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REVIEW

Alcohol ingestion and temperature regulation during cold exposure*

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Outdoor or wilderness activities are sometimes combined with the ingestion of alcoholic beverages. Despite the feeling of warmth induced by alcohol ingestion, it is widely believed that alcohol actually causes a decrease in body core temperature and increases the risk of hypothermia during cold exposure. However, the literature on the effects of alcohol ingestion on thermoregulation is conflicting. This review summarizes the scientific findings concerning this topic and identifies a number of confounding factors that may explain the conflicting observations. These factors include quantity of alcohol ingested, severity of the cold stress, nutritional state of the individual, composition of the drink, body composition of the individual and alcohol tolerance of the individual. When these factors are considered, it appears that (1) alcohol acts as a poikilothermic agent, causing a reduction in body core temperature during cold exposure, with the magnitude of reduction related to blood alcohol concentration, (2) the severity of cold and the individual's body composition modify the thermoregulatory effects of alcohol, and (3) hypoglycemia greatly exacerbates the reduction in body temperature caused by alcohol ingestion. Furthermore, the primary mechanism by which alcohol ingestion exacerbates the fall in body core temperature during cold exposure appears to be via an impairment of shivering thermogenesis resulting from alcohol-induced hypoglycemia, rather than by increasing heat dissipation via vasodilation as commonly believed.

Key words: cold exposure, hypothermia, poikilothermic, thermoregulation, alcohol ingestion, hypoglycemia

Introduction

Outdoor wilderness activities are sometimes combined with alcohol ingestion: an ice fisherman having a nip of whisky to keep warm while waiting for a bite, hikers returning to camp sipping a bit of cognac, skiers relaxing with an Irish coffee. Although alcohol ingestion often induces a perception of warmth, it is widely believed, especially in the medical community, that ingestion of even a small amount of alcohol can alter thermoregulation.

*The views, opinions, and/or findings in this report are those of the authors and should not be construed as official Department of the Army position, policy, or decision unless so designated by other official documentation.

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A review of alcohol's effects on thermoregulation would be incomplete without some mention of the psychological or behavioral effects of alcohol, including its effects on judgement and perception of cold. These effects may lead to inappropriate behavior or actions that place an individual at a greater risk for cold injury and contribute to cold-related mortality, particularly in urban settings. The behavioral effects of alcohol must not be overlooked; however, the purpose of this manuscript is to critically review scientific studies examining the *physiological* effects of alcohol ingestion on human body temperature regulation.

In thermoneutral conditions, alcohol ingestion has been demonstrated to increase both skin blood flow and body heat loss [1]. On this basis, it has been speculated that alcohol ingestion predisposes an individual to develop hypothermia during cold exposure. Indirect support for this premise is provided by numerous reports of accidental death of intoxicated individuals from cold exposure [2]. In the book *Safety and Survival at Sea*, Lee and Lee [3] cite many examples of alcohol-related deaths in the Arctic, and strongly suggest that alcohol increases the rate of body cooling during cold exposure. Furthermore, Danzl and Pozos [4] have indicated that ethanol ingestion is the most common associated cause for urban hypothermia, while Weyman *et al.* [5] have reported a greater risk for accidental hypothermia in an alcoholic population.

The scientific literature lacks agreement concerning the physiological effects of alcohol ingestion on thermoregulation during cold exposure. Some studies have reported a fall in body core temperature during cold exposure following alcohol ingestion [6–9], while others observed no effect [10–14]. Reports even exist suggesting that alcohol ingestion might be beneficial during cold exposure, since the presumed vasodilatory effects of alcohol may protect against peripheral cold injuries, e.g. frostbite [15], and alcohol's narcotic effect can reduce discomfort and improve sleep during cold exposure [10,11,16]. Presumably, it was these supposed benefits of alcohol that influenced both German [15] and Russian [17] military leaders to recommend the prophylactic administration of alcohol to soldiers during World War II. However, others have argued that the reduced perception of cold in individuals who have consumed alcohol could be extremely dangerous and lead to undue exposure and extreme hypothermia [6].

