

## Nutrition and mental performance

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There is no doubt that nutritional status can have significant and sometimes profound effects on human mental performance (Table 1). This is evidenced by, for example, the consequences of chronic and severe food restriction (Smart, 1993), thiamin deficiency (Kanarek & Marks-Kaufman, 1991), and Fe deficiency (Pollitt, 1987). Acute effects of food and fluid ingestion have also been described, and it is this area that is the main concern of the present review.

### ALCOHOL AND CAFFEINE

The two dietary constituents which have been studied most extensively in relation to such acute effects are alcohol (Steele & Josephs, 1990; Finnigan & Hammersley, 1992) and caffeine (James, 1991; van der Stelt & Snel, 1993). Both these substances produce their various effects on cognitive functioning primarily by direct (pharmacological) action on the central nervous system (CNS), although, for example, some of the consequences of long-term alcohol abuse depend on nutritional factors associated with excessive alcohol consumption (Kanarek & Marks-Kaufman, 1991). Very broadly, alcohol and caffeine can be characterized as having respectively depressant and stimulant psychoactive

Table 1. *Examples of nutritional variables known to affect mental performance\**

Variable	Established	Less-well established
Food restriction	Early life undernutrition Chronic semi-starvation	Dieting to lose weight Short-term fasting (e.g. missing a meal)
Pre- v. postprandial state		
Dietary macronutrient composition		Acute effects Chronic effects
Micronutrient deficiencies	Thiamin (Wernicke-Korsakoff syndrome) Fe	
Phenylketonuria		
Diabetes	Hypoglycaemia	Chronic effects
Alcohol	Acute intoxication Chronic effects, including fetal alcohol syndrome	
Caffeine and caffeine withdrawal		

\* For sources, see text and Kanarek & Marks-Kaufman (1991).

effects, and at least under some circumstances caffeine can offset alcohol-induced performance deficits (Hasenfratz *et al.* 1993).

A difficulty in interpreting the outcomes of studies on caffeine, however, is that this substance is consumed both widely and very frequently, and cessation of consumption is accompanied in many regular users by adverse changes such as increased incidence of headache, drowsiness and fatigue (James, 1991). Indeed, some of the studies on the withdrawal effects of caffeine are similar in design to many of the studies purporting to show improved mood and/or performance efficiency following administration of caffeine. A typical procedure is to compare the effects of caffeine and a placebo in caffeine-deprived subjects (e.g. Clublely *et al.* 1979; Bruce *et al.* 1986; Lieberman *et al.* 1987). The problem with this approach is that it leaves open the question as to whether the findings are due to beneficial effects of caffeine or to deleterious effects of caffeine deprivation (or a combination of both of these).

Surprisingly, this issue has received very little attention. The effects of caffeine in non-users of caffeine (i.e. in individuals who do not regularly consume coffee, tea, cola etc.) have not been examined in detail, perhaps partly because of the ethical questions this raises. In any case, differences between the responses to caffeine of users and non-users of caffeine could be due to a number of factors. For example, caffeine users were found to score higher than non-users on measures of addiction, extraversion and impulsivity (Eysenck Personality Scales; Eysenck & Eysenck, 1991; N. J. Richardson & P. J. Rogers, unpublished results). Other results show that on certain tasks high impulsives, assumed to have a relatively low level of endogenous arousal, benefit more from the performance-enhancing effects of caffeine than do low impulsives (Smith *et al.* 1991). There is also a strong relationship between the level of use and the pharmacokinetics of caffeine, which has been interpreted as indicating that users absorb caffeine more efficiently than non-users (Kuznicki & Turner, 1986). Another approach, which again has been used only very rarely, is to test the effects of caffeine in users who have been 'fully' withdrawn from caffeine. This requires subjects to be deprived of caffeine for a minimum of 1 week and preferably longer (Griffiths & Woodson, 1988). Bruce *et al.* (1991) found increased tiredness in caffeine users deprived of caffeine for 24 h compared with users deprived for 7 d. The 24 h group also showed a more rapid decline in performance on a simple tapping task. When the subjects were given caffeine this reduced (compared with placebo) tiredness and also headache only in the 24 h group. Since very high doses (250 and 500 mg) were administered, it is unlikely that altered caffeine pharmacokinetics alone can account for the lack of significant effects of caffeine in the 7 d-deprived group.

