Introduction

Socio-cultural issues can impact on diabetes self-care resulting in erratic and poor glycaemic control. One social activity that can have detrimental effects on blood glucose homeostasis is the consumption of alcoholic beverages. In 2017, 57% out of 7100 adults in the general population of Great Britain had consumed alcohol in the previous week. Furthermore, in 2016, 26% of all Scottish adults had drunk at hazardous or harmful levels (>14 units per week), with males consuming significantly more alcohol on their heaviest drinking day than females. In particular, among the 16–24 year group men consumed 12.1 units vs 9.3 units in females on the heaviest drinking day, which is the highest consumption out of all age groups. In people with type 1 diabetes mellitus (T1DM), the intake and pattern of drinking influence the risk of life-threatening hypo- or hyperglycaemia. Therefore, for young people (YP) with T1DM it could be presumed that abstention from alcohol would be the preferred choice, as hypoglycaemia is the most feared and anxiety-provoking challenge of having diabetes. However, data suggest the contrary, in that adolescents with T1DM have similar or only slightly lower rates of participation in alcohol consumption compared to peers without diabetes. In one study of 17–18 year olds, reported drunkenness was higher in the group with T1DM vs those without T1DM (37.4% vs 18.9%), with significantly higher alcohol consumption over the previous year (78.3% vs 64.6%).

Despite the reported excess consumption in YP, this age group has a lower weekly intake compared to other age groups, with a tendency towards binge drinking. This could...
be due to the increasing prevalence of recreational drug use which, for a person with T1DM, can have a lesser impact on blood glucose levels, especially overnight and the following day, with hyperglycaemia being the most common effect.

To date there are no systematic reviews that synthesise the research evidence on the acute effects of alcohol on blood glucose related to specific alcoholic beverages. There is a gap in the existing literature regarding the impact on blood glucose and potential self-care strategies to overcome glucose challenges in real life.

Aims

The purpose of this narrative review is to investigate the acute effect of alcoholic beverages on blood glucose, and to use this evidence to recommend self-care advice for people with T1DM to help them maintain safe glycaemic control.

Methods

A systematic search of eight bibliographic databases was performed during October and November 2015 and updated in June 2019 using: Applied Social Sciences Index and Abstracts (ASSIA); Cochrane Controlled Trials Register; Cumulative Index to Nursing and Allied Health Literature (CINAHL); Embase; MEDLINE; PsycINFO; PubMed; and Social Science Citation Index (SSCI).

The following search terms were used: ‘Type 1 diabetes’ and ‘Alcohol drinking. Alcohol consumption or Alcoholsim’ and ‘Alcoholic beverage, Beer, Wine, Alcopops, or Spirits’ and ‘Blood glucose monitoring’ and ‘Self-care’ and ‘Hyperglycaemia’ and ‘Hyperglycaemia’ and ‘Patient education’ and ‘Health intervention’ and ‘Psychology’ and ‘Social/Community networks’. The search strategy included MeSH and terms were truncated with (*) to allow for multiple spellings and endings. The search identified 1029 records and titles screened, 273 abstracts were screened, and 115 full-text articles were assessed for eligibility.

Inclusion criteria

Results were limited to: (A) type 1 diabetes; (B) individuals over 18 years; (C) alcohol consumption; (D) all types of insulin administration or insulin regimen; (E) effects on blood glucose control, behaviour, and self-care strategies; and (F) English language.

Exclusion criteria

The exclusion criterion were: (A) studies which included other types of diabetes rather than type 1, because these individuals may have different pathophysiology and subsequent response to alcohol consumption; (B) children under 18 years of age; (C) articles which only reported basic science data, where the findings could not be translated into self-care and real-life; (D) participants with alcohol dependence or who have alcohol-related health problems; (E) studies examining the effects on long-term behaviour and glycaemic control, because the present study was concerned only with the acute response to alcohol consumption; and (F) animal studies.

Effects of acute alcohol consumption on blood glucose

The search resulted in three review publications, 15 original research publications, and 10 related articles which are included in this review.