Table 1. Alcohol doses and resulting blood alcohol concentration.

Alcohol dose		Blood alcohol concentration[b]
No. of drinks[a]	100% ethanol	
1 (low)	0.16 g per kg body wt	23 mg per 100 ml
2	0.32 g per kg body wt	46 mg per 100 ml
3	0.47 g per kg body wt	69 mg per 100 ml
4	0.63 g per kg body wt	92 mg per 100 ml
5 (high)[c]	0.79 g per kg body wt	115 mg per 100 ml

[a]One drink = 12 oz beer, 4 oz wine or 1 oz distilled spirits.
[b]Legal limit for driving in most states = 100 mg per 100 ml.
[c]The term 'high' is used only for relative comparison between studies presented. Clinicians regularly encounter individuals with much higher blood alcohol concentrations.

Table 2. Air and water temperatures associated with cold stress.

Relative stress	*Air*	*Water*
Low	> 15° C	> 25° C
Moderate	5–15° C	15–25° C
High	< 5° C	< 15° C

Confounding factors

It appears that alcohol's effects on thermoregulatory responses to cold are modulated by parameters such as (1) quantity and rate of alcohol ingestion, (2) severity of cold stress, (3) nutritional status, (4) carbohydrate content of the drink, (5) body composition and (6) alcohol tolerance. Each of these confounding factors will be addressed.

Since the studies reviewed have reported alcohol dosages in a variety of units, we have converted the amount of alcohol ingested to the equivalent amount of ethanol (100%) expressed in g per kg body weight. Table 1 provides a guide to the relative alcohol doses administered in the various studies, as well as an estimate of the resulting blood alcohol concentration, for a 70 kg individual. It should be noted that individual differences in alcohol tolerance and clearance can cause extreme variation in these estimated values. Furthermore, while the estimates of blood alcohol concentration assume a 1 hr ingestion period, in many of the studies reviewed ingestion occurred more rapidly i.e. over 15–20 min.

Table 2 presents a guideline for comparing studies with respect to the relative stress of the cold exposure. While the table only addresses temperature, it should be noted that exposure duration, relative humidity and wind speed will also influence the magnitude of the cold stress.

One factor impacting on whether or not alcohol ingestion alters the thermoregulatory response to cold exposure appears to be quantity and rate of alcohol ingested and the resulting blood alcohol levels achieved. Studies indicate that at low dosages of alcohol (see Table 1) the ability to thermoregulate is unaffected [7,16], while at higher blood alcohol levels the incidence of thermoregulatory disturbance is increased [6,8]. Haight and Keatinge [7] compared two groups of individuals. One group received a relatively low dose of alcohol, i.e. approximately 0.34 g per kg body wt, which elicited a blood alcohol level of approximately 23 mg per 100 ml, the other group served as a control and ingested only water. After 30 min in a cold room at 14.5° C no impairment of thermoregulation, as measured by rectal temperature was observed. Hobson and Collis [16], using a moderate alcohol dose (0.52 g per kg body wt), measured the time for rectal temperature to reach 35° C after subjects were immersed in 15° C water, and found that the rate of body core cooling was actually *reduced* during the alcohol trial compared with an identical exposure in which no alcohol was consumed. It should be noted, however, that the methodologies utilized by Hobson and Collis are questionable, and the authors were unable to confirm their findings in a later study [18]. In that later study, Fox *et al.* [18] immersed ten subjects in 10° C water for 45 min and reported that, despite relatively high alcohol dosages (0.86 g per kg body wt), core cooling rates were not

significantly different from controls. The authors went on to report that alcohol ingestion reduced shivering and metabolic rate by 13%, although this did not affect cooling rate. It was concluded that alcohol at these dosages did not influence the rate of progression to hypothermia.

