

Beer, Carbohydrates and Diet

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ABSTRACT

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The enormous incidence of excess body weight in the population of the United States and the attendant risks that obesity brings has stimulated unprecedented interest in diets, especially those that do not leave an individual feeling hungry. In particular this has led to so-called ‘low carb’ diets. Beer has suffered unfairly through erroneous claims made in connection with at least one of these diets and has been unfairly categorised as being ‘high carb’. In the face of this – and despite the fact that the vast majority of beers contain low levels of so-called ‘carbs’ – there have been certain brands specifically branded as low carb products. Brewers intent on marketing products that may genuinely be considered to be part of a ‘calorie counting’ diet should focus on developing products of excellence that contain low levels of alcohol, the latter molecule being the major source of calories in most beers. They may also do more to press the claim of beer as being a source of ‘good carbs’, for the soluble fibre and prebiotic molecules that it contains and which are derived from the β -linked glucans and arabinoxylans that derive from the cereal cell walls.

Key words: Alcohol, calories, carbohydrate, diet, fibre, glycaemic, glycaemic index, glycaemic load, metabolism, obesity.

INTRODUCTION

Two thirds of Americans are now considered to be overweight²⁵, a state defined as having a Body Mass Index [BMI, i.e. weight (kg)/height squared (m^2)] in excess of 25. One third of Americans fall into the category of obese (BMI \geq 30). Of these, 3 million suffer from life-threatening obesity. Indeed, obesity has been identified as the second-leading cause of preventable deaths in the United States (after smoking). A comparable situation exists in other Western cultures²². The simple explanation for this is that these people consume more calories than they expend: they over-eat and under-exercise¹⁸.

TRENDY DIETS

This scenario is highly conducive to those seeking a marketing opportunity by promoting diets of diverse types. Perhaps the most publicised of these have been the so-called ‘low carb’ diets, especially that of Atkins⁴. Such

diets are not novel; indeed William Banting championed such an approach to dieting in the mid-19th century with his *Letter on corpulence*⁷. He claimed to have lost 46 pounds in a year without ever getting hungry.

The Atkins approach is that the dieter should severely restrict carbohydrate intake while eating more meat and other protein-rich, high-fat, high-calorie food. Part of the rationale is that the person will feel less hungry (and therefore tempted) than they would on an overall reduced calorie regime.

In view of their very high level of carbohydrates, cereals and cereal-derived products are to be avoided in the Atkins diet. This situation is surely peculiar when one considers that in some African and Asian countries people may eat 300 g milled cereals per day and obtain 80% of their calories from this source and yet excessive weight and obesity are rare exceptions in such societies. Indeed, those tempted to embrace such diets might heed Ornish²⁷ who, in observing that 70% of patients on an Atkins diet for 6 months were constipated, 65% had halitosis, 54% reported headaches, and 10% had hair loss, inferred that ‘you may lose weight and start to attract people but, when they get too close, it may be counterproductive.’

Atkins’ premise is that the body responds to high carbohydrate loading by increasing the production of the hormone insulin, which regulates blood sugar levels. This increase (and an ensuing enhanced resistance of the body to the impact of insulin) in turn encourages an increase in body weight.

The Atkins regimen has spawned a number of other related diets, perhaps the most publicised of which is the South Beach Diet¹. Agatston’s ‘refinement’ of the Atkins approach is that there are ‘good carbs’ and ‘bad carbs’ (indeed, also ‘good fats’ and ‘bad fats’). The South Beach thesis is that ‘good carbs’, namely the less digestible carbohydrate polymers, stop insulin resistance and cure cravings. ‘Bad carbs’ have the contrary effect, it is claimed. Leaving aside momentarily the precise accuracy of these statements, the premise is at least in part based on inaccurate reporting. Specifically in relation to beer, in *The South Beach Diet*, Agatston states

‘In general, though, because beer has the sugar maltose in it, it is by far the most fattening of all alcoholic beverages. Most alcoholic beverages when consumed with a meal help delay digestion and thereby have a favourable effect on the glycaemic index of the meal. The maltose in beer is digested more rapidly than any other food and causes large swings in blood sugar and insulin levels. This is the origin of a beer belly. We do not get wine bellies because wine does not contain maltose. Light beers with lower carbohydrate content are better than

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regular beers but I would be very careful and monitor my response.”

