

Effects of alcohol on blood glucose levels in people with type 1 diabetes:

A systematic review

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Article points

1. Three key themes were apparent from the studies on the effects of alcohol consumption on blood glucose levels in people with type 1 diabetes: hypoglycaemia; changes in glucose action; and impaired hormonal response.
2. Current literature regarding the effects of alcohol on glycaemic control in type 1 diabetes is conflicting.
3. There is a need for large-scale, valid randomised control trials focused on the effect of alcohol on blood glucose levels to improve evidence-based education for diabetic patients who drink alcohol.

Key words

- Type 1 diabetes
- Alcohol
- Glycaemia
- Systematic review

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This systematic review focuses on the effect of alcohol on blood glucose levels in patients with type 1 diabetes, aiming to investigate whether evidence from recent randomised controlled trials support the hypothesis that alcohol does not have an effect on glycaemic control. Current literature regarding the effects of alcohol on glycaemic control in type 1 diabetes is conflicting, and up-to-date, large-scale randomised control trials are scarce.

Diabetes mellitus affects an estimated 180 million people worldwide, a figure that is set to double by 2030, making diabetes a condition of epidemic proportions (Wild et al, 2004; Williams and Pickup, 2004; World Health Organisation, 2006). This article will focus on insulin dependent, or type 1, diabetes, which accounts for between 5% and 15% of the 2.3 million people with diabetes in the UK (Diabetes UK, 2006a).

A normal blood glucose level is between 3.5mmol/L and 6.0mmol/L (Williams and Pickup, 2004; Yamada et al, 2007), however when there are insufficient amounts of or no insulin released, as occurs in type 1 diabetes, blood glucose levels can rise causing long-term complications such as retinopathy, neuropathy and atherosclerosis (Diabetes UK, 2007; Day, 2002). Short-term health complications of type 1 diabetes include hypoglycaemia and diabetic ketoacidosis, which can lead to coma and death if untreated (Day, 2002).

In diabetes, alcohol appears to have hypoglycaemic-inducing properties, particularly in the fasting state (Christiansen et al, 1994; Emanuele et al, 1998; Howard et al, 2004; van de Wiel, 2004). It has also been shown that moderate to high alcohol consumption, above the recommended limits (*Table 1*), can lead to high long-term blood glucose levels, increasing the likelihood of neuropathy and retinopathy (Emanuele et al, 1998; Howard et al, 2004). In contrast, others in the literature state that a moderate amount of alcohol can have a beneficial effect on the cardiovascular system and endothelial cell health of people with diabetes (Howard et al, 2004; Shai et al, 2004). Ahmed et al (2006) found that alcohol consumption was associated with poor adherence to diabetes self-care behaviours – such as monitoring blood glucose and taking diabetes medication. In light of the damaging potential complications of poor glycaemic control it is important that the information received by people with type 1 diabetes is

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1. This systematic review focused around the research question: How does alcohol affect glycaemic control in people with diabetes mellitus?
2. Three main themes emerged from the review of the literature: (i) hypoglycaemia; (ii) changes in glucose action; and (iii) impaired hormonal response.
3. Both alcohol and hypoglycaemia have a marked effect on cognitive ability individually, more so when experienced together.

accurate, retainable and realistic to help maintain health alongside a near normal lifestyle as far as is possible.

Methods

This systematic review focused around the research question: How does alcohol affect glycaemic control in people with diabetes mellitus? The following keywords were used in a search of the databases British Nursing Index, Embase, MEDLINE, PsycINFO, CINAHL: “diabetes mellitus”, “glycaemic control”, “alcohol”, “insulin sensitivity”, “hormone” and “randomised controlled trial”.

Inclusion and exclusion criteria

The titles and abstracts of the studies derived from the database search were screened in accordance with the inclusion criteria base on the Cochrane Collaboration review structure guidelines (Cochrane Collaboration, 2007).

Studies selected investigated the effect of alcohol on type 1 diabetes or glycaemia. Subjects were not required to have diabetes mellitus but studies were required to refer to blood glucose level, glucose uptake or hormonal response to alcohol. Only randomised control trials were used to minimise selection bias. Only studies published after 1997 were used and no age- or gender-related criteria were applied.