Of course such findings by no means exclude the possibility that there is a net benefit to be derived from the regular use of caffeine. Indeed, there is good evidence that caffeine can improve absolute levels of cognitive performance. In particular, it appears to be effective in counteracting deteriorated performance. Examples of this are findings indicating that caffeine has stronger performance-improving effects in older than younger adults (Jarvis, 1993), and that caffeine can offset certain aspects of the 'post-lunch dip' in mental performance (Smith *et al.* 1991) and drug-induced impairments in performance efficiency (e.g. File *et al.* 1982). Nonetheless, some of the benefits experienced following the consumption of caffeine are probably more apparent than real because these effects involve the reversal of the deleterious consequences of caffeine deprivation, for instance, those that occur after overnight withdrawal (Rogers &

Richardson, 1993; N. J. Richardson & P. J. Rogers, unpublished observations). In order to assess the net benefit of the everyday use of caffeine it will be necessary to carry out further investigations where the psychoactive effects of caffeine are evaluated in relation to caffeine withdrawal, and using dose levels and a pattern of administration which model 'real' behaviour more closely.

#### BREAKFAST, LUNCH AND MEAL COMPOSITION

The effects of meals on cognitive efficiency have been recently reviewed in detail by Smith & Kendrick (1992). Relatively little work has been carried out in this area, although some of the earliest studies were reported over 40 years ago. A common finding is that performance is impaired in the early afternoon compared with performance measured in the late morning (the so-called post-lunch dip). For some tasks this occurs whether or not lunch has been eaten, although consuming a meal at lunchtime, particularly if this is a large meal, does appear to contribute significantly to the post-lunch dip. Tasks which are most typically affected are those requiring sustained attention (Smith & Kendrick, 1992). There is, however, a problem concerning the interpretation of results showing impairments when no lunch is eaten, which might indicate effects related either to fasting or to an underlying circadian variation in, for example, alertness. If the latter were responsible, performance would be expected to recover later in the afternoon. Unfortunately, repeated assessment of performance efficiency, by introducing fatigue or practice effects, may itself influence the outcome of the experiment. Additionally, it is important to note that post-lunch impairments in performance are by no means always observed. In particular, several results suggest that meals consumed at lunchtime have least effect on performance efficiency under conditions of high arousal; for instance, when subjects are given caffeine (see p. 444), or when they are exposed to a high level of background noise (Smith & Miles, 1986). The effects of lunch on performance also appear to be quite strongly related to personality factors such as extraversion and neuroticism (Craig *et al.* 1981; Smith & Miles, 1986).

In contrast to the effects of lunch, there is a popular belief that missing breakfast can have an adverse effect on cognitive efficiency. When taken together, however, the published experimental evidence does not substantially support this belief. A series of studies conducted by Tuttle and colleagues (for review, see Editorial, 1957; Smith & Kendrick, 1992) indicated somewhat poorer reaction-time performance in subjects who had not been given breakfast, and recently a similar result has been reported for memory performance (Benton & Sargent, 1992). In addition, 9–11-year-old children were found to score significantly less well on a problem-solving task (matching familiar figures test) when tested in the morning following an overnight fast compared with when they were tested following breakfast (Pollitt *et al.* 1981, 1983). This latter work has received a considerable amount of attention, but the finding of a detrimental effect of missing breakfast should be viewed in the context of other studies where this was not observed (e.g. Dickie & Bender, 1982; Smith & Kendrick, 1992). Also, given the fairly long period of food restriction imposed in the experiments by Pollitt *et al.* 1981, 1983; (the children were tested either 3 or 18 h after their last meal), it could be argued reasonably that the lack of more substantial effects indicates that cognitive performance is relatively invulnerable to short-term fasting. In fact, of the various measures of performance taken, only one showed a statistically significant deterioration related to missing

breakfast, while one other (frequency of recall of the last item on a test of immediate memory) showed a small but significant improvement (Pollitt *et al.* 1981).

