

Review Article

The role of diet and behaviour in childhood

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Abstract: This short review summarizes the most important research, particularly that from 1985 to 1995, on the relationship between diet and behaviour. Relevant studies particularly those using double-blind placebo controlled food challenge methodology were selected, and are presented within a historical context. Summary tables of the early development of concepts and later pertinent studies are provided. The research has shown that diet definitely affects some children. Rather than becoming simpler the issue has become demonstrably more complex. The range of suspect food items has broadened, and some non-food items are relevant. Symptoms which may change include those seen in attention deficit disorder (ADD) and attention deficit hyperactivity disorder (ADHD), sleep problems and physical symptoms, with later research emphasizing particularly changes in mood. The reports also show the range of individual differences both in the food substances producing reactions and in the areas of change.

Key words: ADD; ADHD; diet; hyperactivity; irritability.

ROLE OF DIET IN BEHAVIOUR IN CHILDREN

The often quoted position that 'the relationship between diet and hyperactivity has not been proven' was based on research in the 1970s. It is now timely to consider the useful studies of the 1980s and 1990s that clearly show a relationship. The early research was useful as it clarified that small amounts of artificial colour did not cause hyperactivity. This resolved the public health issue: added colour did not need to be banned; but it did not resolve the question completely as some children were seen to react.^{3–5}

HISTORICAL CONTEXT

It is important to realize that the conceptualization of the issue changed greatly from 1975 to 1985. The initial debate was all the more controversial at a time of strong anti-technology feeling in the community. Feingold¹ had reported that a small amount of any one suspect chemical would produce significant change in all sensitive children. The early research disproved his claim. But as this research and early clinical reports provided additional information, workers changed aspects of the methodology and through their findings new information was added to the total picture. The most relevant information which contributed to change up to the mid-1980s is summarized in Table 1. Until the late 1970s samples usually comprised children diagnosed as attention deficit hyperactivity disorder (ADHD) at tertiary referral centres with no history of diet as suspect. By the 1980s recognition of the complexity of the issues was reflected in methods that investigated more suspect substances and monitored a wider range of areas of change.

The broader hypotheses posed the question: do normal amounts of different chemicals or common whole food allergens (together or separately) affect some children's behaviour or physical symptoms?

RESEARCH FROM 1985 TO 1995

Table 2 provides a summary of relevant research from 1985 to 1995. Most workers used double-blind placebo-controlled food challenges (DBPCFC) or repeated measures methodology, with statistical evaluation. While the connection cannot be said to be simple or neat, these studies have definitely demonstrated a role for diet in behaviour in some children.

SAMPLE SELECTION AND STUDY DESIGN

Later research provided clearer results because of improved methodology. Changes began with sample selection. Subjects were usually those with ADHD but some had other criteria specifically noted; whether the parents thought the child reacted to food, physical symptoms, or settling and sleep problems. In most research studies the presence of atopy was not considered until 1985.²⁶ One report tabulates 11 studies up to 1988 in which there was no evaluation of allergy in the sample.³⁸ An Australian study found a history of allergic symptoms in 68% although they had not been expected to lead to health problems.³⁴ The numbers studied in the recent studies collectively are significant. Diet establishment time was considered and the period on diet was longer, as were the challenge phases and wash-out periods. With these changes in study design the problems of order effect in earlier studies were resolved.

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Table 1 The historical development of concepts in the role of diet and hyperactivity from mid 1970s to mid 1980s**USA**

1973 Feingold first linked the ingestion of artificial colours, flavours and salicylates with hyperkinesis and learning difficulties [salicylate data from 1932].¹

1975 The book *Why your child is hyperactive* was published.²

1976–78 Three studies which refuted Feingold's claim published.^{3–5} Order effect found; parent and teacher ratings did not agree.³ Reported hyperactivity reduced in only a small number with preschoolers rated better on diet.⁴ Researchers challenged with 26 mg dye, the estimated daily dye intake.⁵ Chocolate was often used as a mask in test and control foods.^{4,5,16}