An appraisal checklist by Urschel (2005) was used alongside a numerical scoring scheme by Jadad et al (1996) to systematically assess the methodological quality and the internal and external validity of the papers selected.

Results

The seven studies selected for review met all the inclusion criteria. All were related to diabetes, glycaemia or alcohol, all are randomised controlled trials and all were published within the previous 10 years.

This paper will focus on the important findings from the results of the systematic review and will discuss the three main themes that emerged: (i) hypoglycaemia; (ii) changes in glucose action; and (iii) impaired hormonal response.

(i) Hypoglycaemia

Turner et al (2001, study 2) concluded that: “In type 1 diabetes moderate consumption of alcohol in the evening may predispose patients to hypoglycaemia after breakfast the next morning.” Following breakfast at 8am, five of the six participants had symptomatic hypoglycaemia requiring treatment between 10am and midday, with a range of 1.9–2.9mmol/L.

Cheyne et al (2004, study 3) conclude that both alcohol and hypoglycaemia have a marked effect on cognitive ability individually, more so when experienced together. The study tested reaction time, trail making, digit symbol substitution, visual change detection, hazard perception and hypoglycaemia symptom scores. Hypoglycaemia and alcohol combined significantly impeded the ability of participants to perform well in these tests. The final test, a driving-related hazard perception test, was unaffected by alcohol, hypoglycaemia or both combined.

Cheyne et al (2004, study 3) also assessed participants according to hypoglycaemic symptoms. Both autonomic symptoms (palpitations, sweating, shaking, hunger) and neuroglycopenic symptoms (confusion, drowsiness, odd behaviour, speech difficulty, incoordination) increased significantly when alcohol and hypoglycaemia were experienced together. Alcohol reduced the ability of the subjects to recognise hypoglycaemia when it occurred; two of the 17 participants reported

Table 1. Recommended alcohol consumption for people with diabetes (Department of Health, 2006).	
Men	No more than 3–4 units* per day
Women	No more than 2–3 units* per day
*A unit of alcohol is defined as half a pint of ordinary strength beer, lager or cider, 125mL of wine or 25mL of spirits.	

hypoglycaemic symptoms when blood glucose was normal after having consumed alcohol.

Richardson et al (2005, study 4) conclude that: "A moderate amount of alcohol with an evening meal was associated with an increased risk of delayed hypoglycaemia." After an alcoholic drink participants were twice as likely to report hypoglycaemic symptoms. The mean number of participants reporting symptoms of hypoglycaemia with consumption of alcohol was 1.3, compared with 0.6 from the control group ($P=0.02$). Those who had consumed alcohol had an average interstitial blood glucose level 1.2mmol/L lower than that of the control group ($P=0.02$). While this varied in magnitude and time during the 24-hour study period, the prevailing blood glucose led to an increased risk of hypoglycaemia during the study period and through the next day.

(ii) Changes in glucose action

Wan et al (2005, study 5) studied rats, and found that whole-body insulin action was reduced following ethanol administration. It was also found that all rats receiving alcohol showed elevated alanine aminotransferase and aspartate aminotransferase levels, indicative of alcohol-induced liver injury. They also found that insulin-stimulated glucose disposal was significantly inhibited in rats receiving higher concentrations of alcohol. This was demonstrated *in vitro* in the isolated gastrocnemius muscle in rats receiving higher concentrations of alcohol, which showed significantly impaired insulin-induced glucose uptake.

Ismail et al (2006, study 6) found: "In an uncontrolled, social context, moderately heavy alcohol consumption appears to be associated with increased glycaemic variation but not with low glucose levels." Overall glucose levels between study and control periods did not vary significantly (11.8mmol/L versus 10.6mmol/L, respectively; $P=0.43$). No significant difference was found between the period of time that study and control groups spent at normal glycaemic (4–10mmol/L) or hyperglycaemic (>10mmol/L) levels.

(iii) Impaired hormonal response

Flanagan et al (1998, study 1) tested epinephrine, norepinephrine, cortisol and glucagon responses in people without diabetes following gin and carbohydrate-containing tonic, gin and carbohydrate-free tonic and carbohydrate-containing tonic alone. In terms of hormone response, Flanagan et al (1998, study 1) found that epinephrine, norepinephrine, cortisol and glucagon responses were similar between all groups overall. Growth hormone (GH) was significantly suppressed by drinking gin and both types of tonic.

