



DIET & BEHAVIOUR

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1. INTRODUCTION

A belief that diet affects behaviour is probably as old as humanity. It is enshrined in such proverbs as the English "a man is what he eats" and the German pun "was Mann esst, das er ist". In primitive times, and among primitive people today there was and is a belief that by eating a particular food the eater could acquire the desirable characteristics of the animal (or more rarely, plant) which furnished that food. Thus venison would confer swiftness, lion-flesh bravery, certain plants virility, and so on. Such sources of food were prized. By the same token, sources of food associated with undesirable qualities such as sloth and timidity were avoided (Frazer).

These beliefs provided one of the motives for cannibalism. To eat the flesh and drink the blood of an opponent whose behaviour had been conspicuously brave would transfer that bravery to the eater. They also formed the basis of numerous taboos and religious observations, some of which survive today.

With the passage of time and the divergent cultural attitudes of different peoples, beliefs become less clear-cut. The Cherokee prized venison because deer are swift; the Dyaks avoided it because deer, though swift, are also timid; the Kayans accepted that venison could confer both swiftness and timidity but believed that cooking venison out of doors allowed the spirit of timidity to escape.

Beliefs such as these, and many other less simple ones, were the result of abstract reasoning without the benefit of observations, or of observing a correlation and assuming that it must be causal. They are not all confined to primitives times or primitive peoples.

The constitution which Napoleon devised for England (which he did not have the opportunity to impose) took account of what he saw as that nation's characteristics as contrasted with the individual characteristics of other nations, as manifested by their different patterns of behaviour. He ascribed these differences to differences in diet (de la Lazere, 1837).

In the late 19th and early 20th centuries the rapid growth of scientific knowledge led many to a new view, that behaviour was determined wholly by heredity and environment and that diet had nothing to do with it.

Diet could influence a person's gross physical characteristics and health according to its quantity and basic composition, but it could not influence that person's nature and behaviour.

(An exception to this was the discovery in 1938, that the disease Pellagra which often caused depression, confusion and delusions was the result of a deficiency of niacin in the diet). The consensus of scientific opinion today is that diet can influence behaviour, but in ways more subtle and complex than had ever been imagined. Today also, a subculture of ancient folk-beliefs still persists, for example that red meat provokes aggressiveness, or that shellfish and crustaceans provoke lechery. Recently added to these are more widespread popular beliefs which derive from unsound scientific work, or from sound scientific work which has been misleadingly reported.

These popular beliefs now current mainly concern antisocial behaviour: hyperactivity, aggressiveness and violence, which in the main are ascribed to sugar (sucrose), food additives, and sometimes to all forms of processed food. They have persuaded some people to change their own or their children's diets, and led some schools and penal institutions to change the diets of their pupils and inmates.

The sections which follow will discuss some of the evidence for and against these popular beliefs.

2. CAN ADDITIVES AFFECT BEHAVIOUR ?

Additives are blamed for many things. Among these, hyperactivity is prominent. The claim for a link dates from 1975 (Feingold). Feingold claimed that food-colours, some preservatives and certain natural foods (those which contain salicylates) could provoke hyperactivity in children. This hyperactivity could be cured by eliminating these items from the diet. The so-called Feingold diet has become widely known and widely used. It has even led to claims in lay publications that all additives of whatever kind cause hyperactivity, regardless of their composition (Griggs 1983). Such claims can be dismissed, as contrary to reason.

To test the hypothesis that certain additives (or anything else) cause hyperactivity entails particular difficulties. There is no universally accepted criterion of hyperactivity. Its incidence has been reported as 0,1 % in the U.K. and 10 % in the U.S. (Dickerson 1980). Such a divergence must be mainly or wholly explained by differences in diagnostic criteria. Any valid test must measure the degree of hyperactivity and this is necessarily difficult and again there is no universally agreed method.

Underlying these difficulties is a basic problem: the difference between what a properly controlled "double-blind" test reveals, and what an individual parent may believe. If a mother has an unshakeable conviction that a particular change of diet will cure her child's hyperactivity, that change may induce a real (or at any rate a perceived) improvement by reason of the mother's demeanour.