1977 and 1980 National Advisory Committee reports: no data suggests changes in food manufacture needed; effects asserted by Feingold not found.⁶

Clinical report findings: families vary in preference for dietary treatment; some diet responders still need medication as well; poor school work continued in some; chocolate reported as another aggravating substance; brain-damaged children did not improve.⁷

The Clinical ecology movement influenced public opinion.⁸

Australasia

From 1976 Stricter diet used with some 1974 salicylate data. Clinical findings: individual variation in additives tolerated; some take days to reach threshold; petrol fumes and felt pens could trigger reactions.⁹

Food craving involved: relief by repeated ingestion.¹⁰

First trial of artificial flavour [nature identical mango] conducted; not tolerated.¹¹ Is dose important? Artificial flavours in foods are used in 10 times the dose of colours (Hulscher, pers. comm., 1988).

Moulds, mites and aromatic trees considered.¹²

Diet affected symptoms in parents and siblings; foods, as well as additives, implicated in some; concomitant reactions (bed-wetting, 'neurotic' and physical allergic symptoms) decreased; ? different expression in susceptible females.¹³

1978 New Zealand report: reaction to additives and salicylates differed; few responders tolerated salicylate.¹⁴

Infections, stress, inhalants increase severity of food allergic symptoms.¹⁵ Double-blind dye challenge studies found short duration effect.^{16,17}

A report stated 26 mg dye as only 40% of daily intake.¹⁸

1980 100 mg dye effect on learning tasks peaked by 1.5 h, lasted 3 h.¹⁹

Sugar connected to delinquent behaviour in popular press,²⁰ but not implicated in research.²¹

1981 Allergic exposure may provoke both physical and psychological symptoms.²²

1986 Symposium on diet and behaviour, good overview.²³

Expectations if mechanisms were pharmacological.²⁴

1987 An overview reference on food allergy and intolerance.²⁵

possible problems (in 15 of 24).³⁰ Where a family diet history revealed sensitivity to some food in the patient or a first degree relative that food was limited or excluded in the test diet in an Australian study.³⁴ These less restricted diets made compliance easier while the diet remained stricter than that used by Feingold. As well as foods, perfumes, fumes and suspect environmental inhalants were minimized by many of the later workers.

CHALLENGES

Instead of challenging with artificial colours only, the later workers challenged with several additives, natural chemicals and whole foods. Or, if the total diet was provided, as by Kaplan's group, the challenge diet reintroduced all suspect items and foods together.³⁰ Where dye was included in challenges the later workers used information from re-evaluation of the average daily intake available in the late 1970s.¹⁸ Their dye doses increased from 26 mg to 50–250 mg. Natural chemicals ranged from 3 to 600 mg and whole foods from 5 to 30 g. In order for whole foods to be disguised in double-blind trials the dose had necessarily to be much smaller than the probable usual daily usage.

RESULTS SHOWED A ROLE FOR DIET

The most important finding was that in almost all studies there was a statistically significant change in behaviour with dietary intervention. A degree of change was noted with partial and full responses occurring rather than the all-or-nothing earlier expectation. Presenting factors found to correlate with a beneficial response were atopic history^{34,37,38} family history of migraine,³⁷ young age^{16, 34} and a family history of definite food/reaction connection.³⁴ Nutritional issues were monitored carefully by Kaplan's group with blood tests of nutrients showing no change on diet, and that poorly nourished children were not more susceptible.³⁰

INDIVIDUAL VARIATION OCCURRED

An important finding was that there were individual differences both in the variation of suspect chemicals or foods not tolerated both in early^{7,9,13,14} and later research^{26,28,30,31,33–35,38} as well as in the pattern of symptoms changed by diet.^{29,30,34,37}