Kerr et al (2007, study 7) compared alcohol and placebo consumption during normoglycaemic and hypoglycaemic states and found that in the hypoglycaemic study periods, glucagon response was not significantly different with alcohol or placebo. During a hypoglycaemic state GH response was significantly lower after alcohol consumption compared to placebo but there was no significant influence of either alcohol or hypoglycaemia independently upon peak cortisol levels.

Epinephrine and norepinephrine concentrations were similar in both studies. Insulin sensitivity in the study by Kerr et al (2007, study 7) was significantly reduced in the alcohol consuming groups. Following the consumption of alcohol a glucose infusion rate of 150mL/hour was required to maintain normoglycaemia, compared to a rate of 171.4mL/hour for the placebo ($P<0.05$). For hypoglycaemia, mean glucose infusion rate was 112.4mL/hour for the placebo group, compared to 91.5mL/hour for those that consumed alcohol ($P=0.0008$).

Discussion

The purpose of this review was to determine the effect that alcohol consumption has on blood glucose levels in people with type 1 diabetes. Three themes became apparent when looking at the findings from the chosen randomised controlled trials. These findings will be discussed in the context of the wider literature using the same thematic structure.

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1. The purpose of this review was to determine the effect that alcohol consumption has on blood glucose levels in patients with type 1 diabetes.
2. The literature suggests that alcohol effects glycaemia by: (i) inhibiting gluconeogenesis; (ii) altering carbohydrate absorption; and (iii) by suppressing growth hormone.
3. Those who had consumed alcohol had an average interstitial blood glucose level 1.2mmol/L lower than that of the control group.

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1. The literature suggests that alcohol affects glycaemia by: (i) inhibiting gluconeogenesis; (ii) altering carbohydrate absorption; and (iii) by suppressing growth hormone.
2. It is thought that alcohol causes hypoglycaemia by inhibiting gluconeogenesis as a result of its metabolism.
3. It is thought that hepatic gluconeogenesis is decreased by up to 45% following the consumption of alcohol.
4. Growth hormone levels appear to be lower during sleep after alcohol consumption.

Hypoglycaemia

The literature suggests that alcohol affects glycaemia by: (i) inhibiting gluconeogenesis; (ii) altering carbohydrate absorption; and (iii) by suppressing GH.

(i) Inhibition of gluconeogenesis

Turner et al (2001, study 2) found that both morning and post-meal blood glucose levels were lower following moderate alcohol consumption due to the inhibition of gluconeogenesis, as this is responsible for a significant proportion of glucose output in people with diabetes.

It is thought that alcohol causes hypoglycaemia by inhibiting gluconeogenesis as a result of its metabolism (Cryer, 1997). Alcohol is metabolised by alcohol dehydrogenase to acetaldehyde and then to acetate by aldehyde dehydrogenase, which depletes hepatic levels of nicotinamide-adenine dinucleotide, a critical factor in the gluconeogenic pathway (Cryer, 1997; MacDonald, 1999). It is thought that hepatic gluconeogenesis is decreased by up to 45% following the consumption of alcohol (Meeking and Cavan, 1997; van de Wiel, 2004).

(ii) Altered carbohydrate absorption

Meeking and Cavan (1997) also discuss the possibility of altered carbohydrate absorption as a result of alcohol, stating that alcohol accelerates the rate of carbohydrate absorption in the human small intestine. Conversely, other studies found that alcohol absorption was significantly slowed following a solid meal, particularly containing carbohydrate (Paton, 2005; Horowitz et al, 1989). Diabetes UK (2006b) advise having a carbohydrate-containing meal or snack prior to alcohol consumption to prevent hypoglycaemia, in spite of there being conflicting evidence surrounding this.