In this paragraph, Agatson totally ignores the role of fermentation in removing maltose. One major brewing company confronted that author over the claims and it appears that Agatson has recanted his position on his Web site. The actual words on the site now say

“This diet is constantly changing based on new research so some of the info in the book is out of date. One of those items, you will be happy to know, is the ban on beer.”

The notion of fermentation being “new research” is clearly incorrect, but at least a wrong is righted. One presumes this correction will also be made in revisions of Agatson’s plethora of publications and in any new books. However the claims have damaged sales of “standard” beers, at the same time repositioning certain light beers, triggering the emergence of “low carb” beers and advertising opportunities for spirits

It seems timely to review the *actual* situation with regard to the carbohydrate content of beer. Although there is as yet little published experimental data to underpin the statement, intuitively it seems that most consumers asked to place beer either in a “healthy” or “unhealthy” pigeon-hole would opt for the latter. Certainly there is little in the popular dietary press to give firm guidelines to consumers. Indeed, tables of food compositions (see for example²³) bracket all beers into one or at most two (“normal” and “light”) lines, whereas all manner of other foodstuffs have multiple entries, one for each brand. Turkey lunch meat, for example, warrants over nine pages worth of entries and even Trail Mix receives ten times more space than does beer. It is unhelpful that the consumer should be led to believe that a heavy stout has the same analytical composition as a high adjunct lager-style beer.

THE CARBOHYDRATE COMPOSITION OF BEER IN RELATION TO OTHER FOODSTUFFS

The Nutrition Labelling Regulations of the US Food and Drug Administration²⁴ state that *total carbohydrate must be calculated by subtraction of the weights of crude protein, total fat, moisture and ash from the total weight of food*. In other words, carbohydrate is determined by difference, which is an inherently inaccurate approach whenever any of the other components are present in large proportions.

Table I lists the published carbohydrate values for a range of foodstuffs (as typically listed in web pages and books of dietary information for the health conscious). Perhaps it is more informative to compare beer alongside other drinks (Table II). Spirits of course are logically zero in carbohydrate – but many drinkers do not partake of them alone, but rather with mixers such as ginger ale, cola, tonic or tomato juice, and the resultant drink is substantially more charged with carbohydrate than is beer.

Tables III and IV present more specific data for the carbohydrate content reported in different beers.

THE GLYCAEMIC INDEX

In order to bring some scientific rationale into the debate on carbohydrates of different types, the concept of the glycaemic index (GI) was introduced by Jenkins¹⁶. It is a ranking of carbohydrates based on their immediate effect on blood glucose (blood sugar) levels. It compares foods, gram for gram of carbohydrate. Carbohydrates that break down quickly during digestion have the highest GI’s and the blood glucose response is fast and high. Carbohydrates that break down slowly, releasing glucose gradually into the blood stream, have low GI’s. It has been argued that high glycemic foods, with the attendant hyperinsulinemia and insulin resistance, may contribute to the onset of diabetes, cardiovascular disease and cancer⁵. However, the American Institute for Cancer Research² draws attention to many of the uncertainties which exist in the claims made for diets such as The South Beach Diet, revealing the vagaries inherent in the methodology of Glycaemic Index measurement.

Table I. Total carbohydrate contents of various foodstuffs.

Food	Carbohydrate (g) per serving
Bagel	49
Banana	27
Bread (wheat)	12
Kit Kat	27
Corn flakes	24
Cookies (peanut butter)	12
Danish (fruit)	34
Grapes	29
Ice cream (vanilla)	16
Lentils (cooked)	40
Macaroni (cooked)	40
Melon	15
Milk	11
Noodles (chow mein)	26
Pie (apple)	58
Potato (baked)	34
Rice (cooked)	45
Soup (tomato)	22
Yogurt (plain)	16
Beer	10–20
Light and low carb beer	2.5–10

Table II. Total carbohydrate content of beverages.

Food	Carbohydrate (g) per serving
Coffee (brewed)	0.8
Cappuccino	23
Coffee liqueur	24
Tea (brewed)	0
Tea (iced, flavoured)	25
Pina colada	32
Dessert wine	14
Red wine	2
White wine	1
Sherry	5
Apple juice	29
Cola	40
Ginger ale	32
Grape soda	42
Tomato juice	10
Tonic water	30
Beer	10–20
Light and low carb beer	2.5–10

The interactions of foodstuffs individually and co-operatively with the body are complex and far from being able to be encapsulated in one or even a very few measurements such as the Glycaemic Index, even supposing that the quotient has merit. By way of illustration, it was demonstrated that the glycaemic index of a carbohydrate may play a role for cardiovascular risk factors but that there is so far no evidence that low-glycaemic index foods facilitate weight control³. Actually these authors say that intervention studies showed that sugar in *drinks* is more likely to produce weight gain than solid sugar in *solid foods*. However alcoholic beverages promote a positive energy balance, and wine may be *more* obesity-promoting than beer.