Very little information is available on the effects of meals eaten at other times of the day. However, there has been more interest in the influence of meal composition on cognitive performance. This is because of certain theoretical predictions concerning the proposal that the proportion of protein relative to carbohydrate in a meal can modify the ratio of the plasma concentration of tryptophan to the other large neutral amino acids (Trp: $\Sigma$ LNAA), and thereby alter brain tryptophan uptake, brain serotonin synthesis and serotonergic function (Fernstrom & Wurtman, 1972; Wurtman *et al.* 1981; Fernstrom, 1994; Fernstrom & Fernstrom, 1994). The behavioural consequences which have been hypothesized to follow from these meal-induced changes in neurotransmission include altered food choice and food intake, and changes in pain sensitivity, aggressiveness, mood, alertness and cognitive performance. Accordingly, the relative increase in brain serotonergic activity that supposedly follows the consumption of a high-carbohydrate *v.* a high-protein meal can be expected to give rise to a decrease in alertness and an increase in sleepiness, and consequently a fall-off in performance efficiency (for reviews, see Spring *et al.* 1987; Young, 1991). Although the evidence is not strong, where differences have been found, these were in the direction of poorer performance after a high-carbohydrate compared with a high-protein meal (Spring *et al.* 1983; Lieberman *et al.* 1986b). Reaction time and tasks requiring sustained attention were most affected. Nonetheless, even in these studies, a large majority of the comparisons between subject groups or performance measures failed to reveal statistically significant effects of meal composition. In addition, administration of tryptophan alone was found to have only a marginal effect on one (simple auditory reaction time) of four tests of performance, despite the fact that the dose used, approximately 3.5 g, was sufficient to produce a number of measurable subjective changes, including decreased alertness and vigour, and increased fatigue (Lieberman *et al.* 1983; Hrboticky *et al.* 1985; Spring *et al.* 1987).

A further difficulty for the proposed relationships between the protein-carbohydrate content of a meal and mood and performance, is that the observations relating brain serotonin and meal composition have been obtained from studies on rats, not humans. Studies on humans have shown statistically significant differences in plasma Trp: $\Sigma$ LNAA values following 'meals' of starch, sugar and protein, but it appears that the magnitude of these effects is probably too small to produce functionally significant changes in brain serotonergic activity (Leathwood, 1987; Young, 1991). The actual changes observed were small increases in plasma Trp: $\Sigma$ LNAA following starch and sugar meals and a larger decrease in this ratio after high-protein meals. Differences in Trp: $\Sigma$ LNAA did not reach their maximum until 2–3 h after these meals, with almost no change occurring within the first hour after a carbohydrate meal (e.g. Lieberman *et al.* 1986a; Christensen & Redig, 1993). In the latter study there were no corresponding effects on mood and alertness. Also, meals which are more typical in composition are unlikely to produce any distinct differences in postprandial Trp: $\Sigma$ LNAA values, since the presence of a small amount (perhaps as little as 4% energy) of protein in a high-carbohydrate meal is sufficient to block any meal-induced increases in plasma Trp: $\Sigma$ LNAA (Leathwood, 1987; Teff *et al.* 1989; Wurtman & Wurtman, 1992; Christensen & Redig, 1993).

Other studies examining the effects of meal composition include an investigation by Smith *et al.* (1988) who found that isoenergetic, high-carbohydrate and high-protein meals affected different aspects of attention. However, because the high-carbohydrate