(iii) Suppression of growth hormone

In addition to the finding that alcohol consumption leads to lower blood glucose

levels the following morning, Turner et al (2001, study 2) found that GH levels appeared to be lower during sleep after alcohol consumption. Although this was not statistically significant ($P=0.15$), it may be clinically significant due to the role of GH in the hypoglycaemic pathway, particularly in light of the reduced rate of gluconeogenesis, which would lessen hypoglycaemia recovery following alcohol. Acute hypoglycaemia stimulates the secretion of GH as it acts to limit utilisation of glucose and results in raised blood glucose levels (Cryer, 1997; Smith and Thorner, 2000). GH and other counterregulatory hormones will be discussed further in the section on impaired hormonal response.

Cognitive effects of hypoglycaemia and alcohol

Cheyne et al (2004, study 3) remark on the cognitive effect that both hypoglycaemia and alcohol have on people with type 1 diabetes. Discussion centred around the impact on diabetes education in relation to driving, stating that while people with diabetes who drive are no more likely to have an accident than drivers without diabetes, past studies have reported progressive deterioration in driving ability as blood glucose levels fall. Study participants did not perceive a greater risk when driving following alcohol with hypoglycaemia compared with alcohol and normoglycaemia. However, data from Cheyne et al's study have shown the combination of hypoglycaemia and alcohol to be the most detrimental to driving ability.

While Cheyne et al (2004, study 3) found that people were less likely to report hypoglycaemic symptoms following alcohol consumption, Richardson et al (2005, study 4) found that their study participants were twice as likely to report hypoglycaemic symptoms following alcohol consumption. The conflicting findings here highlight the need for more research to provide a clearer understanding of this relationship.

Richardson et al (2005, study 4) found that average interstitial blood glucose was

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1. Alcohol's hypoglycaemic properties can impair counterregulatory hormonal responses to alcohol consumption. This can lead to onlookers misconstruing symptoms of hypoglycaemia as intoxication and thus react inappropriately.
2. Alcohol appears to result in hypoglycaemia primarily due to its role in the inhibition of gluconeogenesis. This may be due to (i) decreased insulin sensitivity, and (ii) impaired self-care behaviours.
3. Ismail et al (2006) found that moderate alcohol consumption was associated with increased glycaemic variation but not with low glucose levels.
4. Meeking and Cavan (1997) suggest that alcohol consumption can also affect glycaemic control indirectly by missing meals, reducing the person's ability to accurately administer medications and reducing the ability to accurately balance carbohydrate and exercise.

1.2mmol/L lower with alcohol than with placebo ($P=0.02$). The authors describe a lack of consistent advice given to people with diabetes with regards to alcohol and glycaemic control. The authors discuss the hypoglycaemic properties of alcohol, which impairs counterregulatory hormonal responses to alcohol consumption, and the possible misunderstanding of onlookers who may misconstrue symptoms of hypoglycaemia as intoxication and react inappropriately (Richardson et al, 2005, study 4). Richardson et al (2005) aim to provide information to healthcare professionals who educate people with diabetes, and people with diabetes themselves, about the potential risk periods for hypoglycaemia following alcohol consumption. The conclusion drawn from the data is that hypoglycaemia can occur at any point in the 24-hour period after alcohol consumption. Richardson et al (2005) conclude that informing people with diabetes about the prolonged hypoglycaemic effect of alcohol will enable them to adjust insulin dosage accordingly, with the aim of reducing the risk of hypoglycaemia following alcohol consumption.

Gibson and Tinggen (1998) advise drinking alcohol with diet or low calorie mixers containing fructose or saccharin, as it was found that alcohol had a significant hypoglycaemic effect when drunk with mixers containing glucose or saccharose. They also speculate that this is because alcohol potentiates the insulin response to sucrose ingestion, thus promoting a delayed and prolonged hypoglycaemia. Despite the original study cited by Gibson and Tinggen (1998) being over 20 years old at the time of publishing, this is used as evidence to support their recommendations, and concurs with current advice given by Diabetes UK (2006b). Perhaps there is a need for new research to confirm that this advice remains appropriate.

Changes in glucose action

Alcohol appears to result in hypoglycaemia primarily due to its role in the inhibition of

gluconeogenesis (Cryer, 1997; van de Wiel, 2004). This may be due to (i) decreased insulin sensitivity, and (ii) impaired self-care behaviours.