Graham and Baulk [6] also studied the effects of a relatively high dosage of alcohol (0.79 g per kg body wt) on the thermoregulatory responses to subjects immersed in 13° C water for 24 min, and found that core temperature decreased significantly faster after alcohol ingestion. Of note is that the amount of alcohol administration in the study by Fox *et al.* [18] (see above) was slightly greater than that of Graham and Baulk [6] and yet the subjects of Graham and Bault achieved higher blood alcohol concentrations (92 mg per 100 ml *vs* 82 mg per 100 ml). Although the rate of alcohol ingestion was similar between the two studies, Graham and Baulk [6] began cold exposure 15 min following ingestion, while Fox *et al.* [18] waited 40 min. These differences in experimental methodology may explain the differences in blood alcohol concentrations achieved and may account for the conflicting findings.

Graham and Dalton [8] studied the thermoregulatory responses to exercise in the cold following alcohol ingestion. Subjects clothed in a 'sweatsuit' ingested solutions containing water, water and alcohol, or water, alcohol, and dextrose. Again there was a relatively high dose of alcohol (0.79 g per kg body wt). Subjects then performed intermittent exercise for 2 hr while exposed to cold air at −5° C. Exercise in the cold during the control condition (water alone) resulted in a slight increase in rectal temperature, while exercise in the cold during the alcohol trial produced a fall in core temperature. The responses to the alcohol and dextrose trial will be discussed later.

Animal studies also suggest a relationship between alcohol dose and the thermoregulatory response. Under thermoneutral conditions mice and rats have shown a greater degree of hypothermia with increasing doses of alcohol [19,20]. Murphy and Lipton [21] studied monkeys in both thermoneutral and cold environments and reported lower core temperatures in both environments with higher doses of alcohol. Furthermore Gilliam [22], using different methods of alcohol administration in mice, demonstrated that the more rapidly blood alcohol concentration rose, the greater was the fall in body core temperature. Importantly, it should be noted that most evidence suggests that the relationship between alcohol dosage and the subsequent effects on thermoregulation is not linear, but more likely a 'threshold phenomenon'. This hypothesis helps to explain the reports indicating that low to moderate doses of alcohol have no effect on thermoregulation during cold exposure, yet at high or extreme doses, thermoregulation is significantly altered.

Also of importance is the work of Koren *et al.* [23] who demonstrated in piglets that apparently alcohol ingestion not only affects the body's responses to cold exposure, but the reverse is also true, i.e., cold exposure affects the body's responses to alcohol. This was demonstrated by an alteration in the pharmokinetics of ethanol elimination. Cold exposure resulted in blood alcohol concentrations remaining elevated for a significantly longer period of time than under thermoneutral conditions.

Another factor appearing to modulate the effects of alcohol on thermoregulation is the magnitude or severity of the cold stress. The subject's heat balance may markedly modify the vasomotor response to cooling [24,25]. Under thermoneutral conditions, a decrease in core temperature is indeed found following alcohol ingestion, apparently the result of an increased body heat loss through peripheral vasodilation. Gillespie [1] reported that

alcohol ingestion (0.63 g per kg body wt) produced large increases in peripheral blood flow of subjects resting in a thermoneutral environment. Especially noteworthy were the five- to six-fold increases in peripheral blood flow to the hands and feet. However, Keatinge and Evans [11] studied the effects of alcohol ingestion (59.2 g per subject; body weights were not provided) on the finger blood flow of subjects before and during immersion into 15° C water for 30 min. While pre-immersion finger blood flow was significantly higher during the alcohol trial, during the cold water immersion, finger blood flows declined to similarly low values in both the alcohol and the non-alcohol trials. This strongly suggests that the vasodilatory effects of alcohol evident under thermoneutral conditions are overridden by the vasoconstrictor response elicited by exposure to cold.