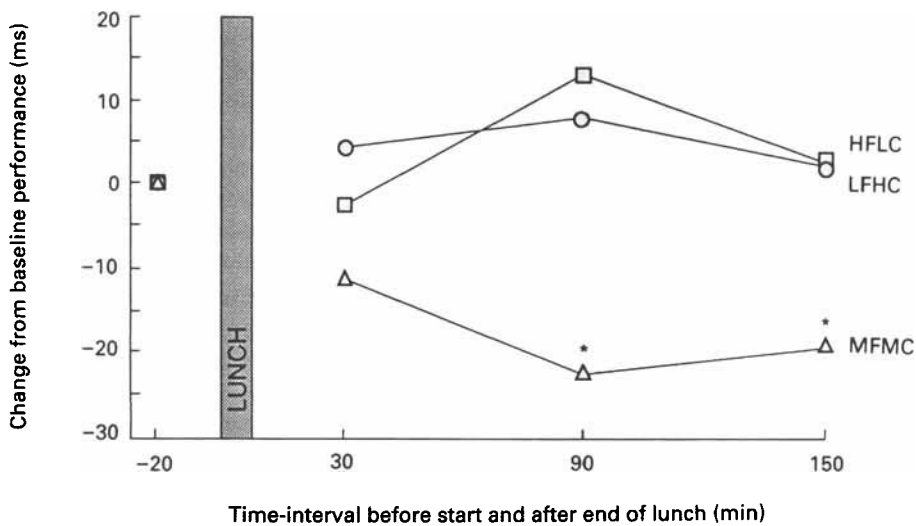


Fig. 1. Changes in simple reaction-time performance following isoenergetic (2.93 MJ (700 kcal)) lunches differing in fat and carbohydrate content. Baseline measures were taken 20 min before lunch (-20). (○), low-fat (29% energy), high-carbohydrate (55% energy) meal (LFHC); (△), medium-fat (45% energy), medium-carbohydrate (42% energy) meal; (□), high-fat (62% energy), low-carbohydrate (24% energy) meal (HFHC). Mean values were significantly different from those for LFHC and HFHC: \* $P < 0.05$ . Adapted from Lloyd *et al.* (1994).

meals contained 15% energy from protein and testing began within 1 h after lunch it is very unlikely that this dissociation was mediated by differences in Trp:ΣLNAA. Moreover this mechanism can not account for recent results showing poorer reaction-time performance and lower alertness following isoenergetic lunchtime meals high in fat or high in carbohydrate compared with a meal containing intermediate levels of fat and carbohydrate (Lloyd *et al.* 1994). The findings for reaction time are presented in Fig. 1, which shows that the different effects of the three lunches were present to a moderate degree within 30 min of finishing the meal. At this time there were also differences in mood, including significantly greater 'drowsiness' following the high-fat and high-carbohydrate meals. On the other hand, several other measures of mood and performance (e.g. immediate recall, rapid information processing) were unaffected by meal composition. A further observation was that the medium-fat, medium-carbohydrate meal contained proportions of fat, carbohydrate and protein similar to those of the lunches usually eaten by the subjects. Perhaps, therefore, postprandial effects on mood and performance are a factor influencing food choice (and liking), such that choices producing desired effects are more likely to be repeated on future occasions (Booth, 1978; Rogers *et al.* 1992; Rogers & Richardson, 1993). This suggestion is supported by the results of a second study in which subjects' mood and performance were assessed in the morning following breakfasts containing different proportions of fat and carbohydrate. Again, optimal effects were seen after the meal with the macronutrient composition which most closely matched that normally consumed by these subjects at breakfast; in this instance, however, this was the meal having the highest carbohydrate and lowest fat content (62 and 28% energy respectively; H. M. Lloyd, unpublished results).

An important aspect of these last two studies was that the meals did not differ significantly (as measured by subjects' ratings) in appearance or orosensory qualities. For example, each of the three lunches consisted of a cheese and salad sandwich and a strawberry milk shake. Fat and carbohydrate content were manipulated by using low-fat cheese and spread and adding either cream or maltodextrin to the milk shake as appropriate (Lloyd *et al.* 1994). Clearly, if meal appearance, taste and other variables are not controlled, the effects of these variables, perhaps due to subjects' expectations, may be confounded with the effects of nutrient composition. Unfortunately, it is very difficult to alter the protein:carbohydrate value of a meal to any substantial extent without also altering its orosensory qualities, and typically there has been no attempt to disguise this manipulation. For instance, in the study carried out by Lieberman *et al.* (1983) subjects were provided with a meal consisting of either trimmed turkey breast (high-protein meal) or non-dairy sherbet (sorbet; high-carbohydrate meal). Similarly, the emotional impact and expectations arising from missing a meal, or being given an obviously much larger or smaller meal than usual, makes it difficult to draw unambiguous conclusions about the nutritional effects on mood and performance of these manipulations (Brooke, 1973).