(i) Insulin sensitivity

Wan et al (2005, study 5; performed in rats) found that alcohol inhibited insulin-stimulated glucose uptake. The authors discussed the role of Gs-alpha in insulin-induced glucose uptake and alcohol's action within this, concluding that Gs-alpha has an effect on alcohol-induced insulin resistance via an unclear mechanism requiring further study. Gs-alpha is a member of the G proteins, a group of trans-membrane signalling molecules that allow interactions between cell-surface receptors and their specific effector molecules (Ashley et al, 1987).

Wan et al (2005, study 5) found that whole body insulin action was reduced following ethanol administration and that insulin-stimulated glucose disposal was significantly inhibited in the rats fed with high levels of alcohol. These findings have an important health implication for people with diabetes; if cells are unable to take up glucose, blood glucose becomes elevated, chronic hyperglycaemia ensues, which can lead to serious health complications. However, trials in humans are needed.

(ii) Self-care behaviours

Ismail et al (2006, study 6) found that moderate alcohol consumption was associated with increased glycaemic variation but not with low glucose levels. Participants consuming alcohol had a higher percentage of normal blood glucose readings and significantly less time with hypoglycaemia than after consuming placebo, contrary to the findings of Turner et al (2001, study 2) and Richardson et al (2005, study 4).

Meeking and Cavan (1997) suggest that alcohol consumption can also affect glycaemic control indirectly by missing meals, reducing the person's ability to accurately administer medications and

1. Ahmed et al (2006) discuss the importance of maintaining a balance between the cardiovascular benefits of alcohol and the potential for poor adherence to self-care behaviour, citing diabetes healthcare providers as important in assisting patients to preserve this balance.
2. There is a need for accurate and realistic education given to people with diabetes about how to consume alcohol safely.
3. The findings of this current review have highlighted the risk of prolonged hypoglycaemia up to 24 hours after alcohol consumption. This information would enable people with diabetes to adjust insulin dosage and prevent delayed hypoglycaemia. In addition, long-term alcohol consumption and alcoholism in people with diabetes requires close monitoring and support to minimise long-term health risks.

reducing the ability to accurately balance carbohydrate and exercise. Johnson et al (2000) concur with this view, finding that people who had consumed alcohol within 30 days of the study were less likely to adhere to dietary or medication regimens and consequently had higher blood glucose levels. Similarly, Ahmed et al (2006) found that alcohol was associated with poor adherence to self-care behaviours such as blood glucose monitoring, taking medication and maintaining a healthy diet and exercise regimen. Ahmed et al (2006) discuss the importance of maintaining a balance between the cardiovascular benefits of alcohol and the potential for poor adherence to self-care behaviour, citing diabetes healthcare providers as important in assisting people to preserve this balance.

In individuals without diabetes counter-regulatory hormones such as epinephrine, norepinephrine, cortisol, growth hormones and glucagon are secreted when blood glucose reaches around 3.8mmol/L to stimulate biological mechanisms to raise blood glucose (Cryer, 1997; Ross et al, 2005). In people with diabetes however, this may be different due to altered glycaemic thresholds dependent on prior glycaemic control (Ross et al, 2005). Glucagon, epinephrine and norepinephrine are key hormones in the rapid response to hypoglycaemia (Cryer, 1997; Marieb 2004; Ross et al, 2005). Cortisol and growth hormone offer a more prolonged response to hypoglycaemia by limiting glucose utilisation and promoting glucose production in the liver, giving what is effectively an insulin resistant state (Cryer, 1997).

These findings can be linked with Turner et al (2001, study 2) and Richardson et al (2005, study 4) as prolonged suppression of growth hormone following alcohol consumption may explain why people report hypoglycaemia the morning after a night of drinking alcohol (Christiansen et al, 1994; Emanuele et al, 1998; Howard et al, 2004; Van de Wiel, 2004). The advice given to people with diabetes wishing to drink alcohol in the form of spirits is to use low-

calorie mixers (Diabetes UK, 2006b), this coincides with the findings of Flanagan et al (1998, study 1), which showed that blood glucose levels were higher following gin and carbohydrate-free tonic, thus reducing the risk of hypoglycaemia.