The general breakdown of GI is made as follows:

- Low GI = 55 or less
- Medium GI = 56–69
- High GI = 70 or more

Table V lists a selection of high GI foods, while Table VI gives a range of low GI foods. It will be noted that no GI is listed for beer. Although there are those who have reported values, it is generally agreed that it cannot be done. To measure GI, the individual is subjected to overnight starvation and then fed with 50 g of carbohydrate in 15 minutes before monitoring their blood glucose level. This is to be done in duplicate. To do this with beer would generally demand that the subject consume five or so regular beers in a 15 minute period, and as many as 15 or more light beers. It has been suggested that the GI test can be scaled down to a consumption of only 10 g, however it is less than clear that there is a linear relationship between GI and the amount of carbohydrate consumed.

Table III. Carbohydrate content of beers.

	% (w/v) as glucose
Total carbohydrate	0.28–6.1
Dextrin	0.7–3.9
Maltotriose	0.13–0.74
Maltose	0–0.2
Glucose	0–0.8
Fructose	0–0.55
b-Glucan	0.18–1.03

data from Buckee and Hargitt¹¹

Table IV. Sugar content of a range of beers.

Beer	Glucose	Maltose	Malto-triose	Malto-tetraose
Lager	0	0.008	0.001	0.019
Light lager	0	0.01	0.001	0.016
Lager	0.0024	0.018	0.034	0.022
Light lager	0.0320	0.019	0.015	0.016
Lager	0.0032	0.028	0.024	0.068
Light lager	0.0016	0.021	0.001	0.007
Lager	0	0.001	0.011	0.022
Lager	0.0024	0.023	0.044	0.026
Lager	0.0016	0.016	0.012	0.042
Pale ale	0.0008	0.003	0.002	0.077
Porter	0.0008	0.003	0.001	0.039
Stout	0.0012	0.002	0	0.052

All values in % (w/v) and derived from Thomas et al³¹

The concept of GI has been further refined into the so-called Glycaemic Load (GL). This takes into consideration the fact that some foods might contain a lot of carbohydrate but that carbohydrate does not have a major impact on blood glucose and vice versa. Conversely other foods might contain high GI carbohydrate, but only small amounts of it per serving.

$$GL = GI (\%) \times \text{grams of carbohydrate per serving}$$

One unit of GL approximates to the glycaemic effect of 1 gram of glucose

Low GL = 10 or less

Medium GL = 11–19

High GL = 20 or more

To illustrate, the GI of pumpkin is rather high at 76. However the sugar content per se is very low, so the GL is also small. By contrast long grain rice contains carbohydrate with a relatively low GI; however it is so rich in carbohydrate that the net GL is high. As we cannot strictly have a GI for beer, it is intuitively the case that its GL must be zero. However one web page (<http://www.montignac-intl.com/En/m7en.html>) makes the claim that beer has a GI of 110 and is “5 g of pure carbohydrate”. If this were true the GL would still only be 5.5.

Alcohol may actually lower glucose and insulin responses. Brand-Miller et al¹⁰ showed that if GI is measured after a person consumed bread accompanied by

Table V. Foods with high glycaemic index.

Food	Glycaemic index	Carbs (g per serving)
Instant rice	124	44
Corn Flakes	119	24
Rice Krispies	117	29
Jellybeans	114	27
French fries	107	53
Soda crackers	106	9
Potato (boiled/mashed)	104	27
White bread	100	13
Melba toast	100	4
Couscous	93	36
Ice cream	87	16
One minute oats	87	25
Digestive cookies	84	17
Table sugar	83	4

Table VI. Foods with low glycaemic index.

Food	Glycaemic index	Carbs (g per serving)
Popcorn	79	20
Oat bran bread	72	13
Parboiled rice	68	43
Pumpernickel	66	15
All-Bran	60	22
Sweet potato	54	28
Skim milk	46	12
Pasta	40 to 70	40
Baked beans	40 to 69	52
Apple/banana/plum	34 to 76	7–26

water, beer, wine or gin, then the presence of alcohol actually lowered the glucose levels.

