence was inadequate and recommended a wide ranging programme of research. There is in fact more agreement between these studies than the controversy might lead one to suppose and several lines of evidence suggest that the Feingold diet is occasionally valuable but usually a placebo.

Open trials, as always, showed the power of an uncontrolled and new treatment to earn 'high ratings': they no longer contribute to the debate. Two blind comparisons of the modified Feingold diet with a control diet did not produce dramatic results.^{5, 6} One showed a statistical superiority for the exclusion of additives,⁵ the other did not.⁶ In both, however, there was an interaction between order of treatment and its success, with the Feingold diet being superior only when it followed the control diet, and a good response was confined to a minority of children.

Double blind challenge studies in which groups of children on the exclusion diet are given additives or 'placebo' were the next logical step. The most relevant are those which have been carried out on children already defined as 'diet responders'.^{3, 7–9} These studies showed that, as a group, even apparent responders did not deteriorate, or at worst

showed a fleeting and small worsening of behaviour, when the additives were reintroduced.⁸ Other challenge studies, carried out on children defined by their hyperactivity rather than their response to diet, showed no response to tartrazine,¹⁰ no behavioural response but a possible cognitive change to large doses of additives,¹¹ or a small behavioural response.¹² There was no change in study of an institutionalised, mildly retarded group of children,¹³ and a study of an 'allergy vaccine'¹⁴ seemed to suggest that children's behaviour could in some circumstances actually be improved by allergens compared with placebo.

These predominantly negative results came from group studies which might have missed changes in small numbers of children. Single case studies suggest that exactly this has happened—a few children do behave much worse with additives than with 'placebo'^{15–17} but the mechanism is unknown. Future research in this area needs to characterise these children further and describe the mode of action—not merely to multiply the number of group trials.

Effects of other defined diets

One obvious reason for the small number of genuine responders could be that the others are intolerant of different components of the diet. This is, however, an unlikely explanation for the challenge studies on people who have responded to the diet, since these studies, by definition, involved the replacement of what was eliminated. The lesson from the relatively well studied Feingold diet has to be that placebo effects are likely to be strong and must be fully taken into account before a diet can be scientifically justified. In general, this has not yet been done. There is some evidence¹⁸ that hyperactive children have more signs of allergies to a wide range of foodstuffs than normal children, and that removing allergens leads to small but real improvements in behaviour, but this kind of individualised approach warrants further trials. Furthermore, not too much should be made of any specific effect on 'hyperactivity'. This is (in American psychiatry) a common and heterogeneous set of behaviours, often associated with other kinds of conduct problem. The assess-

ment of behaviour change should be individual for each child's problems, not confined to attention deficit or locomotor excess. If this treatment approach proves to be justified, it will be by the presence of allergy rather than hyperactivity.

Refined sugar in the diet, vitamin and mineral deficiencies, and functional deficiencies of essential fatty acids requiring gammalinolenic acid supplementation all figure prominently in the fringe medical writings. So far their importance rests on confident assertion only, not evidence. (There is one exception—a survey¹⁹ has suggested that mildly hyperactive children eat less sugar, but severely hyperactive the same amount of sugar as controls. Uncontrolled factors such as drug treatment could, however, account for this finding, which is in any case hard to interpret as a cause of disorder.) Nevertheless, a normal diet contains psychotropic agents. Soft drinks, as well as tea and coffee, contain caffeine and children can take enough to produce the effects of motor restlessness and possible irritability.²⁰ A high intake needs to be borne in mind as a possible contribution to difficult behaviour.

Should the diets be prescribed?

Clinicians will interpret the results on the Feingold diet in different ways. Some will argue that it does no physical harm, so why not prescribe it for the sake of the usual placebo and the occasional idiosyncratic effects? Others will see it as charlatanism to prescribe such a regimen in the knowledge that it will not usually help. Two points need to be borne in mind. Firstly, it does little good to quarrel with parents who are likely to be better supported by a sceptical but interested paediatrician than by private clinics that can deliver no other treatment. Secondly, dietary treatment can sometimes become an overvalued idea, committing children to a treadmill of successively more rigorous diets while psychological help is neglected. It seems to follow that useful support to parents will include openness about the likely lack of value of dietary treatment according to present knowledge, willingness to help evaluate the effects of diet for families who nevertheless embark upon it, and keeping open the approaches to psychological treatments.

One advantage in the involvement of the media and campaigning groups is that this may lead to the labelling of substances added to food during manufacture. A disadvantage is that it could divert clinical attention and research funding disproportionately to this one aspect, at the ex-

pense of more promising lines of approach to helping psychologically troubled children.

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