There is little evidence to suggest that alcohol should be totally avoided by people with diabetes. However, studies highlight that alcohol may have an effect on blood glucose both in the short-term, via hypoglycaemia, and in the long-term, by reducing adherence to diabetes regimens, and therefore, linking to increased risk of diabetic complications such as retinopathy and neuropathy.

There is a need for accurate and realistic education given to people with diabetes about how to consume alcohol safely. Current advice issued by Diabetes UK (2006b) would appear to fit with the wider evidence, by emphasising the potential for hypoglycaemia for some time after alcohol consumption.

Conclusion

The findings of the studies in this review were conflicting. Three of the seven studies found alcohol to induce hypoglycaemia via various mechanisms while conversely, two of the seven studies found that alcohol was associated with impaired glucose uptake and consequently more variable blood glucose levels.

This systematic review found that there were few recent, large-scale randomised controlled trials available. There is a need for more valid, up-to-date studies into the subject to ensure an accurate and appropriate evidence base for current alcohol-related diabetes education.

The findings of this current review have highlighted the risk of prolonged hypoglycaemia up to 24 hours after alcohol consumption. This information would enable people with diabetes to adjust insulin dosage and prevent delayed hypoglycaemia. In addition, long-term alcohol consumption and alcoholism in people with diabetes requires close monitoring and support to minimise long-term health risks. ■