There is, nonetheless, a measure of agreement in the findings from this work. For instance, although alertness tends to be increased and performance efficiency improved following a high-carbohydrate breakfast, meals with a high-carbohydrate content eaten at lunch have, if anything, the opposite effect (Lieberman *et al.* 1983; Lloyd *et al.* 1994; Wells & Read, 1994; H. M. Lloyd, unpublished results). The appearance of certain mood and performance effects within 0.5 h of eating suggests that pre-absorptive and/or early post-absorptive mechanisms are implicated. At present, however, the nature of these mechanisms is unclear. Indeed, it is difficult to be more specific than suggesting, for example, that many of the effects following lunch are consistent with a postprandial increase in parasympathetic activity. One study, though, has demonstrated substantial, dose-related effects of intravenously administered cholecystokinin (CCK) on alertness and performance (Stacher *et al.* 1979). This is of particular interest because fat and protein are more potent releasers of CCK than carbohydrate (Liddle *et al.* 1983). Therefore, CCK may provide part of a mechanism mediating effects of meal composition as well as meal size. In addition, adaptation of the gastrointestinal and other physiological responses to food associated with medium- to longer-term changes in diet composition (e.g. Cunningham *et al.* 1991) suggests that the behavioural effects of food can also be expected to vary as a function of individual dietary history. An implication of this is that, as well as exploring the mechanisms underlying the effects of meals on mood and performance, a priority for studies in the future is the investigation of the consequences of longer-term dietary changes.

#### EFFECTS OF GLUCOSE DRINKS

Table 2 summarizes the results of thirteen experiments which investigated the effects of glucose drinks on cognitive performance. Eight of these experiments revealed a significant difference between the effects of a glucose drink compared with a 'placebo' drink on at least one measure of performance. With one exception the differences were in the direction of better performance after the glucose drink. (The study by Brooke & Toogood (1973) is included on the assumption that their results reflect an effect on mainly cognitive rather than physical performance.) A clear finding is that the effects on

Table 2. Summary of some studies of the effects of glucose drinks on cognitive efficiency

Reference	Subjects	Treatments	Tests	Results	Notes
Brooke & Toogood (1973)	Fifty-eight male, forge workers Within-subjects design	Glucose syrup drink (2.97 MJ (710 kcal)) or equal volume, non-sweet drink, given in morning	Dependent variable was accident rate	*	No dietary restrictions Reduction in accident rate associated with glucose supplementation was more apparent in the morning than the afternoon work session
Birch <i>et al.</i> (1977)	Approximately 120 Age and gender not specified Within-subjects design	Drinks containing 50 g high- or low-molecular-weight glucose polymer syrups or iso-sweet saccharin drink, given 15 min before start of tests	'Intelligence test' consisting of verbal, numerical and visuospatial tasks	*	Testing started at 11.00 hours after 3 h fast
Lapp (1981)	Fifteen female and twenty-one male high school students (mean age 16.4 years) Between-subjects design	Carbohydrate (300 g) then glucose (150 g) drink 1 h later, given at least 15 min before start of test, control subjects given nothing	Paired-associate learning (memory)	*	Deprived from 22.00 hours previous night No information on exact time of testing Subjects receiving carbohydrate and glucose were included in the analysis only if their blood glucose levels were >7.2 mmol/l
Benton <i>et al.</i> (1987)	Thirty female and thirty male schoolchildren aged 6-7 years Between-subjects design	Glucose (25 g) in orange-flavoured drink or saccharin in same drink, given 15 min before start of tests	Simple reaction time Behavioural response to frustration	*	Testing started 2 h after lunch
Hall <i>et al.</i> (1989)	Eleven elderly (mean age 67 years), twelve students (mean age 20 years) Within-subjects design	Glucose (50 g) in lemon-flavoured drink or saccharin in same drink, given 10 min before start of tests, elderly given a second drink part way through tests (testing took 75 min for elderly and 45 min for younger subjects)	Several tests of memory modified from Wechsler memory scale	* (elderly) NS (young)	Tested in morning after overnight fast Results are a combined score for all tests Effects of glucose on test scores did not differ significantly between elderly and younger subjects Peak blood glucose above basal negatively correlated with test scores in elderly for both glucose and saccharin treatments