In 2018, one systematic review of 13 studies investigating alcohol, plasma glucose and hyperglycaemia found mixed effects of alcohol on glucose. However, the majority of studies found that alcohol was associated with an increased risk of hyperglycaemia (eight studies). Potential mechanisms included impaired hepatic gluconeogenesis, impaired growth hormone response, and alcohol-induced impaired awareness of hyperglycaemia. The time of increased hyperglycaemia risk was concluded to be 8–12 hours after consumption, with no risk in the shorter term.

An earlier review in 2017 described similar physiological mechanisms, namely alcohol-induced inhibition of gluconeogenesis and growth hormone secretion, as well as increased lipolysis with impaired fatty acid oxidation. The delayed onset of hyperglycaemia at 8–12 hours was also found. In addition, it was suggested that early death in YP can be related to alcohol and drug use with a mortality risk greater in YP with diabetes than in those without. Although in this review hyperglycaemia was not described, it was identified that people consume pre-mixed and sweetened drinks to counter the increased hypoglycaemia risk. Also discussed was the risk of ketosis which is seen in both people with and without diabetes due to increased lipolysis after consuming alcohol.

In patient guidelines, hyperglycaemia as a consequence of drinking alcoholic beverages is not commonly acknowledged, despite studies demonstrating hyperglycaemia rather than hypoglycaemia. Furthermore, in clinical practice and experience, hyperglycaemia is observed to occur, which may be a result of a hypoglycaemia prevention strategy, with carbohydrate consumption being used deliberately to cause hyperglycaemia. Hypoglycaemia is the greatest risk of alcohol consumption described in guidelines for people with T1DM written by various different ‘diabetes associations’ (n=13). Recommendations mainly promoted alcohol avoidance and food consumption. General hypoglycaemia prevention strategies for only light alcohol consumption were described with no recommendations for binge drinking or consumption of moderate to high amounts of alcohol.

Glycaemic effects of alcoholic beverages, food consumption, and insulin administration in T1DM

Study findings regarding the effects of alcohol on glycaemia predominantly refer to hypoglycaemia. However, alcoholic beverages vary considerably with the carbohydrate and glucose content, the alcohol by volume (ABV) of the drink, plus volume/amount of drink consumed, as shown in Table 1.12 When considering physiological effects on glucose and alcohol metabolism, this could be assumed to cause variability in glucose concentrations. It has been suggested that alcohol has no effect on insulin requirements and external influences must affect the risk of hypo- and hyperglycaemia.

In one study, simulated social drinking conditions using a vodka aperitif, evening meal with wine, followed by cognac resulted in superimposable blood glucose levels and insulin profiles in the control and alcohol groups with no impact.
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Glycaemic findings were demonstrated in the afternoon when low carbohydrate beer was consumed without food, although data collection was only for 3 hours. In comparison, consuming higher carbohydrate beer with low alcohol ABV showed less hypoglycaemia at breakfast the following day.

Vodka. Spirits usually lower the blood glucose. This is dependent on the mixer drink and if sugar-free or glucose-containing drinks are used. One study used an adjusted insulin/glucose clamp to prevent hypoglycaemia occurring. The impact of vodka on interstitial glucose has also been explored and twice as many episodes of hypoglycaemia were reported by other authors.

To explore possible reasons for these differences, the evidence regarding glycaemic risks for specific alcoholic beverages is discussed below, based on the results of 15 studies of participants with T1DM, greater than one year duration, hypoglycaemia awareness, and consuming weekly alcohol. Study characteristics are shown in Appendix 1 (available in Practical Diabetes online at www.practicaldiabetes.com).

### Hypoglycaemia

**Beer.** Low carbohydrate beer with high ABV, in conjunction with an evening meal, has been shown to increase hypoglycaemia at bedtime and at breakfast. Comparable glycaemic findings were demonstrated in the afternoon when low carbohydrate beer was consumed without food, although data collection was only for 3 hours. In comparison, consuming higher carbohydrate beer with low alcohol ABV showed less hypoglycaemia at breakfast the following day.