LOW CARB BEERS

With the rapid emergence of low carb beers, the Alcohol and Tobacco Tax and Trade Bureau (TTB) have set interim standards for use of the words “low carbohydrate”: a beer must deliver no more than 7 grams of carbohydrate per serving. The clear risk in overtly marketing low carb beers, of course, is that, by inference, other “normal” beers must be “high carb”. Of course, they are not (see earlier).

COUNTING CALORIES

When people realize that the only sustainable and sensible way to lose weight and avoid weight gain is to avoid excess calorie intake while burning off surplus calories, they will re-focus on the calorie content of all foods, including beer.

The calorific value of beer is usually calculated from the equation

$$\text{Calories (kcal per 100 g)} = 6.9 (A) + 4 (B - C)$$

where A = alcohol (% by weight), B = real extract (% by weight) and C = ash (% by weight).

Alternatively the equation used is

$$\begin{aligned} \text{Calorific value (kcal/100 mL)} \\ = [\text{ethanol (g/100 mL)} \times 7] \\ + [\text{total carbohydrates (as glucose g/100 mL)} \times 3.75] \\ + [\text{proteins (g/100 mL)} \times 4] \end{aligned}$$

The latter formula reminds us all too forcibly that the carbohydrates in beer (usually low, see earlier) actually make a much lesser contribution to calories than does the alcohol (Fig 1). Beers genuinely targeted on the dieter would be from a low-protein grist, have all of the α -glucan converted into alcohol (i.e. superattenuated) and adjusted to the lowest alcohol content commensurate with other quality considerations, notably flavour. Brewers do not have the same opportunity that is available to other drinks producers (Table VII). Table VIII shows the carbo-

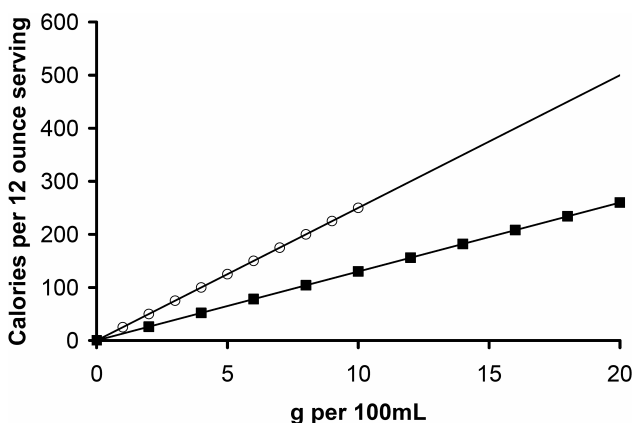


Fig. 1. The impact of levels of alcohol and carbohydrate on calorie content of beer. ○, alcohol; ■, carbohydrate.

hydrate, alcohol and calorie content for a range of beer styles.

HOW THE BODY DEALS WITH CARBOHYDRATES

The body must deal with different types of carbohydrate. For the purposes of this paper, I will restrict attention to those carbohydrates found in cereal and cereal products, notably beer.

Considering the starch of grain (for example that in bread), then its digestion commences in the mouth through the action of salivary α -amylase¹². Clearly, if the starch is not gelatinised then the extent of any action will be limited. Gelatinised starch, however, will be significantly converted to dextrans containing 8 to 10 glucosyl units by the salivary ptyalin.

The food passes into the stomach, the acid conditions of which preclude the action of enzymes other than pepsin (a proteinase). Indeed the acid will destroy or inhibit much enzymic activity introduced earlier in the digestive tract during mastication. The food passes to the small intestine (duodenum). The arrival of the partially-digested “chime” in the small intestine provokes the duodenal mucosa to produce the hormones secretin and cholecystokinin, which trigger the pancreas to release enzymes amidst alkaline juice into the intestinal lumen. Notable amongst the enzymes is pancreatic α -amylase and so the primary products at this stage are yet smaller dextrans of 3-5 glucosyls, together with isomaltose (the disaccharide comprising two glucosyls linked through a α 1→6 linkage) and some maltose. In the brush border membranes there is a further series of enzymes, including maltase, isomaltase and dextrinase, the combined action of which allows the total conversion to glucose, which is absorbed in the portal blood en route to the liver. However the time requirement for this means that oligomers and polymers of glucose have a far lesser impact on GI than has glucose per se. The residual starch-derived carbohydrate in beer –

Table VII. Carbohydrate and calories in standard and “diet” versions of beverages.