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Table 2. Summary of some studies of the effects of glucose drinks on cognitive efficiency

Reference	Subjects	Treatments	Tests	Results	Notes
Benton (1990)	Expt 1: twenty male students (mean age 20 years)	Glucose (25 g) in lemon-flavoured drink or aspartame in same drink, given 20 min before start of tests	Monitoring task lasting 24 min with concurrent distraction	*	Subjects asked not to eat for 4 h before testing
	Expt 2: twenty female and twenty male students (mean age 21 years)	As Expt 1	Hand-eye coordination	NS	No information on dietary restrictions
	Between-subjects designs				
Manning <i>et al.</i> (1990)	Twelve female and five male elderly (mean age 73 years)	Glucose (50 g) in lemon-flavoured drink or saccharin in same drink, given 10 min before start of tests	Recall of word list Recall of narrative passage Digit span Drawing from memory Selective letter cancellation Finger tapping	* * NS NS NS NS	Overnight (9 h) fast imposed Peak blood glucose above basal after glucose drink negatively correlated with memory performance
	Within-subjects design				
Azari (1991)	Eighteen male students (mean age 21 years)	Water containing 0, 30, or 100 g glucose made equisweet by addition of aspartame, served cold, given 30 min before start of tests	Free recall and recognition memory for word list	NS	Fasted overnight, given breakfast at 07.00 hours, 4.5 h before start of testing
	Within-subjects design				
Benton & Owens (1993)	Expt 1: fifty-three female and 100 male students (mean age 22 years)	Glucose (50 g) in flavoured drink or aspartame and acesulfame-K in same drink, given 15 min before start of tests	Immediate recall of word list Spatial memory	NS NS	No dietary restrictions imposed For glucose and placebo treatments taken together there was better recall in subjects whose blood glucose was increasing during testing, and a positive correlation between blood glucose and recall

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Benton & Owens (1993)	Expt 2: fifty-three female students (mean age 22 years) Between-subjects designs	As Expt 1 plus further 25 g drinks 45 and 75 min later, first drink given 15 min before start of tests	Recall of narrative passage Immediate recall of word list	NS NS	No dietary restrictions imposed For glucose and placebo treatments taken together there was a positive correlation between blood glucose level and recall
Benton <i>et al.</i> (1994)	Expt 1: twenty-seven female students (mean age 22 years)	Glucose (50 g) in flavoured drink, then 25 g drink 25 min later or aspartame and acesulfame-K in same drink, first drink given 20 min before start of tests	'Rapid information processing task': errors other measures Immediate and delayed recall of word list	- NS NS	No dietary restrictions imposed Several significant correlations between blood glucose levels and performance (e.g. higher blood glucose associated with faster reaction times and lower rate of forgetting)
	Expt 2: fifty male students (mean age 22 years) Between-subjects designs	Same as Expt 1, first drink given 40 min before start of tests	'Stoop task' task is to identify as quickly as possible the colour in which different letters or words are presented e.g. colour names written in incongruent or congruent colours	NS	No dietary restrictions imposed Subjects (glucose and placebo treatments taken together) whose blood glucose levels were rising just before test gave faster responses on the incongruent task than subjects with falling blood glucose

\* Better performance after glucose drink; -, poorer performance after glucose drink; NS, the effect of glucose drink was not significant ( $P > 0.05$ ).

performance occurred within 1 h and often sooner after the drink was consumed. Furthermore, these effects were observed in fasted and non-fasted (i.e. last food  $\leq$  4 h before testing) subjects. Table 2 also shows that the tests on young adults yielded relatively few significant results. Overall, however, there is no apparent pattern to the aspects of performance or types of task affected. In addition to these results, Siebert *et al.* (1986) reviewed several papers published in German which report significantly improved performance following consumption of glucose drinks. Vigilance (i.e. sustained attention), in particular, was found to be improved. One experiment, for example, showed that when subjects were given glucose they made very few errors in a simulated driving task, whereas their performance when given a placebo deteriorated markedly towards the end of the test (Keul *et al.* 1982). Finally, there is at least one other report finding little or no improvement in performance following a glucose drink (Horton & Yates, 1987). This study is noteworthy because subjects with high and low dietary intakes of sugar were compared, although unfortunately a placebo test was not included in the experimental design.

