spring of 2018. Relevant articles were identified through the PubMed database using the following keywords: diabetes, insulin, wilderness, expedition, mountaineering, and ultramarathon. The literature search was supplemented by a manual search of articles referenced by articles found in the initial PubMed search. Studies in these categories included randomized controlled trials, observational studies, retrospective studies, case series, and case studies. Abstract-only reports were not included. Conclusions from review articles and recommendations from related practice guidelines were cited to provide background information, but only primary sources were used in the formulation of recommendation grades. When no relevant studies were identified, the panel recommendation was based on perceptions of risk and benefit derived from clinical experience. The panel used a consensus approach to develop recommendations for the wilderness athlete with diabetes, with level of evidence assigned according to the methodology stipulated by the American College of Chest Physicians for grading of evidence and recommendations (see online Supplementary Table). These recommendations are graded on the basis of the quality of supporting evidence and balance between the benefits and risks or burdens for each modality or intervention.

Background

Diabetes mellitus is a metabolic disease characterized by elevated blood glucose resulting from a lack of insulin secretion, reduced insulin sensitivity, or both. Type 2 diabetes is the most common form (90% of individuals with diabetes have type 2 diabetes)[15] and is caused by a combination of reduced insulin sensitivity and impaired insulin secretion. Type 2 diabetes has classically been diagnosed in adults, although the rate of diagnosis in adolescents is increasing, and it is often accompanied by hypertension, hyperlipidemia, and obesity. Type 1 diabetes is typically diagnosed in children or young adults and is caused by autoimmune destruction of islet cells in the pancreas, resulting in insulin deficiency. People with type 1 diabetes require treatment with subcutaneous insulin, whereas those with type 2 diabetes may or may not require insulin treatment.

Strenuous exercise and wilderness environments can complicate glycemic control. In managing type 1 and type 2 diabetes, exercise and environmental stressors may also have a complex interaction with commonly used medications, glucose monitoring, and medication delivery. The effects of exercise alone on diabetes have been discussed in detail elsewhere,[16,17] so this guide will briefly review physical activity and environmental considerations in the pathophysiology of diabetes.

Glycemic Pathophysiology in Physical Activity

Physical activity in wilderness environments often involves prolonged activity and a combination of aerobic, anaerobic, and high-intensity exercise, so it is important to understand how neurohormonal responses differ with each type of exercise. Different types of physical activity cause distinct metabolic responses in individuals without diabetes. Aerobic activity involves repetitive glucose uptake by large muscle groups. To balance exercise-induced increases in glucose uptake from muscle, insulin secretion decreases. At the same time, counterregulatory hormones, including glucagon, cortisol, growth hormone, and catecholamines, are released, promoting endogenous glucose production through glycogenolysis and gluconeogenesis.[17-19] In athletes with diabetes, these counterregulatory hormones may lead to hyperglycemia if adjustments in carbohydrate consumption and insulin dose are not made.

In individuals without diabetes, insulin levels in anaerobic and high-intensity exercise (>80% of $\dot{V}O_{2\ max}$) do not decrease as significantly as in aerobic activities. Rather, catecholamines play a more prominent role in glucose metabolism, although growth hormone and cortisol are important as well.[17,20] After anaerobic and high-intensity exercise in individuals without diabetes, insulin levels rise to compensate for the effects of elevations in counterregulatory hormones[18,21] (Figure 1).

After exercise, glucose uptake remains elevated by both insulin-dependent and insulin-independent processes. If exercise is prolonged, insulin-dependent mechanisms can continue up to 48 h after exercise to replenish muscle glycogen stores.[16] Exercise lasting greater than 60 min has been shown to increase peripheral insulin sensitivity for up to 48 h postexercise.[22-25] The basic management strategy for type 1 diabetes of decreasing basal insulin rate while increasing carbohydrate intake during exercise to match energy utilization is based on these principles.[15,26,27] It is important to understand the increased risk of hypoglycemia that occurs after exercise, as frequent hypoglycemic events in individuals with diabetes have been linked with long-term morbidity and mortality.[28] The glycemic response to exercise changes with time of day, and risk for nocturnal hypoglycemia is highest when exercise is completed in the evening.[29]

In summary, brief high-intensity exercise has been shown to cause hyperglycemia,[30] whereas long-duration aerobic exercise is more likely to cause hypoglycemia[31] (Figure 1). The glycemic response to exercise is complex