SUSPECT ITEMS EXCLUDED

By the mid 1980s many more suspect items were excluded during the initial trial elimination diets. Egger *et al.* emphasized exclusion of most whole foods in the 'oligoantigenic' diet, which also excluded additives,²⁶ whereas Loblay and Swain emphasized additives and the natural chemicals, salicylates, amines and monosodium glutamate, while minimizing some whole foods as well.²⁸ This approach allowed expansion of the range of substances possibly implicated. To minimize the need for these very strict diets other researchers varied exclusions as addressed by the families, or as guided by the researcher. Kaplan's group excluded foods or chemicals considered suspect by individual families (e.g. apples, carrots, or salicylates; in four of 24), as well as dairy if the family reported a history of

IMPORTANT DIET EXCLUSIONS

Having established that there is a diet-behaviour connection, the issue of which diet exclusions are important, and which behaviours are affected next needs to be clarified. The detail of these aspects can be obtained from the various researchers' work but the important findings can be summarized. With regard to diet exclusions it reflects a major change in thinking that whole foods commonly implicated in allergy produced reactions as often as artificial colours. While this may be affected by selection factors, especially inclusion of atopic children, it does show that those investigating only suspect chemicals, or only whole foods, would have missed some diet responders. Boris and Mandel's double-blind challenge was with the item which

Table 2 Summary of significant diet/behaviour research from 1985–96

Authors	Type of study	Sample ADHD + / -	Number	Age range (Average)	Diet type	Days no	Outcome symptoms measures	Response % + ve
Egger <i>et al.</i> , 1985 ²⁶	Elim Dt	ADHD	76	3–12	1	7–14	B A M C	81a
	DBPCFC		28	(6.6)	c	d	R H	$P < 0.01$
David, 1987 ²⁷	Low add	ADHD	24	1–12	5	1	B	Not sig
	DBPCFC	Atopic	24	(5.2)				
Loblay and Swain, 1988 ²⁸	Elim dt	Atopic	140	4–16	1	1	B A C R	70a
	DBPCFC	+ ADHD	14		c	d	N etc	12/14
Rowe, 1988 ²⁹	Elim dt	ADHD	55	3–15	5	7	B A M	72a
	DBPCFC	+ sleep	8	(8.5)	c			
Kaplan <i>et al.</i> , 1989 ³⁰	Diet supplied	ADHD	24	3.5–6	1b	21	B A M R S	14a
		atopic	24	(4.5)	c	d	Halitosis	$P < 0.0001$
Rock and Atkins, 1990 ³¹	Elim dt	Atopic	480	0–19	2	1	C R G	
	DBPCFC		480				B M	39
Pollock <i>et al.</i> , 1990 ³²	Elim dt	Atopic	39	2–15	4	7	B C R G	
	DBPCFC		19	(8.9)		d		$P < 0.01$
Egger <i>et al.</i> , 1992 ³³	Elim dt	ADHD	185	3–15	1	1	B A H G	62.7
	EDP*DBC	Atopic	20	(9.3)		d		$P < 0.01$
Breakey, 1993 ³⁴	Repeated measures	ADHD +	112	2–16	3b	7	B A M C	69.7
		behav	112	(7.8)	c	d	R G S	$P < 0.05$
Carter <i>et al.</i> , 1993 ³⁵	Elim dt	ADHD	78	3–12	1	7	B A	75.6
	DBPCFC		19			d		$P < 0.05$
Francis and Rowe, 1993 ³⁶	Elim dt	ADHD +	2		5	1	B M S G	100
	DBPCFC	behav	2	3 and 6	c			2/2
Rowe and Rowe, 1994 ³⁷	Elim dt	ADHD +	200	2–14	4	1	B M S G	75
	DBPCFC	behav	54	(7.1)	c		C R H	$P < 0.05$
Boris and Mandel, 1994 ³⁸	Elim dt	ADHD +	26		5	7	B A	73
	DBPCFC	ATOPIC	19	(7.5)		d		$P < 0.001$

DBPCFC, Double-blind placebo controlled food challenge; a, partial as well as full responders noted; Outcome symptoms measured,—Behavioural, ADHD, Mood, Cutaneous, Respiratory, Headaches, GIT, Sleep; b, individual or family differences incorporated; c, perfumes, fumes or inhalants were minimised or excluded; d, washout time was incorporated between challenges; Diet type, 1 = most foods and additives; 2 = foods commonly implicated in allergy, 3 = additives, salicylates, amines, MSG, 4 = additives only, 5 = additives and some whole foods; ADHD, attention deficit hyperactivity disorder.