A stimulus for some of the more recent studies in this series was provided by findings showing that glucose administration can enhance learning and memory performance in rats (e.g. Gold, 1986). This, together with other evidence, has been used to support the suggestion that the effects of cognition-enhancing drugs (nootropics) are mediated primarily by the stimulation of increases in blood glucose levels and in turn increased uptake and utilization of glucose by the brain (Wenk, 1989). There can be no doubt that starving the brain of glucose has severe effects; however, it is much less clear that raising blood glucose above the normal fasting or premeal levels recorded in the studies in Table 2 (i.e. not less than 4.5 mmol/l) will have a significant impact on cerebral metabolism. Lowering blood glucose leads rapidly to a substantial deterioration in cognitive performance, but typically this is observed only once the level has fallen to below about 3 mmol/l (e.g. Pramming *et al.* 1986; Richardson, 1990; Amiel, 1994). Therefore, hypoglycaemia sufficient to cause cognitive dysfunction would not normally be expected to occur in healthy individuals. In other words, under most circumstances improvement in performance following the consumption of a glucose drink, or after eating a meal or snack (see p. 445, p. 448 and Kanarek & Swinney, 1990), is unlikely to be due to any major alteration in the supply of glucose to the brain.

A further result reported in some of the studies summarized in Table 2 was that blood glucose levels or changes in blood glucose levels correlated significantly with one or more measures of performance. These observations have also been used to support the view that beneficial effects of glucose administration are due to direct effects on brain functioning (e.g. Benton & Owens, 1993; Benton *et al.* 1994; see also Benton & Sargent, 1992). Other aspects of the results, however, suggest an indirect relationship. For instance, positive correlations between performance and blood glucose levels measured before glucose administration, and also between performance and changes in blood glucose levels, were found both in the absence of a significant beneficial effect of the glucose drink and irrespective of which drink (glucose or placebo) the subjects had received. That is, the effect of the drink on blood glucose did not reliably predict performance and, at least from these studies, the conclusion must be that there is no enhancement of performance by glucose *per se*. Perhaps the most reasonable interpretation of the reported correlations is that high baseline levels of blood glucose or rapidly rising levels of blood glucose reflected increased sympathetic activity, including increased

secretion of adrenalin and cortisol, which in turn benefited performance. Although the physiological interactions which underlie this correlation between arousal (alertness) and performance (Johnson *et al.* 1992) may well include increased glucose uptake and utilization by the CNS (Wenk, 1989), there is no firm evidence that this is the mechanism which is primarily responsible for the performance-enhancing effects of stress, or the administration of either glucose or nootropic drugs.

#### CONCLUSIONS

The studies reviewed here have demonstrated acute effects on cognitive performance of certain dietary manipulations with varying degrees of relevance to everyday eating habits. Caffeine is consumed widely and frequently and has clear effects on performance, although certain results may have exaggerated the benefits of caffeine use because the possible negative consequences of caffeine withdrawal were not taken into account. Indeed, there is perhaps a more general tendency to overestimate the importance of the effects revealed by studies in this field, due in large part to the inclusion of multiple tests and multiple measures of performance. This arises because of a failure to define hypotheses predicting specific performance effects. As a result, undue weight is often given to measures revealing statistically significant effects, with little discussion of areas of performance which are unaffected. This bias is probably also compounded by the failure to report experiments which do not find any significant treatment effects. The paper by Azari (1991) is a notable exception in this respect. There is, nonetheless, accumulating evidence that meal size and meal composition can influence postprandial performance efficiency, although their exact impact is modified by a variety of factors including time-of-day and other contextual variables. In particular, effects are most evident under low levels of arousal. This is important because often the testing procedures used are mildly stressful and, therefore, may interfere with the very phenomena they are designed to measure. Finally, there is also a need for further investigation of the physiological mechanisms mediating the performance and mood effects of foods. In the case of carbohydrates the proposed mechanisms and predicted effects will differ according to whether performance is measured within the first 0.5 h after the 'meal', or after 7 or more hours. To date, however, most experimental studies have included performance assessments at intervals relevant to only one of these time-points.

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