**Vodka.** Spirits usually lower the blood glucose. This is dependent on the mixer drink and if sugar-free or glucose-containing drinks are used. One study used an adjusted insulin/glucose clamp to prevent hypoglycaemia occurring. The impact of vodka on interstitial glucose has also been explored and twice as many episodes of hypoglycaemia were self-reported after vodka during the following 24-hour period, especially the following morning, although no episodes requiring third-party assistance occurred.

**White wine.** White wine consumed after the evening meal rather than with food showed no significant difference in blood glucose during the evening or overnight, after a reduction of 70% mealtime soluble insulin. However, the fasting, morning and post-breakfast levels were significantly lower, requiring hypoglycaemia treatment after breakfast despite the insulin reduction. There was also a significant fall in growth hormone secretion between midnight and 4am after wine consumption. Other studies of wine did not continue data collection overnight, hence delayed hypoglycaemia effects cannot be compared.

### Strategies to counterbalance the hypoglycaemia risk

Carbohydrate food consumption before and after drinking, the inclusion of some sweetened mixer drinks, reduced insulin doses and reduced physical exercise would be strategies to prevent hypoglycaemia. The consumption of higher fat content foods to delay carbohydrate/glucose absorption and possibly increase blood glucose levels when hypoglycaemia is a risk could be considered. For evening drinking with sugar-free alcoholic beverages, taking these before bed will give a delayed glucose absorption to counteract early morning hypoglycaemia. For insulin adjustment, modest amounts of glucose-free alcohol ingestion consumed with food can increase the risk of immediate hypoglycaemia and bolus insulin may need to be reduced by up to 70%. For evening drinking, delayed hypoglycaemia is also a risk, and overnight basal insulin may need to be reduced. Another important factor is to check blood glucose in the morning at breakfast time, and also possibly reduce breakfast bolus insulin.

### Hyperglycaemia

**Beer.** In one study, ordinary beer that had a high glycaemic index (comparable to white bread), required prandial insulin; however, as the study duration was limited to 3 hours, the delayed effect was not studied. Bolus insulin may be required with ordinary and high carbohydrate beer with low <5% ABV, as hyperglycaemia may occur after consumption.

**White wine.** White wine consumed at lunchtime showed no impact on blood glucose, but there was a significant rise in post-prandial β-hydroxybutyrate levels, which remained elevated for 4 hours after food consumption. The carbohydrate type was pasta, which can have a delayed absorption with consequently delayed hyperglycaemia that requires insulin. The ketone levels were statistically significant but not clinically significant or probably relevant for patient education, which may be due to the impaired oxidation of fatty acids and the increased amount of alcohol.

### Table 1. Alcoholic beverages, with alcohol by volume (ABV) and units of alcohol

<table>
<thead>
<tr>
<th>Drink type, volume (ml) and ABV (10ml or 8g pure alcohol = 1 unit)</th>
<th>Units of alcohol per drink</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single small shot of spirits (25ml, ABV 40%)</td>
<td>1 unit</td>
</tr>
<tr>
<td>Single large shot of spirits (35ml, ABV 40%)</td>
<td>1.4 units</td>
</tr>
<tr>
<td>Alcopop (275ml, ABV 5.5%)</td>
<td>1.5 units</td>
</tr>
<tr>
<td>Small glass of red/white/rose wine (125ml, ABV 12%)</td>
<td>1.5 units</td>
</tr>
<tr>
<td>Standard glass of red/white/rose wine (175ml, ABV 12%)</td>
<td>2.1 units</td>
</tr>
<tr>
<td>Large glass of red/white/rose wine (250ml, ABV 12%)</td>
<td>3 units</td>
</tr>
<tr>
<td>Bottle of lager/beer/cider (330ml, ABV 5%)</td>
<td>1.7 units</td>
</tr>
<tr>
<td>Can of lager/beer/cider (440ml, ABV 5.5%)</td>
<td>2 units</td>
</tr>
<tr>
<td>Pint of lower-strength lager/beer/cider (ABV 3.6%)</td>
<td>2 units</td>
</tr>
<tr>
<td>Pint of higher-strength lager/beer/cider (ABV 5.2%)</td>
<td>3 units</td>
</tr>
</tbody>
</table>