Some data suggest that the fall in body temperature observed after cold exposure and alcohol ingestion may be due to a reduction in metabolic heat production rather than an increased heat loss via vasodilation. Graham and Baulk [6] attributed the significantly greater fall in body core temperature during cold exposure following alcohol ingestion to a delayed onset and decreased duration of shivering, as assessed by surface electromyography (EMG). Murphy and Lipton [21] exposed monkeys to cold air (18.5° C) after alcohol ingestion (1.0 g per kg body wt) and found the significant fall in core temperature to be concomitant with a reduction in oxygen consumption. They further reported that tail skin temperature also decreased following alcohol ingestion and cold exposure, indicating that the vasoconstrictor response to cold was preserved. Again, these studies suggest that alcohol affects thermoregulation during cold exposure by inhibiting shivering, rather than through peripheral vasodilation, as seen in thermoneutral conditions.

The decreased ability to thermoregulate in the cold after alcohol ingestion may be related to the effect of blood alcohol on plasma glucose concentration. It has been demonstrated that alcohol ingestion inhibits hepatic glucose production and leads to hypoglycemia [26,27]. Hypoglycemia itself may affect thermoregulation through (1) increased sweating [28], (2) peripheral vasodilation [14,28] and (3) reduced or eliminated shivering [8]. It appears that when exercise and/or fasting are associated with alcohol ingestion, the hypoglycemia, and hence its effects on thermoregulation are exacerbated. Haight and Keatinge [7] reported that when cold exposure was preceded by ethanol ingestion and prolonged exhaustive exercise, there was an increased incidence of hypoglycemia and a greater fall in core temperature compared with a control group that consumed only water. Furthermore, subjects had a lower metabolic rate during the exercise and alcohol trial than during the control trial. This was attributed to the abolishment of shivering during the alcohol trial. These authors went on to demonstrate that, following the same exercise regime, if dextrose were consumed in addition to alcohol, the hypoglycemia could be prevented, and metabolic rate and core temperature were better maintained during cold exposure [7]. In a similar study, Graham and Dalton [8] examined the effects of alcohol ingestion on thermoregulation during exercise in the cold, and reported significantly lower core temperatures during exercise following alcohol ingestion concomitant with significant reductions in plasma glucose concentrations. These authors also demonstrated that ingesting dextrose in addition to the alcohol prevented both hypoglycemia and thermoregulatory disturbances.

Similar results have been produced in studies utilizing insulin infusion to induce hypoglycemia prior to cold exposure [28]. In these studies, shivering consistently ceased

when insulin infusion caused plasma glucose concentrations to drop below approximately 2.5 mmol l^{-1}. Therefore, it appears that regardless of the method used to reduce plasma glucose concentration, hypoglycemia is associated with decreased shivering and metabolic heat production, and an accelerated fall in body temperature. If plasma glucose is restored by glucose infusion or ingestion, shivering returns.

In this regard, it appears that the drink composition in which the alcohol is administered may affect the thermoregulatory responses to cold exposure. This may help to explain the results of Martin and associates [12] who gave subjects an amount of alcohol that yielded average blood ethanol levels of approximately 90 mg per 100 ml. These relatively high blood alcohol concentrations may have been predicted to induce hypoglycemia and thermoregulatory disturbances, yet after a 20-min exposure to 13° C water, neither occurred. It seems likely that the 200 ml fruit juice in which the alcohol was administered compensated for the tendency of alcohol to induce hypoglycemia and hypothermia. Unfortunately neither shivering nor metabolic heat production was assessed.

The effects of carbohydrate infusion or ingestion on thermoregulation during cold exposure, described above, could be interpreted to suggest that carbohydrate availability is a limiting factor for shivering. However, the metabolic cost of shivering can be met by various metabolic substrates, and the increase in plasma catecholamine concentrations induced by cold exposure facilitates the mobilization of these substrates from both glycogen and triglyceride stores [14]. Martineau and Jacobs [29] reported significant reductions of muscle glycogen content of the vastus lateralis during cold exposure, which were attributed to the metabolic cost of shivering. Young *et al.* [30], in a similar study, did not see such glycogen depletion, and furthermore demonstrated that depletion of muscle glycogen stores induced by a low carbohydrate diet and exercise prior to cold exposure had no effect on shivering, metabolic heat production, or the thermoregulatory response to cold exposure. Importantly, for reasons discussed above, none of Young's subjects developed even a tendency toward hypoglycemia. While muscle glycogen may be an important substrate for shivering under certain circumstances, the mechanisms for alcohol's inhibition of shivering thermogenesis appears independent of any local effect on substrate availability. Gale *et al.* [28] demonstrated that the abolished shivering induced by hypoglycemia could be restored, within seconds, by intravenous glucose infusion, even in muscles of a limb removed from circulation by arterial occlusion. This strongly suggests that hypoglycemia affects temperature regulation through a central, rather than peripheral mechanism.