Product	Carbohydrates (g)	Calories
Cola (12 fl oz)	27	100
Diet cola (12 fl oz)	0	0
Beer (12 fl oz)	13	150
Low carb beer (12 fl oz)	<7	<100

Table VIII. Carbohydrate, alcohol and calorie content of different beer styles.

Beer style	Alcohol (% vol/vol)	Carbohydrate (g/12 fl oz)	Calories (per 12 fl oz)
Lager	5.0	10.6	143
Pale ale	5.6	12.3	200
Wheat beer	5.4	12.9	171
Light lager	4.2	6.6	110
Ice beer	5.5	8.9	148
Barley wine	9.6	24.6	285
Low alcohol beer	0.4	5.8	96
Stout	4.0	10.0	125
Low carb lager	4.2	2.6	95

viz. isomaltose and other dextrans – fall into this “slow release” category, and should thus be classified as “good carbs” according to the Agatson terminology¹.

HOW THE BODY DEALS WITH ETHANOL

Ethanol has a calorific value of 7.1 kcal/g, as opposed to carbohydrate which has a value of 3.75 kcal/g. In other words ethanol is an extremely rich energy source. In America it has been claimed that 5.6% of the total energy consumption is from ethanol, even though a third of the population is understood to be teetotal⁹.

The link between alcohol consumption and body weight “remains an enigma to nutritionists and in many instances paradoxical”¹⁷. There are many conflicting opinions concerning what this means for health and, most notably weight, although we can firmly refute the notion of beer comprising empty calories, as it contains significant levels of vitamins, antioxidants, minerals and fibre⁶.

Moderate alcohol consumption increases dietary uptake, the 24-h energy intake being higher on days when an aperitif is taken³³. However ethanol can cause satiety because it delays gastric emptying²¹.

Alcohol is for the most part taken up from the duodenum, into the portal vein, and transported to the liver. Here there are two pathways of ethanol metabolism. The primary route is by alcohol dehydrogenase, with the attendant reduction of NAD to NADH. In turn the acetaldehyde is oxidized to acetate by acetaldehyde dehydrogenase, again with the reduction of NAD. The acetate enters normal metabolic pools, for the most part outside the liver, and is ultimately converted to CO₂ and H₂O. The NADH enters into the respiratory chain through which it is regenerated and, accounting for draw off of a proportion of the ATP produced, the consequence of ethanol oxidation in this route is the production of 16 moles of ATP per mole of ethanol.

The second pathway is relatively unimportant in moderate ethanol users, but induced in chronic alcoholics²⁶. It is known as the microsomal ethanol oxidizing system (MEOS), and actually demands reducing power in the form of NADPH. As a consequence there is substantially less production of ATP (10 mol ATP/mol ethanol).

When moderate amounts of ethanol (5–10% of total daily calories) are added to a person’s diet, there is no change in the Resting Energy Expenditure. Rimpler²⁸ also showed that the efficiency with which the body uses alcohol in the maintenance of metabolisable energy is the same as for carbohydrate.

Subjects given 24 g ethanol displayed increased amounts of fat synthesis in the liver²⁹. It is suggested that, as ethanol is converted to acetate, the latter inhibits fat breakdown¹⁴. However, moderate ethanol drinkers (less than 50 g per day) actually show weight loss^{13,20}. It is believed that ethanol induces an increase in the sensitivity of muscle to insulin with a down-regulation of the effect of insulin on adipose tissue, so fat mass decreases¹⁹.

One can safely drink up to 30 g alcohol per day and avoid liver damage, independent of BMI and type of drink⁸.

But what about the beer belly? Wannamethee and Shaper³² conclude

“While metabolic studies indicate fairly unequivocally that alcohol consumption even in moderate amounts contributes to weight gain, the epidemiological evidence on the relationship between alcohol intake and body weight based on cross-sectional studies is conflicting... Findings... suggest that light to moderate drinking is not associated with weight gain but that heavier levels (>30 g alcohol per day) contribute to weight gain and obesity in men and women. Overall evidence from prospective studies supports the concept that alcohol is a risk factor for obesity, as one might expect if the energy derived from alcohol consumption was added to the usual dietary intake.”