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PRACTICAL DIABETES Vol. 37 No. 1
**Red wine.** In an attempt to mimic social drinking practices, one study analysed the effect of red wine (6 units) with a standard Italian lunch and supper. The authors found that red wine with an Italian meal produced no change in blood glucose over 15 hours, but stated that alcohol intake was associated with an increased insulin requirement. Another study group also concluded that red wine (4 units) taken with a meal consisting of bread and pasta had no effect on glucose or insulin, after the start of the meal and for 6 hours after consumption. Although an increased insulin requirement after 2 hours in the alcohol group was required, this was not statistically significant. Both studies used water in the control group and provided similar carbohydrate type, implying that red wine was the causative factor for increased insulin requirements. Changes in levels of lactate and ketone bodies were also described which, although not significant, led authors to suggest that alcohol consumption has the potential to increase the risk of ketoacidosis and lactic acidosis in people with T1DM with poor glycaemic control in the context of reduced insulin availability.

**Glucose variability**

Various/mixed alcoholic beverages. Studies using these drinks are scarce, but one study group used a socially-relevant design, which alluded to pre-mixed sweetened drinks including beer, wine and spirits. Other factors were acknowledged that may coincide with social drinking such as dancing, walking, sex and convenience foods that had not been accounted for, and no doubt would have a substantial effect on blood glucose and self-care. Also insulin absorption, and duration of diabetes, should all be considered. The study results demonstrated increased glucose variability within the alcohol group. In the control group, a significant increase in low glucose levels was found which possibly occurred because of increased physical activity.

**Impact of alcohol consumption on cognitive function**

Some symptoms of hypoglycaemia or hyperglycaemia are similar to those of alcohol intoxication, which could impair blood glucose awareness. The blood alcohol concentration (BAC) is a measure of the ethanol content in the blood, and the pharmacological effects on cognitive function in a human are apparent although minimal, even when plasma concentrations are small (46mg/100ml). An example of plasma ethanol amounts in daily life is that at 80mg/100ml it is illegal to drive in most of the UK, although in Scotland the limit is 50mg/100ml. Correlations between BAC and clinical symptoms demonstrate, at 10–50mg/dL, mild euphoria, decreased inhibitions and diminished judgement, which are similar to mild hypoglycaemia. At this stage, polyuria and thirst are common which may portray hyperglycaemia. At 50–100mg/dL, impaired coordination, loss of critical judgement, impaired memory and concentration occur, and then progress to confusion, disorientation, impaired balance, slurred speech and exaggerated emotional states at 150–300mg/dL. Then 250–400mg/dL presents sleep, stupor, incontinence and muscular incoordination, and coma and possible death at 400–500mg/dL. These signs do replicate the effects of mild-to-severe hypoglycaemia, and why the effects of moderate alcohol consumption can mask hypoglycaemia awareness when awake. To verify this, blood glucose perceptions after alcohol consumption in one study were 75% inaccurate at bedtime, which corresponded with an increased risk of nocturnal hypoglycaemia.

**Vulnerable drinkers with T1DM**

When considering at-risk groups in T1DM, results differ where one study group found no statistical differences in alcohol consumption between those with higher or lower HbA1c, compared with another where a significant association between high alcohol consumption and higher HbA1c was revealed. In T1DM, the prevalence of intoxication within the last month was associated with lower HbA1c (p=0.021), and also associated with a higher incidence of severe hypoglycaemia (p=0.04) in one study. Whereas, another study indicated an increased risk of hyperglycaemia with potential diabetic ketoacidosis (DKA) among alcohol consumers compared to abstainers. The DKA prevalence was significantly increased in those classified as high-risk drinkers with poor glycaemic control. The investigation did not include beverage or food types.
With regard to people at risk, an increased significant risk of severe hypoglycaemia with drinkers compared to abstainers of alcohol has also been identified. There was no significant difference with reported incidence of hypoglycaemia between low- and high-risk drinkers although a significant difference was shown with abstainers. Intoxication within the last month was associated with a significantly higher incidence of severe hypoglycaemia when compared to abstainers. Another review also supported the increased hypoglycaemia risk, and in an analysis of YP’s self-reported behaviours and experiences, along with any self-care research findings, it was stated there was a lack of evidence that could be subsequently translated into evidence-based educational tools.