In such a case the diet-change would be effective as far as that particular mother was concerned even though a double-blind test might show that it was not.

In these circumstances it is not surprising that one review concludes that the Feingold hypothesis is broadly correct (Weiss, 1982), another that it is not (Matters and Gittelman 1981) while a third finds that the evidence for and against it is insufficient to justify any firm conclusion (Nat. Inst. Health 1982).

Two typical early studies were those of Connors (1976) and Larley (1978). They compared the effects of a modified Feingold diet with a controlled diet. The former showed a statistical superiority for the diet without artificial colours but the latter did not. The effect, where found, only occurred when the colour-free diet followed the control, and only with a few of the children.

Many other tests, with groups of children who had already been identified as hyperactive or as diet-responsive, either showed no adverse effect from additives (Mattes 1981, Harley 1980, Connors 1980) or an effect which was small and short-lived (Goyette 1978).

When children have been assessed in individual experiments, a small proportion have been found to respond favourably to additive-free diets. A typical trial in this category (Weiss 1980) examined 22 children who had already been diagnosed as diet-responsive. Each child was put on a diet free from artificial flavours and colours, the preservatives BHA and BHT, and various fruits and vegetables which were believed to exert a bad effect on their behaviour. Each day, each child was given either a soft drink containing artificial colours or an indistinguishable placebo. Behaviour was assessed on the basis of several standard indicators.

Of the 22 children, 20 showed no response to the challenge-drink, one responded slightly and one dramatically.

Bearing in mind that this and similar trials were restricted to children who were already believed to respond badly to additives (albeit not by way of controlled double-blind experiments), it appears that such additives are not likely to worsen the behaviour of most children though they will affect a few.

3. SUGARS AND BEHAVIOUR, THE ROLE OF NEUROTRANSMITTERS.

Neurotransmitters are substances which regulate the functions of the central nervous system. About forty of them have been identified so far. Of these, serotonin is the most studied. Levels of serotonin in the brain influence sleep, sensitivity to pain, motor function, mood and possibly food selection.

The ways in which diet may affect neurotransmitters and thereby behaviour was reviewed in papers presented at a meeting of the International Institute of Life Sciences and the American Medical Association (ILSI-AMA) held in Virginia U.S.A. in 1984 (Wurtman, Fenstrom, Young, Pardridge).

It has been shown that increased levels of serotonin produced after eating carbohydrate decrease "sleep latency", i.e. the time it takes to get to sleep.

The same effect can be elicited by administering the aminoacid tryptophan, which is the precursor of serotonin. However, tryptophan does not occur as such in food, but only in combination with other aminoacids in the form of protein. In this situation, which is the normal one, the effects of tryptophan are modified. This is because tryptophan must compete with other aminoacids for transport into the brain.

There is evidence that a meal rich in carbohydrate stimulates the synthesis of serotonin whereas a meal rich in protein depresses it. The effects on behaviour of a meal rich in carbohydrates (sugars and starches), are in the direction of relaxation and calm, even to the point of lethargy (Wurtman, ILSI-AMA 1984). It is a matter of common observation that a heavy lunch may induce lethargy for a time. This evidence suggest that such an effect is more likely to occur if the meal is rich in carbohydrate (including sugar).

Although many studies have confirmed this effect, there is evidence that the differing effects of carbohydrate and protein may not be found when studied over a long period, rather than in a single short experiment as is usually the case (Gerstrom 1984).

At the time of writing, it is clear that diet may influence behaviour by way of its effects on the synthesis of serotonin (and other neurotransmitters). However, it is probable that if fully confirmed these effects will not prove to be large, and certain that they will not be in the direction of hyperactive, aggressive or criminal behaviour.

A recent study of the effects of diets high in sucrose and aspartame on behaviour and cognitive performance in children showed no effect of even high levels of sucrose on children's behaviour or cognitive function (Wolraich, 1994).