Sonko et al³⁰ opined that alcohol has a fat-sparing effect, only causing fat gain when it is consumed in excess of normal energy needs.

β-LINKED CARBOHYDRATES IN BEER: AN OPPORTUNITY?

We have not yet considered one of the major carbohydrate families present in beer, namely the β-linked materials, derived from the β-glucan and arabinoxylan present in barley. Such molecules are neither metabolized by brewing yeast nor by the human body. Accordingly if they are present in beer they pass through the body to the large intestine. If they are high molecular weight (viscous) polymers inefficiently dealt with in the brewery (e.g. in brews from relatively under modified malt mashed-in at high temperatures) then, whilst likely having challenged the brewer for their impact on filtration rates, their presence in the final beer is favourable from a perspective of soluble fibre. If they are of low molecular weight – and it does appear that in malting and brewing sizeable proportions of the β-glucan and pentosan survive as oligosaccharides or disaccharides (such as cellobiose and laminaribiose) – then they are still highly likely to be of value as pre-biotics. These are substances that represent feedstock for the beneficial microflora of the lower gut. Already there is evidence that beers average around 2 g/L of soluble fibre, but that some contain three-fold more than this¹⁵, see also Table III). The Food and Drug Administration classifies a food as a good source of a nutrient if it provides 10% of the Daily Value (DV), i.e. the daily requirement for an individual. The DV figure used for labelling purposes in the case of fibre is 25 g per day, ergo some beers might quite clearly be ascribed as being a good source of this key dietary element. This whole area is clearly worthy of urgent study.

CONCLUSIONS

1. All beers can be taken in moderation as part of so-called “low carb” diets.
2. Beers overtly positioned as part of a “calorie-counting” diet should be of low alcohol content.
3. There is scope for positioning beers as being a source of “good carbs”, i.e. soluble fibre and pre-biotics.