Limitations of studies
Data extraction for 15 studies is presented in Appendix 1. The length of time blood glucose was monitored following alcohol consumption varied between individual studies with seven running >12 hours overnight and four for <3 hours duration. Those with shorter periods of measurement clearly had the potential to miss late changes in blood glucose. Most studies were laboratory based (n=11), and the variation in study designs reflected the difficulties in attempting to reproduce typical social drinking conditions (n=2). This causes challenges in applying the data into a ‘real-life’ context. Most original studies were published prior to 2008 (n=12) with alcoholic beverages such as beer, wine and vodka. Only one study was identified that examined the effects of currently popular beverages such as alcopops, flavoured cider, shots and spirits with different mixer drinks. The current trend of binge drinking has not been alluded to in any study. Most studies had ≤10 participants (n=10 studies), and participants were predominantly male (n=11), with participants acting as their own control (n=12). The impact on self-care of using newer technologies such as insulin pumps and flash glucose monitoring has not been studied, nor the impact of more recently available insulin analogues.

**Box 1. Key messages from original studies to apply into clinical practice and alcohol education for people using basal (intermediate/long-acting insulin) and bolus (fast-acting insulin) insulin**

**POSSIBLE APPLICATION OF FINDINGS INTO REAL-LIFE**

**General suggestions**
- In a fasting state, ethanol/alcohol given in a controlled environment does not affect insulin requirements, blood glucose or consumed glucose. External influences such as consumed carbohydrates, alcoholic drink ingredients, physical activity, and insulin doses must have an effect on the risk of hypo/hyperglycaemia. Carbohydrate consumption is also used to deliberately cause hyperglycaemia as a hypoglycaemia prevention strategy.
- Alcohol can adversely affect a person to predict the blood glucose level accurately. During and after drinking alcohol it is important to check blood glucose levels regularly and not guess them. Alcohol can reduce the awareness of hypoglycaemia and it is important not to rely on autonomic symptoms and neuroglycopoenic symptoms as these may be impaired. Hyperglycaemic symptoms can be confused with alcohol consumption, i.e. thirst and polyuria.
- People with a lower HbA1c may provoke a bigger risk of hypoglycaemia compared to more stable or higher glucose control.
- Diabetic ketoacidosis risk is increased with high alcohol drinkers. If blood glucose is higher, a blood ketone test is recommended, especially for people with a high HbA1c.

**White wine and spirits usually lower the blood glucose**
- When drinking modest amounts, bolus and basal insulin doses may need to be reduced.
- However, when drinking white wine, there is an increased risk of ketosis if blood glucose is higher (although still in the normal range). If blood glucose is higher and especially if drinking sweet wine, check blood glucose levels and if hyperglycaemic it may be useful to check for blood ketones.

**Beer can cause hypoglycaemia and hyperglycaemia**
- For a person with type 1 diabetes, it would be useful to understand the effects of alcohol and carbohydrate (CHO) content of beer. A low CHO (3–7g) content beer (per half-pint or 1 unit of alcohol), with 5% or less ABV should cause less glycaemic effects.
- Low <5% ABV and ordinary or high CHO beer can cause hyperglycaemia.
- High >5% ABV and low CHO beer may cause or increase the risk of hypoglycaemia within 3 hours after consumption.

**Red wine can cause hyperglycaemia**
- Two glasses of wine consumed with a low glycaemic index meal does not appear to cause glycaemic risks.
- When drinking red wine at lunchtime, increased bolus insulin may be required to prevent post-prandial hyperglycaemia (depending on the amount of alcohol consumed).

