The International Journal of Indian Psychology
ISSN 2348-5396 (e) | ISSN: 2349-3429 (p)
Volume 1, Issue 1, DIP: 18.01.013/20130101
DOI: 10.25215/0101.013
http://www.ijip.in | October- December, 2013

The International Journal of INDIAN PSYCHOLOGY

Research Paper

Fitness Workouts and Insulin Sensitivity

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ABSTRACT

To summarise, a large body of evidence suggests that physical conditioning is an important determinant of insulin sensitivity and overall glucose tolerance. Both acute and chronic physical training are linked to improved glucose disposal. Physical inactivity, on the other hand, leads to a decline in glucose tolerance. Muscle is the primary tissue in charge of accelerated glucose disposal following exercise. Enhanced glucose transport and increased glycogen synthesis are largely responsible for the improvement in glucose tolerance after an acute bout of exercise. Multiple factors appear to explain the beneficial effects of chronic physical training on glucose metabolism, including increased muscle mass, increased muscle blood flow and capillary area, increased mitochondrial oxidative enzyme capacity, and activation of the glucose transport system. Despite these well-documented effects of exercise on glucose metabolism, the exact role of exercise in the treatment of diabetic patients is unknown. Acute exercise has been shown to be a helpful adjunct in establishing good glycemic control in insulin-dependent (type I) diabetic individuals. However, little attention has been paid to the role of acute exercise in helping to smooth out glycemic control in non-insulin-dependent (type II) diabetic patients. The role of chronic physical training in the treatment of insulin-dependent (type I) and non-insulin-dependent (type II) diabetics is unknown.

Keywords: *Workout, Fitness, Insulin, Diabetes*

Over the last decade, there has been a resurgence of interest in using physical training as an adjunctive therapy to treat a variety of metabolic disorders, including diabetes, hyperlipidemia, and obesity. Such training programmes have also been shown to benefit blood pressure control, cardiovascular function, and the incidence of coronary artery disease (see review by Schneider et al. in this issue). Hyperglycemia, hyperinsulinemia, and hyperlipidemia have all been shown to be significant risk factors for cardiovascular disease. Numerous studies over the last few years have found that sedentary people have insulin resistance, glucose intolerance, and hyperlipidemia. Physical activity, on the other hand, has been shown to reverse all of these metabolic disturbances (see review by Schneider in this issue). In this review, we will look at the effects of repeated bouts of sustained physical exercise on insulin sensitivity in healthy subjects as well as in a variety of disease states characterised by insulin resistance (obesity, type I and type I diabetes meelitus). Furthermore,

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Received: November 1, 2013; Revision Received: December 21, 2013; Accepted: December 25, 2013

we will review recent research on the target tissues and cellular mechanisms by which physical exercise improves insulin-mediated glucose disposal.

A. Athletes Who Have Been Trained

Well-trained runners have low fasting plasma insulin levels and a reduced insulin response to glucose challenge despite normal glucose tolerance. This set of findings indicates the presence of increased tissue insulin sensitivity. Using the euglycemic insulin clamp technique," we looked at insulin-mediated glucose disposal in sedentary and well-trained athletes. In a nutshell, during an insulin clamp study, a primed-continuous infusion of regular insulin raises and maintains plasma insulin concentrations at approximately 100 pU/ml above fasting levels. The plasma glucose concentration is held constant at the basal level by adjusting an exogenous glucose infusion. The rate of glucose infusion must equal the rate of glucose uptake by all tissues of the body under these steady-state conditions of euglycemic hyperinsulinemia, providing a measure of tissue sensitivity to the infused insulin. It should be noted that this calculation assumes that endogenous hepatic glucose production is completely suppressed. If the liver's glucose production is not suppressed, the residual rate of hepatic glucose output (as measured with 3H-3-glucose) must be added to the rate of exogenous glucose infusion to determine the true rate of glucose uptake by the entire body.

B. Previously Sedentary Subjects Get Physical Training

Cross-sectional studies in runners do not reveal how much of the improved insulin sensitivity is due to training and how much is an inborn trait. The maintenance of high insulin sensitivity in master athletes after several years of deconditioning, in particular, would lend support to the inborn contribution theory. We studied six healthy, young (25 2 2 years) previously untrained subjects to see how regular training affected insulin sensitivity. The subjects completed a 6-week training programme that included 1 hour of cycle ergometer exercise four times per week at 70% of their V,max. The V02 max increased by 20% in each subject after the 6-week training programme (I' 0.01). Insulin-mediated glucose uptake increased by 30% using the insulin clamp technique, with no change in steady-state plasma insulin levels achieved during the study.

TYPE I DIABETES MELLITUS

Regular exercise has been suggested as one of the cornerstones of treatment for type I diabetes in addition to nutrition and insulin (IDD). The following two observations serve as the foundation for this advice: Acute exercise's ability to lower blood sugar levels and greater activity, such as that seen in summer camps for kids, both lead to decreased insulin requirements. However, it is unknown to what degree a decrease in insulin dosage in these studies is attributable to increased physical activity in and of itself as opposed to stricter dietary adherence under more stringent medical monitoring. We thought it would be interesting to investigate whether physical training could lessen the insulin resistance in these patients and, if so, whether overall glucose control would be improved. It is now well established that the majority of patients with long-term type I diabetes are insulin resistant.