- Ahmed AT, Karter AJ, Liu J (2006) Alcohol consumption is inversely associated with adherence to diabetes self-care behaviours. *Diabetic Medicine* 23: 795–802
- Ashley PL, Ellison J, Sullivan KA, Bourne HR, Cox DR (1987) Chromosomal Assignment of the Murine Gi-alpha and Gs-alpha Genes: Implications For The Obese Mouse. *Journal of Biological Chemistry* 262: 15299–301
- Cheyne EH, Sherwin RS, Lunt MJ, Cavan DA, Thomas PW, Kerr D (2004) Influence of alcohol on cognitive performance during mild hypoglycaemia; implications for Type 1 diabetes. *Diabetic Medicine* 21: 230–7
- Christiansen C, Thomsen C, Rasmussen O, Hauerslev C, Balle M, Hansen C, Hermansen K (1994) Effect of alcohol on glucose, insulin, free fatty acid and triacylglycerol responses to a light meal in non-insulin-dependent diabetic subjects. *British Journal of Nutrition* 71: 449–54
- Cochrane Collaboration (2007) What is a Cochrane Review? Available at: <http://www.cochrane.org/reviews/revstruc.htm#TOC> (accessed 08.07.08)
- Cryer PE (1997) *Hypoglycemia: Pathophysiology, Diagnosis, and Treatment*. Oxford University Press, Oxford
- Day JL (2002) *Living with Diabetes, 2nd edn*. John Wiley & Sons, Chichester
- Department of Health (2006) *How Much Is Too Much? Drinking, You and Your Mates*. Department of Health Publications, London
- Diabetes UK (2006a) *What is Diabetes?* Available at: http://www.diabetes.org.uk/Guide-to-diabetes/What_is_diabetes/What_is_diabetes/ (accessed 16.09.08)
- Diabetes UK (2006b) *Enjoying Yourself at Christmas: Topsy Tips*. Available at: http://www.diabetes.org.uk/en/Guide-to-diabetes/Guide-to-Diabetes-extra-pages/Christmas-2008/Enjoying_yourself_at_Christmas/Tipsy_tips/ (accessed 16.02.07)
- Diabetes UK (2007) *Introducing to Diabetes*. Available at: http://www.diabetes.org.uk/Guide-to-diabetes/What_is_diabetes/ (accessed 15.08.07)
- Emanuele NV, Swade TF, Emanuele MA (1998) Consequences of Alcohol Use in Diabetics. *Alcohol Health and Research World* 22: 211–9
- Flanagan D, Wood P, Sherwin R, Debrah K, Kerr D (1998) Gin and Tonic and Reactive Hypoglycemia: What Is Important – the Gin, the Tonic or Both? *Journal of Clinical Endocrinology and Metabolism* 83: 796–800
- Gibson EM, Tinggen MS (1998) Nursing care for diabetic patients with alcohol induced hypoglycemia. *Journal of Emergency Nursing* 24: 165–8
- Horowitz M, Maddox A, Bochner M, Wishart J, Bratasiuk R, Collins P, Shearman D (1989) Relationships between gastric emptying of solid and caloric liquid meals and alcohol absorption. *American Journal of Physiology* 257: 291–8
- Howard AA, Arnsten JH, Gourevitch MN (2004) Effect of alcohol consumption on diabetes mellitus: a systematic review. *Annals of Internal Medicine* 140: 211–24
- Ismail D, Gebert R, Vuillermin PJ, Fraser L, McDonnell CM, Donath SM (2006) Social consumption of alcohol in adolescent diabetics is associated with increased glucose lability, but not hypoglycaemia. *Diabetic Medicine* 23: 830–3
- Jadad AR, Moore A, Carrol D, Jenkinson C, Reynolds DJM, Gavanaghan DJ, McQuay HJ (1996) Assessing the quality of reports of randomised clinical trials: Is blinding necessary? *Controlled Clinical Trials* 17: 1–12
- Johnson KH, Bazargan M, Bing EG (2000) Alcohol consumption and compliance among inner-city minority patients with type 2 diabetes mellitus. *Archives of Family Medicine* 9: 964–70
- Kerr D, Cheyne E, Thomas P, Sherwin R (2007) Influence of acute alcohol ingestion on the hormonal responses to modest hypoglycaemia in patients with Type 1 diabetes. *Diabetic Medicine* 24: 312–6
- MacDonald I (1999) *Health Issues Related to Alcohol Consumption, 2nd ed*. Blackwell Publishing, Oxford
- Marieb EN (2004) *Human Anatomy and Physiology, 6th ed*. Pearson Benjamin Cummings, San Francisco
- Meeking DR, Cavan DA (1997) Alcohol Ingestion and Glycaemic Control in Patients with Insulin-dependent Diabetes Mellitus. *Diabetic Medicine* 14: 279–83
- Paton A (2005) Alcohol in the body. *British Medical Journal* 330: 85–7
- Richardson T, Weiss M, Thomas P, Kerr D (2005) Day after the night before. Influence of evening alcohol on risk of hypoglycaemia in patients with type 1 diabetes. *Diabetes Care* 28: 1801–2
- Ross LA, Warren RE, Kelnar CJH, Frier BM (2005) Pubertal stage and hypoglycaemia counterregulation in type 1 diabetes. *Archives of Disease in Childhood* 90: 190–4
- Shai I, Rimm EB, Schulze MB, Rifai N, Stampfer MJ, Hu FB (2004) Moderate alcohol intake and markers of inflammation and endothelial dysfunction among diabetic men. *Diabetologia* 47: 1760–7
- Smith RG, Thorner MO (eds) (2000) *Human Growth Hormone: Research and Clinical Practice*. Humana Press, Totowa
- Turner BC, Jenkins E, Kerr D, Sherwin R, Cavan DA (2001) The Effect of Evening Alcohol Consumption on Next-Morning Glucose Control in Type 1 Diabetes. *Diabetes Care* 24: 1888–93
- Urschel J (2005) How To Analyze an Article. *World Journal of Surgery* 29: 557–60
- van de Wiel A (2004) Diabetes Mellitus and alcohol. *Diabetes Metabolism Research and Reviews* 20: 26–7
- Wan Q, Liu Y, Guan Q, Gao L, Lee KO, Zhao J (2005) Ethanol Feeding Impairs Insulin-Stimulated Glucose Uptake in Isolated Rat Skeletal Muscle: Role of Gs and cAMP. *Alcoholism: Clinical and Experimental Research* 29: 1450–6
- World Health Organization (2006) *Diabetes*. Available at: <http://www.who.int/mediacentre/factsheets/fs312/en/> (accessed 15.08.07)
- Wild S, Roglic G, Green A, Sicree R, King H (2004) Global Prevalence of Diabetes. Estimates for the year 2000 and projections for 2030. *Diabetes Care* 27: 1047–53
- Williams G, Pickup JC (2004) *Handbook of Diabetes, 3rd edn*. Blackwell Publishing, Oxford
- Yamada K, Milbrandt EB, Moore J (2007) Intensive insulin therapy in the medical ICU – not so sweet? *Critical Care* 11: 311