**Recommendations for self-care strategies to reduce the risks of alcohol consumption in T1DM**

The recommendations from ‘diabetes associations’ mainly promote alcohol avoidance and this advice can be futile. There are various issues that require consideration when contemplating why blood glucose disparities occur and how they can be prevented in T1DM, and from our review we identified six underlying themes (see Table 2). These themes interact and must be considered as a whole for patient education and subsequent self-care. Hence, it would be advantageous to examine all of these in future studies. The biochemical response to alcohol, exogenous insulin, the presence and timing of carbohydrate food consumption, the constituents and amount of alcohol in individual beverages, can all have an important impact on glucose homeostasis. The actual cognitive effect of alcohol can also adversely affect awareness of hypoglycaemia and the ability to treat such episodes.

For patient education, we are not aware of any exploratory research findings being developed into glycaemic prevention strategies for real life. Two studies investigated...
Alcohol consumption and type 1 diabetes

self-care preventative strategies regarding insulin adjustment and carbohydrate intake, but the corresponding effect on glucose was not described and so the effectiveness cannot be determined. From the publications discussed in our review, key messages have been extracted for clinical practice and are summarised in Box 1.

Conclusion
In summary, there is a dearth of research evaluating self-care strategies for prevention of either hypoglycaemia when consuming alcohol, and in particular for binge drinking. Research in this area is required to inform patient education for people with TIDM. Different beverage types have different effects on blood glucose levels. Many people with TIDM currently adopt self-care behaviours based on individual trial and error with inherent risks associated. The introduction of flash glucose monitoring might improve self-care strategies but needs evaluation. However, it is important to highlight the risk of premature death and dangerous episodes of hypoglycaemia and hyperglycaemia, especially in YM with TIDM that can be as a result of drinking alcohol. As recommended by many of the authors cited, evidence based self-care guidelines are required.

From this review, the content of Tables 1 and 2 and Box 1 could be utilised as a toolkit within education sessions when discussing alcohol consumption.

Acknowledgements
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Declaration of interests
There are no conflicts of interest declared.

KEY POINTS

- Hypoglycaemia, hyperglycaemia, glucose variability and unpredictability are all consequences of alcohol consumption in type 1 diabetes
- Carbohydrate, glucose, alcohol by volume (ABV), and volume of beverage consumed alongside food will affect blood glucose concentrations
- The common risk period for hypoglycaemia is 8–12 hours after alcohol consumption
- A strategy to prevent hypoglycaemia is to cause deliberately an episode of hyperglycaemia
- Many people with type 1 diabetes currently adopt alcohol–health related behaviours through trial and error

