HIGHLIGHTED TOPIC | Role of Exercise in Reducing the Risk of Diabetes and Obesity

Type 2 diabetes is the fifth leading cause of death among adults in the United States (2). Long-term complications of the disease include blindness, kidney failure, neuropathy, and deterioration of blood vessels leading to tissue necrosis. Individuals born in the year 2000 have an estimated risk of development of diabetes during their lifetime of 33% for males and 39% for females (3). Although Type 2 diabetes is a disease of multiple etiologies, it seems clear from both epidemiological and prospective studies that inactivity is a major environmental factor contributing to its onset and progression. Obesity, along with Type 2 diabetes, has become a health problem of epidemic proportions. Between the time periods of 1988–94 and 1999–2002, incidence of obesity increased from 23% to 30% of the adult population in the United States (1). Approximately 65% of adults in the United States are overweight (body mass index greater than 25). During the 1999–2004 period, an average of 85% of all people with diabetes were overweight or obese (1). There is concern that these two related inactivity-related diseases will entirely overwhelm the health care system. The annual costs for diabetes is $132 billion in the United States alone (2).

The July through September 2005 issues of the Journal bring to light the adverse physiological effects of diabetes and obesity, the relationship between inactivity and the development of these conditions, and how incorporation of physical activity into our lifelong lifestyles might reverse the alarming increase in appearance of these metabolic disorders.

Our discussion will begin with how exercise impacts the etiology of Type 2 diabetes. The mini-reviews in the July issue center on the molecular mechanisms of contraction-stimulated glucose transport and insulin sensitivity in muscle. In the mini-review entitled “Contraction signaling to glucose transport in skeletal muscle,” Drs. N. Jessen and L. J. Goodyear discuss potential mediators of contraction-induced glucose transport. Both insulin and contractile activity induce skeletal muscle to take up glucose through distinct mechanisms. Despite intense research that has revealed such potential mediators as AMP-activated protein kinase, calcium, nitric oxide, bradykinin, and the Akt substrate AS160, the mechanisms of glucose transport initiated by muscle contraction are still being actively pursued. Whereas insulin-stimulated glucose transport is impaired in individuals with Type 2 diabetes, contraction-induced glucose transport remains normal. As these authors note, exercise thus holds valuable health benefits for individuals with Type 2 diabetes.

In the mini-review entitled “Exercise-induced increase in muscle insulin sensitivity,” Dr. J. O. Holloszy addresses muscle insulin sensitivity following exercise. Independent of insulin, muscle contraction activates the transport of glucose. Insulin sensitivity increases as the acute effects of exercise-induced glucose transport subside. This increase in insulin sensitivity is instrumental in rapid postexercise accumulation of glycogen within muscle and appears to be mediated by the presence of a serum protein during contractile activity. The increased insulin sensitivity witnessed as an effect of exercise is also evident as effects of both hypoxia and treatment of muscles with AICAR. The postexercise increase in muscle insulin sensitivity has been extensively studied and characterized in considerable detail, yet the basic mechanisms at work remain elusive.

The August issue features two mini-reviews that address obesity and the role of exercise in curbing the effects of obesity. In the mini-review entitled “Fat as an endocrine organ: influence of exercise,” Dr. J. R. Berggren and colleagues address the endocrine properties of adipose tissue. The cytokines leptin, tumor necrosis factor (TNF)-α, interleukin-6 (IL-6), resistin, and adiponectin, together referred to as adipocytokines, possess both insulin-sensitizing and -desensitizing properties. Altered levels of such adipocytokines have shown a strong link to obesity, insulin resistance, and diabetes. Whereas levels of insulin-sensitizing adiponectin are decreased in obese and diabetic persons, the desensitizing TNF-α, IL-6, and leptin levels are increased in these individuals. Exercise training, a common intervention for treatment of metabolic disorders, may modulate the balance of adipocytokines and thereby restore normal insulin action.

In another mini-review entitled, “Role of physical activity in preventing and treating obesity,” Drs. J. O. Hill and H. R. Wyatt examine the role of physical activity for treating and preventing obesity. Individuals who are regularly physically active are clearly at lower risk for gaining weight over time than those whose lifestyles are largely sedentary. Evidently, the amount of physical activity necessary to prevent weight regain after substantial loss is greater than that required to prevent initial weight gain in nonobese individuals. Although there is great consistency among studies with regard to the duration and intensity of daily physical activity required to prevent weight regain, the mechanisms that govern weight maintenance have not yet been identified. These authors explore the possible relationship between physical activity level, energy balance, and healthy body weight.

The September issue features two mini-reviews that address the epidemiology of Type 2 diabetes and associated cardiovascular disease, focusing on exercise as a measure for prevention and treatment of these ailments. In the mini-review entitled “Epidemiologic evidence for the role of physical activity in reducing risk of Type 2 diabetes and cardiovascular disease,” Drs. S. M. Bassuk and J. E. Manson address epidemiologic findings indicating that physically active individuals have a dramatically lower risk of developing Type 2 diabetes. In fact, active persons are one-third to one-half as likely to develop diabetes as inactive persons. Although physical inactivity represents a significant risk factor for the development of Type 2 diabetes and its cardiovascular complications, fortunately, it is also modifiable. Through favorable effects on such factors as body weight, insulin sensitivity, glycemic control, blood pressure, lipid profile, fibrinolysis, endothelial function, and inflammatory defense systems, physical activity slows the initiation and progression of Type 2 diabetes.

Dr. M. J. LaMonte and colleagues also discuss exercise as a means of controlling Type 2 diabetes in the mini-review
entitled “Role of exercise in reducing risk of diabetes.” These authors address the large body of observational and experimen-
tal evidence underscoring the substantially lower risk of de-
veloping diabetes displayed by individuals who engage in
regular activity and have moderate to higher levels of cardio-
vascular fitness. Such extensive evidence supports the widely
held hypothesis that physical inactivity, which results in re-
duced metabolic efficiency in skeletal muscle tissue, is a
preventable cause of insulin resistance.

As populations within the United States and worldwide
become increasingly sedentary and as the prevalence of both
Type 2 diabetes and obesity steadily increase, metabolic and
cardiovascular health is an ever-increasing public health con-
cern. Fortunately, as these mini-reviews and numerous studies
illustrate, there is considerable evidence that physical activity
can be an effective and tangible means of treating and prevent-
ing the onset of diabetes and obesity. The monumental chal-
lenge at hand is the convincing of the general population to
incorporate daily physical activity into their lifestyles.

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F. W. Booth
W. W. Winder
Coordinating Associate Editors
E-mail: william_winder@byu.edu
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