While the relationship between hypoglycemia and hypothermia has been described on numerous occasions, some authors have taken an alternative perspective by suggesting that prolonged hypothermia leads to hypoglycemia, rather than the reverse [14]. In addition, although hypoglycemia can evidently contribute to hypothermia, other factors appear to be involved in the thermoregulatory effects of alcohol. For example, Graham and Baulk [6] reported significantly greater reductions in core temperature during cold exposure following alcohol ingestion than during a similar control trial, despite subjects maintaining normal plasma glucose concentrations during both trials. Furthermore, in mice specially bred to maintain high levels of blood glucose, hypothermia occurred following ethanol administration despite elevated blood glucose concentrations [9].

Ethanol could affect thermoregulation by directly altering the thermoregulatory set-point for body temperature regulation [31,32]. Such a central effect of alcohol is

suggested by studies in which animals are allowed to thermoregulate behaviorally by selecting their own ambient temperature in a gradient environmental chamber [31,32]. Following ethanol administration, both mice [31] and rats [32] were shown to select ambient temperatures that were significantly cooler than during control experiments. This was despite the fact that core temperature was significantly lower following alcohol administration, and was interpreted to indicate that ethanol had altered the 'set-point' for thermoregulation. The reduced thermoregulatory set point theory may also help explain the frequent reports of subjects feeling warmer and more comfortable during cold exposure following alcohol ingestion compared to control trials [6,11,16].

Body composition of the subjects is also likely to modulate the thermoregulatory effects of alcohol during cold exposure. Body composition directly influences the insulatory capacity of the body shell. This interaction is demonstrated by the study of Haight and Keatinge [7] who showed that, despite a dramatic fall in blood glucose concentration, one obese subject did not experience the fall in body temperature that the leaner subjects did during cold exposure. This further supports the notion that, during cold exposure, alcohol's effects on shivering thermogenesis are more important than its effects on vasomotor tone. If vasodilation induced by alcohol ingestion were important, it would be anticipated that both fat and lean subjects would experience similar increases in conductive and convective heat loss. On the other hand, the reduction in shivering thermogenesis would take longer to be manifested as a fall in core temperature in a fat individual because of the greater insulatory capacity of the body shell.

Body composition is also an important consideration when determining the dose of alcohol to be administered. If the dose is calculated solely on the basis of the subject's body weight and the subjects are of varied body composition, then the dose relative to their total body water will not be equivalent and will result in highly variable blood alcohol concentrations between individuals. Martin *et al.* [12] reported greatly varied blood alcohol levels (ranging from 30 to 175 mg per 100 ml) 45 min after giving subjects volumes of ethanol based on body weight alone. Following 20 min cold water immersion, the variability remained large (47–158 mg per 100 ml), with some subjects' blood alcohol levels still climbing while others' had already begun to fall. In addition to differences in the relative dose of alcohol administered, part of the variability in blood alcohol levels achieved is likely due to individual differences in alcohol tolerance or clearance. Studies which report large variability in blood alcohol concentrations, and/or large differences in rate of increase in blood alcohol concentrations, are difficult to interpret regarding the effects of alcohol on thermoregulation.