REFERENCES

1. Agatston, A., *The South Beach Diet: Good Fats, Good Carbs Guide*. Rodale: New York, 2004.
2. American Institute for Cancer Research. "The Glycemic Index: What it is and what it is not" (http://www.aicr.org/press/Glycemic_Index.doc)
3. Astrup, A., Astrup, A., Buemann, B., Flint, A., and Raben, A., Low-fat diets and energy balance: how does the evidence stand in 2002? *Proc. Nutr. Soc.*, 2002, **61**, 299–309.
4. Atkins, R.C., *Dr Atkins' New Diet Revolution*. Avon Books: New York, 2002.
5. Augustin, L.S., Franceschi, S., Jenkins, D.J.A., Kendall, C.W.C. and La Vecchia, C., Glycemic index in chronic disease: a review. *Eur. J. Clin. Nutr.*, 2002, **56**, 1049–1071.
6. Bamforth, C.W., *Beer: Health and Nutrition*, Blackwell: Oxford, 2004.
7. Banting, W. *Letter on Corpulence*. Harrison: London, 1869.
8. Bellentani, S., Tiribelli, C. and Bedogni, G., Alcohol and nutrition as risk factors for chronic liver disease. In: *Nutrition and Alcohol: Linking Nutrient Interactions and Dietary Intake*, R.R. Watson and V.R. Preedy, Eds. CRC Press: Boca Raton, 2004, pp. 73–85.
9. Block, G., Dresser, C.M., Hartman, A.M. and Carroll, M.D., Nutrient sources in the American diet: quantitative data from the NHANES II survey. II. Macronutrients and fats. *Am. J. Epidemiol.*, 1985, **122**, 27–40.
10. Brand-Miller, J.C., Liu, V.K., Holt, S.H. and Liu, S.M., Alcoholic beverages lower acute glucose and insulin responses in healthy subjects. *Diabetes*, 2002, **51** (Suppl. 2), A399, 1637.
11. Buckee, G.K. and Hargitt, R., Measurement of residual carbohydrate in beer. *J. Inst. Brew.*, 1977, **83**, 275–278.
12. Coffee, C.J., *Metabolism*. Fence Creek Publishing: Madison, CT, 1998.
13. Colditz, G.A., Giovannucci, E., Rimm, E.B., Stampfer, M.J., Rosner, B., Speizer, F.E., Gordis, E. and Willett, W.C., Alcohol intake in relation to diet and obesity in women and men. *Am. J. Clin. Nutr.*, 1991, **54**, 49–55.
14. Crouse, J.R., Gerson, C.D., Decarli, L.M. and Lieber, C.S., Role of acetate in the reduction of plasma free fatty acids produced by ethanol in man. *J. Lipid Res.*, 1968, **9**, 509–512.
15. Gromes, R., Zeuch, M. and Piendl, A., Further investigations into the dietary fibre content of beers. *Brau. Int.*, 2000, **18**, 24–28.
16. Jenkins, D.J., Wolever, T.M., Taylor, R.H., Barker, H.M., Fielden, H., Baldwin, J.M., Bowling, A.C., Newman, H.C., Jenkins, A.L. and Hoff, D.V., Glycemic Index of foods: a physiological basis for carbohydrate exchange. *Am. J. Clin. Nutr.*, 1981, **34**, 362–366.
17. Jequier, E., Alcohol intake and body weight: a paradox. *Am. J. Clin. Nutr.*, 1999, **69**, 173–174.
18. Jequier, E. and Tappy, L., Regulation of body weight in humans. *Physiol. Rev.*, 1999, **79**, 451–480.
19. McCarty, M.F., The alcohol paradox. *Am. J. Clin. Nutr.*, 1999, **70**, 940–941.
20. Mannisto, S., Uusitalo, K., Roos, E., Fogelholm, M. and Pietinen, P., Alcohol beverage drinking, diet habits and body mass index in a cross-sectional survey. *Eur. J. Clin. Nutr.*, 1997, **51**, 326–332.
21. Mushambi, M.C., Bailey, S.M., Trotter, T.N., Chadd, G.D. and Rowbotham, D.J., Effect of alcohol on gastric emptying in volunteers. *Br. J. Anaesth.* 1994, **72**, 253.
22. Nammi, S., Koka, S., Chinnala, K.M. and Boini, K.M., Obesity: An overview on its current perspectives and treatment options. *Nutr. J.*, 2004, **3**, 3.
23. Netzer, C.T., *The Complete Book of Food Counts*. Dell: New York, 2003.
24. Nutrition Labelling Regulations of the US Food and Drug Administration (<http://vm.cfsan.fda.gov/label.html>)
25. Obesity.org (http://www.obesity.org/subs/fastfacts/obesity_what2.shtml)
26. Oneta, C.M., Lieber, C.S., Li, J., Ruttimann, S., Schmid, B., Lattmann, J., Rosman, A.S. and Seitz, H.K., Dynamics of cytochrome P4502E1 activity in man: induction by ethanol and disappearance during withdrawal phase. *J. Hepatol.*, 2002, **36**, 47–52.
27. Ornish, D., Was Dr Atkins Right? *J. Am. Diet. Assoc.*, 2004, **104**, 537–542.
28. Rumpler, W.V., Rhodes, D.G., Baer, D.J., Conway, J.M. and Seale, J.L., Energy value of moderate alcohol consumption by humans. *Am. J. Clin. Nutr.*, 1996, **64**, 108–114.
29. Siler, S.Q., Neese, R.A. and Hellerstein, M.K., De novo lipogenesis, lipid kinetics and whole-body lipid balances in humans after acute alcohol consumption. *Am. J. Clin. Nutr.*, 1999, **70**, 928–936.
30. Sonko, B.J., Prentice, A.M., Murgatroyd, P.R., Goldberg, G.R. van de Ven, M.L. and Coward, W.A., Effect of alcohol on post-meal fat storage. *Am. J. Clin. Nutr.*, 1994, **59**, 619–625.
31. Thomas, B.R., Brandley, B.K. and Rodriguez, R.L., Rapid analysis of saccharides in beer via fluorescence-assisted carbohydrate electrophoresis. *J. Am. Soc. Brew. Chem.*, 2000, **58**, 124–127.
32. Wannamethee, S.G., and Shaper, A.G., Alcohol, Overweight, and Obesity. In: *Nutrition and Alcohol: Linking Nutrient Interactions and Dietary Intake*, R.R. Watson and V.R. Preedy, Eds. CRC Press: Boca Raton, 2004, pp. 365–375.
33. Westerterp-Plantenga, M.S. and Verwegen, C.R.T., The appetizing effect of an aperitif in overweight and normal-weight humans. *Am. J. Clin. Nutr.*, 1999, **69**, 205–212.

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