had had the strongest reaction on open trials, and this was a grain or dairy foods as often as additives.³⁸ Overall the suspect substances are whole foods implicated in allergy (e.g. egg, milk, peanut, wheat, fish and soy;^{26,38,40} it is suggested that whole foods to be considered should be those which have produced a definite physical or behavioural reaction in the child or a first degree relative at some time),³⁴ natural and medicinal salicylates, natural and added monosodium glutamate, natural amines and added colour as well as flavour and preservatives. Non-food items that have been implicated are perfumes, fumes, inhalants commonly implicated in allergy, infections and stress. Many researchers report that most subjects react to more than one test item.

SYMPTOMS THAT DIET MAY EFFECT

Which symptoms does diet change? Early studies evaluated outcome by measuring hyperactivity itself and specific learning tasks. Over time, settling and sleep problems were also reported as changing, then physical allergic or food intolerant symptoms. An important unexpected finding is the number of researchers who emphasize that the symptom most affected by diet is mood especially irritability.^{29–31,34,35} One example was a researcher who allowed families to add areas of behaviour not on their questionnaire used if they were concerned about them (e.g. whining)³⁰ They reported that these symptoms also

changed to a significant degree on the diet. This adds weight to the concept that parents and teachers monitor different symptoms and that this difference can add rather than detract from results; a similar difference has been documented in other areas of child psychiatry.³⁹

TOTAL BODY LOAD

While there is considerable overlap of suspect substances studied a valid question arises as to why different researchers emphasize different exclusions, challenge with various additives, natural chemicals or whole foods and still obtain significant results? This is possible if reactions are seen as partly pharmacological in nature. If the 'total body load' of suspect items is reduced enough then each child will show a reaction on challenge to whatever is their individual susceptibility.

RECOMMENDATIONS ON CURRENT PRACTICE

Professionals can now be aware of dietary treatment as an option for some children. They can be supportive of parents who wish to consider diet, particularly as motivation is important in the diet implementation. The 'diet detective process' will clarify whether diet has a useful, minimal or no role in the child's problems.

It is wise to ask about sleep and physical problems in children with attention deficit disorder (ADD) presentations, and behavioural or attentional problems in those with atopic symptoms. Parents are also focused on the presenting problem and may not mention the others unless asked.

Which children are most likely to benefit? A positive outcome is more likely if the child is atopic; if there is a family history of migraine, if the child is young, or if a parent can give a definite example of a food/behaviour change connection. Additions this author would add are atopy in any first degree relative, significant problems still remaining in those who have been helped by medication, (e.g. in the evening after medication effects have worn off), and changes in mood and ADD symptoms that are inconsistent with the usual stresses of childhood.

In the past the procedure seemed simple: investigate artificial colours in hyperactivity, chocolate in migraine, dairy foods in asthma and so on. It has now become clear that such specificity does not apply and any investigation should include the broad range of presenting problems and suspect substances. The diet investigation should consider all suspect foods and suspect non-food items at a level of strictness determined by age, severity of problems and motivation. Individual variation in outcome can be expected in the amount of change, in the food substances producing reactions, and in the areas of change.

Diet therapy involves incorporating family sensitivity history details, a detailed diet instruction with printed support material, involving a minimum of 2 h contact time and phone support as required over a 3-month period. The detail of the diet therapy is not within the scope of this paper. Exclusion of only those foods parents have implicated from obvious reactions is rarely sufficient. The child will increase consumption of other less obvious problem foods and any benefit may be lost. Diet therapy involves understanding interactions of diet issues with family dynamics, difficult behaviours in the child, fussy eaters, social and school food management, and the family coping with relatives and professionals unsupportive of diet use.

Rather than saying diet is too hard, or it is easy (just excluding the well known suspect foods) it can become manageable and be maximally effective with the help of a dietitian, preferably one experienced in this specialized area.

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