References

<table>
<thead>
<tr>
<th>Author</th>
<th>Participant no. and gender</th>
<th>Control Data collection period after alcohol intake (hrs) and glucose method used</th>
<th>Alcohol drink type and route, and control type</th>
<th>UK alcohol unit dose</th>
<th>Food</th>
<th>Insulin route and type</th>
<th>Glycaemic risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avogaro et al. (1983)</td>
<td>10 M</td>
<td>Age and weight matched controls (non-diabetic)</td>
<td>Red wine at lunch and supper. Water control</td>
<td>6 (assumed)</td>
<td></td>
<td>IV infusion</td>
<td>Hyperglycaemia</td>
</tr>
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<td></td>
<td>Alcohol group insulin amount vs control (64±4 units vs 53±4 units)</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Fritsche et al. (1995)</td>
<td>9 (3 F)</td>
<td>Within subject</td>
<td>White wine consumed after evening meal at 2200</td>
<td>0.7g alcohol/kg body weight = 2 large glasses</td>
<td>36g CHO evening meal at 1800</td>
<td></td>
<td>BG perceptions were 25% correct at before bed. Treatment was required to prevent nocturnal hypo and hyperglycaemia</td>
</tr>
<tr>
<td>Gin et al. (1992)</td>
<td>5 M</td>
<td>Within subject</td>
<td>Red wine with lunch. Water control</td>
<td>4 (assumed)</td>
<td></td>
<td>IV infusion</td>
<td>None</td>
</tr>
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<td></td>
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<td></td>
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<td></td>
<td>Alcohol group insulin amount vs control (64±4 units vs 53±4 units)</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Henderson et al. (1987)</td>
<td>7 M</td>
<td>Within subject</td>
<td>Beer A (12.1g CHO, 3.8% v/v), B (8.7g CHO, 3.3% v/v), C (2.9g CHO, 5.5% v/v), D (1.4g CHO, 4.1% v/v). 1 unit given before, during and after the evening meal. No control drink</td>
<td>3</td>
<td></td>
<td></td>
<td>Hypoglycaemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Evening meal and late night snack given matched to individuals CHO consumption</td>
<td>Own insulin administration. Insulin type not stated but one would presume from 1987 it would be soluble/isophane either b.d. fixed mixture or MDI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hermann et al. (2017)</td>
<td>29,630 (53% M)</td>
<td>None</td>
<td>Diabetes registry data analysis</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>Alcohol use higher in males (p&lt;0.05). Adjusted severe hypoglycaemia rate similar for high- and low-risk drinkers, but significantly lower in abstainers (p=0.009). Adjusted DKA risk significantly increased with high alcohol use (p&lt;0.001)</td>
</tr>
<tr>
<td>Ismail et al. (2006)</td>
<td>14 (5 M)</td>
<td>Within subject</td>
<td>7 consumed pre-mixed sweetened drinks only, although 80% consumed these at some point, 4 consumed mixed spirits, 1 mixed spirits plus beer, 3 beer, and 1 wine</td>
<td>M = 9.0, F = 6.3 mean units</td>
<td>13 had dinner prior to drinking. 10 consumed food after drinking</td>
<td>6 used MDI and 8 used twice-daily fixed mixture</td>
<td>Increased variability in alcohol group. No difference in percentage of time spent in normal and high glucose levels in both groups. Higher percentage of time spend in lower glucose in control group (p&lt;0.03)</td>
</tr>
</tbody>
</table>

Abbreviations: CHO = carbohydrate in grams; MDI = multiple daily injections; BG = blood glucose; DKA = diabetic ketoacidosis.

Appendix 1. Study characteristics. (Continued on the next 2 pages)
<table>
<thead>
<tr>
<th>Author (date of study)</th>
<th>Participant no. and gender</th>
<th>Control</th>
<th>Data collection period after alcohol intake (hrs), and glucose method used</th>
<th>Alcohol drink type and route, and control type</th>
<th>UK alcohol unit dose</th>
<th>Food</th>
<th>Insulin route and type</th>
<th>Glycaemic risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kerr et al. (2007)</td>
<td>17 (3 F) Within subject</td>
<td>2.5</td>
<td>Venous plasma glucose</td>
<td>Vodka and sugar-free orange. Sugar-free orange control</td>
<td>3</td>
<td>Fasted</td>
<td>IV infusion</td>
<td>Hypoglycaemia Significant interaction between alcohol and hypoglycaemia (p=0.001). Alcohol significantly reduced insulin sensitivity in both euglycaemic and hypoglycaemic visits (p=0.048)</td>
</tr>
<tr>
<td>Kerr et al. (2009)</td>
<td>10 (3 F) Within subject</td>
<td>4</td>
<td>Venous plasma glucose</td>
<td>White wine over a 90-minute period with lunch. Alcohol-free wine control</td>
<td>M = 8 F = 6</td>
<td>Usual breakfast at home, lunch 83g CHO</td>
<td>Usual MDI using Novorapid premeal, and glargine at bed. Small correction dose given during the morning to attain lunch blood glucose 8–10mmol/L</td>
<td>None</td>
</tr>
<tr>
<td>Kerr et al. (1990)</td>
<td>7 M 8 non-diabetic (6 F)</td>
<td>1 hour and 40 minutes. Venous plasma glucose</td>
<td>Vodka. Water placebo.</td>
<td>0.75g ethanol/kg body weight = 2 or 3 large measures</td>
<td>Fasted for 12–13 hours</td>
<td>IV infusion</td>
<td>None</td>
<td></td>
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<tr>
<td>Koivisto et al. (1993)</td>
<td>10 M Within subject</td>
<td>15 overnight. Plasma glucose</td>
<td>Vodka as aperitif, red wine with meal, cognac after meal. Water control</td>
<td>7.5</td>
<td>Overnight fast. Meals consisted of 45% CHO, 35% fat, 20% protein, split over breakfast, lunch, dinner and 3 snacks. Dinner at 7pm</td>
<td>Short-acting and intermediate-acting insulin at breakfast, short-acting insulin at evening meal, and intermediate-acting at bed.</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Moriarty et al. (1993)</td>
<td>9 (2 F) Within subject</td>
<td>1.5</td>
<td>Ethanol IV (0.5g/kg for 10 mins, then 0.25g/kg over 60 minutes. Normal saline control</td>
<td>6.4</td>
<td>Fasted</td>
<td>IV infusion</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Pastor et al. (2018)</td>
<td>16 – Interview</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>Alcohol rarely predictable for effect on BG. Difficulty interpreting physical symptoms and hypoglycaemia awareness. Difficulty differentiating between a hangover and hypoglycaemia</td>
</tr>
</tbody>
</table>