4. SUGAR AND BEHAVIOUR: DOES HYPOGLYCAEMIA HAVE A ROLE ?

Many lay publications and television and radio programmes blame antisocial behaviour on the overconsumption (or even just the consumption) of sugar. In doing this, the concept of "reactive hypoglycaemia" is often invoked. Reactive hypoglycaemia is a fall in the level of blood-glucose below what is normal after the initial rise which always follows the intake of sugars or starch. Hypoglycaemia only occurs after overproduction of insuline in response to a very high intake.

A typical lay publication (Fredericks, 1969) asserted that for 10 % of the population "sugar is as deadly as carboic acid", by virtue of its hypoglycaemic effect, and claimed it could lead to symptoms ranging from leg-cramps to suicidal actions. Assertions of this kind, widely and persistently repeated, have caught the imagination of the general public and also of social workers, educators, criminologists and others. Recently (1989) hypoglycaemia was even offered in a court, albeit unsuccessfully, as defence against a charge of rape.

Reactive hypoglycaemia has been claimed to be prevalent among juvenile delinquents and convicted criminals with estimates of its incidence ranging up to 90 %. (Schauss 1980; Virkkuner 1982, 1983, 1984; Virkkuner and Huttuner 1982).

In fact, hypoglycaemia (reactive and otherwise) is a rare condition. The accepted definition of hypoglycaemia is a level of blood-glucose less than 40 mg/100 ml; for reactive hypoglycaemia it is this level of blood-glucose associated with symptoms, in which the same symptoms must occur regularly after meals and must be relieved within 10 to 20 minutes by eating.

Hypoglycaemia (and other clinical disorders) is traditionally diagnosed by the five-hour oral glucose tolerance test, or OGTT. In this, the patient takes a large dose - 50 g - of glucose after a 12-hour fast and his or her blood-glucose level is measured at intervals following. The test is an artificial one for some purposes. Persons who develop a low level of blood-glucose during this test often do not do so after a normal meal.

Furthermore, some people may have a blood-glucose level as low as 30 mg/100ml without exhibiting any symptoms (Anderson and Lev-Ran, 1985) while others with a normal glycaemic response may show symptoms nonetheless.

Lastly the symptoms traditionally associated by the medical profession with hypoglycaemia, when they do occur, are those of fatigue, anxiety, lethargy and loss of vitality. They do not include and in fact are opposite of hyperactivity and aggression except where the individual experiences hypoglycaemia due to pre-existing diabetes.

It has been claimed that the high incidence of reactive hypoglycaemia reported by some authors is partly accounted for by the use of incorrect or inappropriate diagnostic criteria (Gray and Gray 1983).

A position paper by the American Dietetic Association, 1985, concluded "Valid evidence is lacking to support the hypothesis that reactive hypoglycaemia is a common cause of violent behaviour". This conclusion would seem to be justified from the available evidence.

5. DIET AND BEHAVIOUR: OTHER STUDIES

Not all studies have been based primarily on the concepts of "reactive hypoglycaemia", neurotransmitter-synthesis or the role of additives.

The suggestion that diet could adversely affect behaviour in some way, was made in scientific literature as early as 1917 (Williams), but for several years there was little follow-up.

In 1979 a detailed comparison was made of the diets of chronic juvenile offenders and of a matched group of disordered but non-delinquent juveniles (Schauss 1979). It appeared that the offenders drank more milk than the controls and took in less micronutrients per 1000 kcal (though they took in more in total).

Schauss pointed out that the data did not determine whether the diets caused the differences in behaviour, or the other way round.

A series of studies reported in 1983 (Schoenthaler 1983) have been widely quoted. These examined the effect of dietary changes on large numbers of criminal or delinquent inmates in several institutions. The dietary changes involved replacing sugar (sucrose) with honey; soft drinks with fruit juice; and cereals, desserts and snacks with fruit, peanut butter and popcorn. The author claimed a dramatic improvement in behaviour following the change in diet.

Although widely quoted in lay publications these and other similar studies have been widely criticised by the scientific community. (e.g. Gray 1986). The main criticisms have been of flawed design and lack of controls. As examples: the improvement in behaviour was attributed to a reduced intake of sugars, but the intakes of sugars were not quantified. The composition of the inmate populations changed during the trials. In one study where the composition of the inmates did not change, the "improved diet" was eaten during the latter half of a 62-day sentence when improved behaviour might have been expected anyway.