Age may also influence the effects of alcohol on thermoregulation. While no human studies have directly examined alcohol-related changes in thermoregulation with respect to age, aging in monkeys has been associated with an increased sensitivity to the hypothermic effect of alcohol. Murphy and Lipton [21] studied young (<4 years), adult (4–9 years), and aged (>9 years) monkeys during exposure to three temperatures (18.5, 25.5 and 30.5° C) following separate trials utilizing three alcohol doses (0.50, 1.0 and 2.0 g per kg body wt). The aged group showed the greatest hypothermic response in all three temperatures, both in terms of magnitude of decrease in rectal temperature and duration of the response. In addition, and as we have previously suggested, the higher the dose of alcohol, the greater the hypothermic response. This was especially evident in the older animals.

The potential interactive effects of alcohol combined with other pharmacological

agents on the thermoregulatory response to cold is worth mentioning. Many agents are known either to exacerbate or modulate alcohol's effects on thermoregulation, although the mechanism(s) for these effects are not clear [14,33]. The interactive effects of these agents on alcohol and thermoregulation are certainly important to the emergency physician who must treat patients who have ingested alcohol in addition to one or more of these pharmacological agents. However, further discussion of this topic is beyond the scope of this paper. For the reader interested in the potential interactive effects of alcohol and drugs, we recommend the excellent review of Kalant and Lee [14].

Conclusion

While the observed effects of alcohol ingestion on the thermoregulatory responses to cold vary greatly among studies, there seems to be sufficient evidence to conclude the following:

(1) It is generally accepted that alcohol has a poikilothermic affect on thermoregulation, resulting in an increase in body core temperature occurring during exposure to warm environments and a decrease in body core temperature during cold exposure [33,34]. The reduction in core temperature induced by alcohol ingestion in thermoneutral environments appears to be due to an increase in body heat loss resulting from peripheral vasodilation.

(2) The effect of alcohol ingestion on the thermoregulatory response to cold exposure is modulated by:
   i the severity of the cold stress,
   ii the dose of alcohol administered,
   iii the individual's nutritional status,
   iv the individual's body composition,
   v the individual's metabolic clearance or tolerance to alcohol.

(3) Alcohol ingestion can induce hypoglycemia, which in turn can result in a reduction or complete loss of shivering, a significant reduction in metabolic heat production, and a more rapid fall of body temperature during cold exposure.

(4) Nearly all subjects report less discomfort during cold exposure following alcohol administration, despite the fact that body core temperature may be lower.

A schematic representation of the various factors influencing the effects of alcohol ingestion on thermal regulation is presented in Fig. 1. While the model may not include all possible interactions, it is intended to provide a visual guide to understanding the information presented. Note that while alcohol may cause peripheral vasodilation and an increase in skin blood flow during exposure to either thermoneutral or warm environments, during cold exposure this cutaneous vasodilation is completely overridden by the powerful vasoconstrictor response to cold exposure. The result is that the reduction in skin blood flow observed during cold exposure remains unaltered by alcohol ingestion.

Summary

It appears that alcohol ingestion can indeed increase the risk of hypothermia during cold exposure. The mechanism, however, appears to be via an alteration in metabolic heat production due to reduced shivering thermogenesis rather than an increased body heat