Abbreviations: CHO = carbohydrate in grams; MDI = multiple daily injections; BG = blood glucose.

Appendix 1. Study characteristics. (Continued on the next page)
<table>
<thead>
<tr>
<th>Author (date of study)</th>
<th>Participant no. and gender</th>
<th>Control</th>
<th>Data collection period after alcohol intake (hrs), and glucose method used</th>
<th>Alcohol drink type and route, and control type</th>
<th>UK alcohol unit dose</th>
<th>Food</th>
<th>Insulin route and type</th>
<th>Glycaemic risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Richardson et al. (2005)</td>
<td>16 (gender not reported)</td>
<td>Within subject</td>
<td>24 overnight. CGM</td>
<td>Vodka and orange juice over 60 minutes. Orange juice control</td>
<td>6.4</td>
<td>Standard meal of 120–150g CHO</td>
<td>Usual MDI insulin</td>
<td>Hypoglycaemia A</td>
</tr>
<tr>
<td>Spraul et al. (1988)</td>
<td>8 M</td>
<td>Within subject</td>
<td>3 Plasma glucose</td>
<td>Diet: CHO-depleted beer (3g CHO), and ordinary CHO-containing beer (18g CHO)</td>
<td>2.5</td>
<td>7-hour fast after breakfast</td>
<td>IV infusion (CSII correction dose given prior to start using soluble insulin)</td>
<td>Hypoglycaemia A After ordinary beer the glucose increased by a maximum of 3.2±0.7mmol/L (N/S). Diet beer decreased by 1.0±0.4mmol/L, and was significantly different from the control group (p&lt;0.05)</td>
</tr>
<tr>
<td>Turner et al. (2001)</td>
<td>6 M</td>
<td>Within subject</td>
<td>15 overnight. Venous plasma glucose</td>
<td>White wine over 90-minute period, 3 hours after evening meal. Water control</td>
<td>6.6</td>
<td>Standard meal at 6pm (50% CHO) and 8am (both matched to individuals average CHO amount)</td>
<td>Usual evening meal dose of regular/soluble insulin reduced by 70%, and IV basal infusion from 11pm till 12 noon. Usual breakfast dose regular/soluble insulin</td>
<td>Hypoglycaemia A No significant differences in evening or overnight glucose levels. In the morning fasting and post-prandial glucose were significantly lower in the alcohol group (p&lt;0.01). None occurred in the control group</td>
</tr>
</tbody>
</table>

Abbreviations: CGM = continuous glucose monitoring; CHO = carbohydrate in grams.

Appendix 1. Study characteristics. (Continued from the previous 2 pages)