Another study reported by Schoenthaler (Schoenthaler 1983) differed from the others in that the inmates were already on a low-sugars diet. Here the main dietary change was the replacement of drinking water by orange-juice at mealtimes. This was followed by an improvement in behaviour. Such improvement could have been the result of several things: one of them, an increase in the intake of simple sugars (since orange-juice contains these).

The position paper by the American Dietetic Association (1985) concluded "Valid evidence is lacking to support the claim that diet is an important determinant in the development of violence and criminal behaviour".

Many studies have investigated the claim that sugar (sucrose) can cause or exacerbate hyperactivity (as opposed to criminal or near-criminal behaviour). These earlier claims were made largely on the basis of anecdotal or testimonial evidence only. The studies summarised below were all done using "blind" techniques, designed to eliminate any possible preconception or bias among the observers.

One such study (Prinz et al 1980) examined the diets of hyperactive and normal children. No difference was found in the sugar-consumption of the two groups, though the hyperactive group ate less total weight of food. No difference was found in the consumption of those items forbidden in the "Feingold Diet".

A positive correlation was found, in the hyperactive group, between sugar-consumption and the degree of certain types of activity. However, the authors stressed that a correlation does not prove a cause-and-effect relationship, and advised further studies.

Bahar et al (1984) measured the effect of challenges with glucose, sucrose and placebo (saccharin) on 21 boys whose parents claimed they reacted adversely to sugar. The intake of sugar was found to decrease motor activity, but observers' behavioural rating found no difference following the various challenges.

Wolraich et al (1984) in a similar trial using sucrose and an aspartame placebo also found no differences in behaviour.

Conners et al (1984) reported two studies designed to test the sugar-behaviour hypothesis. In the first, high doses (50 mg) of sucrose and fructose increased levels of activity compared with controls.

In the second, 38 hospitalised children with behavioural disorders were challenged with sucrose, fructose, and placebo (aspartame). In this trial, the challenges with the two sugars decreased behavioural problems.

Another pair of studies (Ferguson 1984) found no difference in behaviour after challenges with sucrose and placebo (aspartame) although the parents of one group had believed that their children's behaviour was worsened by sugar.

A more extended study (Mahan 1985) examined the behaviour of 16 children whose parents described them as "difficult to manage" rather than hyperactive. The parents had already eliminated sugar from their children's diet and had claimed improvement. The researchers evaluated the children's behaviour before and after they had eaten a candy bar which contained more sugar than the amount claimed to affect behaviour. Seven of the children did react adversely, as measured by observers.

In a second phase of the study, five of the children who had reacted adversely were challenged with mixtures which contained either sucrose, honey, tapioca starch or placebo on four separate non-consecutive days. The smell, taste and appearance of these were disguised.

Two of the children responded adversely to sucrose the first time when tested, but not the second. The effect could therefore have been either one of placebo, or a transient sucrose intolerance.

Gross (1984) reported the case of a boy and his mother both of whom were found to be hypersensitive to sucrose, but not to the sugars glucose and lactose, or to saccharin.

To check whether such hypersensitivity was common, 50 children were given challenge doses of lemonade containing 75 mg of sucrose and lemonade sweetened with saccharin. This was done three times. The mothers had all claimed that they "knew" that their children's behaviour was much worse after they had eaten or drunk anything which contained sucrose. But no difference in behaviour could be found following the challenges with sucrose and saccharin.

Taken together, these studies show that hyperactivity may be caused by hypersensitivity to sucrose but that this is very uncommon. If suspected it can be tested for very simply.

6. IOCCC POSITION

The IOCCC agrees that diet may influence behaviour but not in the manner and to the extent it is popularly believed to do.

In particular, the IOCCC accepts the consensus of scientific opinion that sugar (sucrose) and additives are not common causes of antisocial behaviour. It disapproves of unsubstantiated claims that they are and welcomes any efforts to dispel misconceptions.

Nevertheless, IOCCC realises that a small number of people are sensitive to some food ingredients or additives. To enable those people to avoid these, IOCCC encourages its members to label products with a list of the ingredients used therein.

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