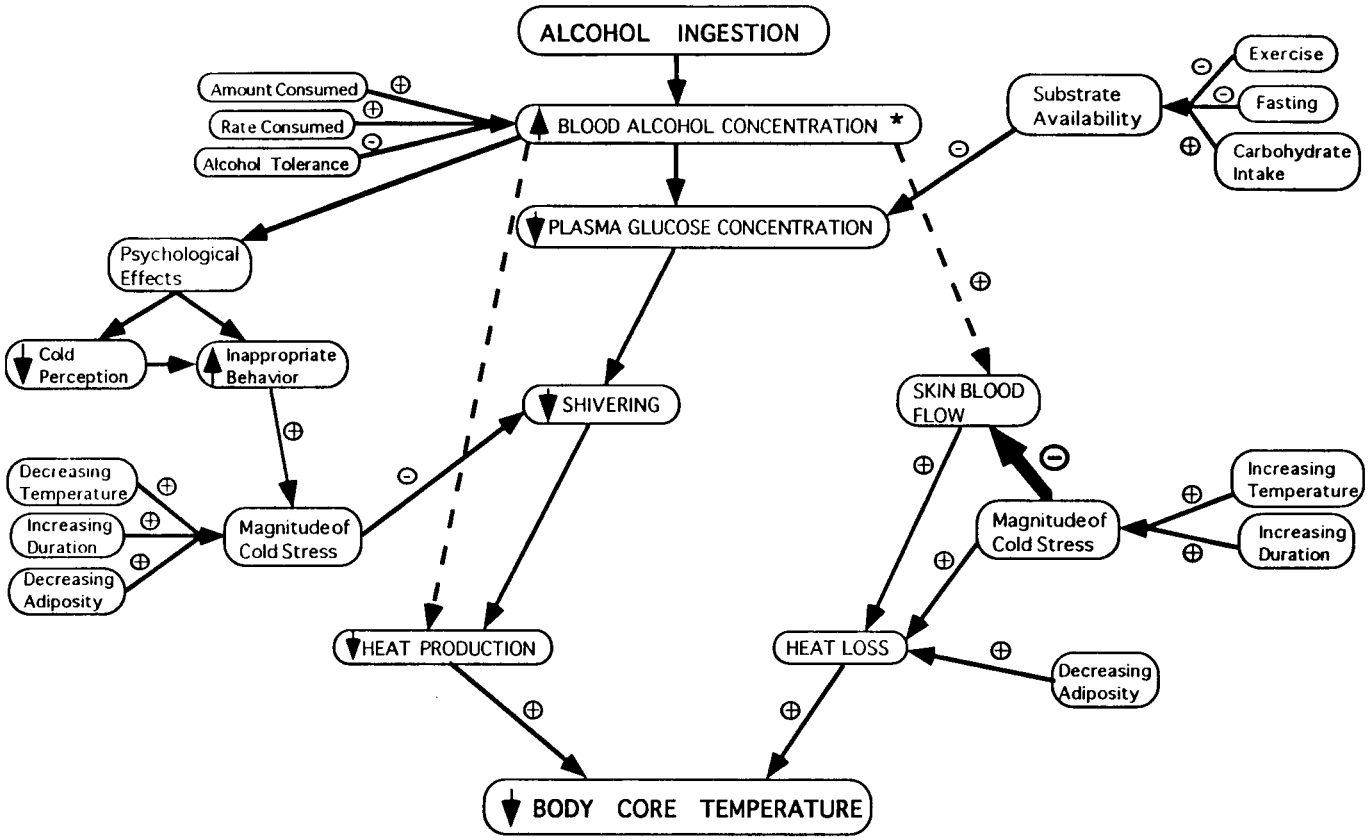

Fig. 1. Effects of alcohol ingestion on themoregulation during cold exposure.

*Relationship between blood alcohol concentration and the subsequent effects on thermoregulation, apears not to be linear but, instead, a threshold phenomenon.

Dashed lines indicate pathways that have not been demonstrated in cold environments.

loss via peripheral vasodilation. Despite initial feelings of warmth following alcohol ingestion, the potential loss of cold perception, as well as the possibility of impaired judgement, places an individual at an increased risk for accidental hypothermia. In addition, as hypothermia progresses, individuals often lose motor control and appear clumsy. If hypothermia is mistaken for intoxication, treatment may be delayed and result in a more severe situation. Although not emphasized in this review, alcohol-induced hypoglycemia, independent of its effects on thermoregulation, can result in extreme clinical manifestations including coma and death [35]. Hypothermia associated with ethanol consumption has been shown to have potential interactive effects with other pharmacological agents [33]. These effects could either exacerbate or modulate the thermoregulatory responses to cold exposure [14]. Individuals who participate in cold weather activities should be aware of the factors that increase their susceptibility to hypothermia. In addition, health care providers should be aware of the importance of restoring not only body core temperature but, if appropriate, plasma glucose concentrations.

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