TYPE 11 DIABETES MELLITUS

Those with impaired glucose tolerance and type I diabetic patients, whether or not they have fasting hyperglycemia, all have insulin resistance. Although type I diabetes and obesity are frequently linked, the development of insulin resistance, which is equally severe in type I diabetic patients who have normal weight, cannot be explained by fat. In the baseline state, the liver overproduces glucose despite heightened plasma insulin levels and an increased insulin release rate. These findings suggest that the liver is resistant to insulin's ability to

control hepatic glucose release in postabsorptive circumstances. In type I patients with fasting hyperglycemia, glucose absorption by peripheral tissues is increased in the postabsorptive state (which must be equal to the rate of endogenous hepatic glucose synthesis). Contrarily, in the insulin-stimulated condition, the reduction of hepatic glucose synthesis is normal, and it has been demonstrated that peripheral tissues are the main site of insulin resistance. A deficiency in insulin action is caused by reduced muscle glucose absorption, according to investigations using euglycemic insulin clamps, 3H-3-glucose, and femoral hepatic venous catheterization. Comparable outcomes have been attained after ingesting glucose.

In the Ruderman et al. investigation, oral glucose tolerance was unaffected by intravenous glucose; nevertheless, there was a very little but statistically significant increase in the K-rate of glucose disappearance. There hasn't been any training research in type I diabetes participants that has demonstrated a reduction in fasting plasma glucose levels. Even while there was no change in glucose tolerance, several trials, including our own, showed a trend for the plasma insulin levels to fall. These findings imply that exercise may have enhanced insulin sensitivity. In order to answer this, we measured the amount of insulin-mediated glucose clearance in six type I diabetic non-obese subjects before and after a conditioning regimen of cycle ergometer workouts lasting an hour, four times per week, at an intensity equal to 70% of their V.

EFFECT OF TRAINING IN NORMAL, OBESE, AND DIABETIC SUBJECTS

Our findings on the impact of exercise on insulin-mediated glucose metabolism. The ideal body weights of the control, non-insulin-dependent, and insulin-dependent diabetic participants were comparable, and in all three groups, they were much lower than those of the controls. It's interesting to note that obese, non-insulin dependent, and insulin-dependent diabetic participants saw a similar reduction in the absolute rate of glucose uptake and the insulin-mediated rate of glucose clearance during the insulin clamp trial (Figure 1).

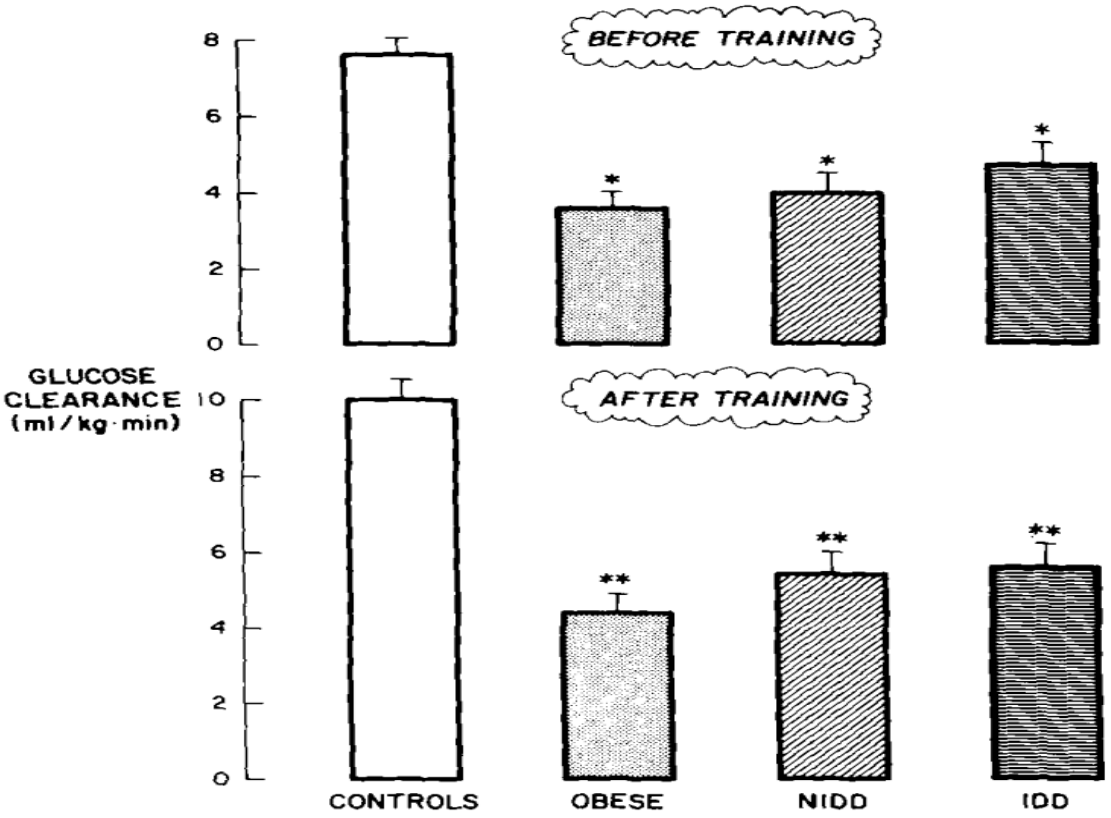

Figure 1 shows the insulin-mediated glucose clearance in normal-weight, non-diabetic, obese, and insulin-dependent diabetic participants before and after a 6-week physical training regimen. *P 0.01 compared to controls; **P 0.05 compared to the insulin clamp research conducted prior to training for each individual group.

Clearly in all four groups (controls, obese, NIDD, IDD). It's important to stress the following points: (1) The magnitude of improvement in insulin action was similar in control, obese, NIDD, and IDD subjects and amounted to a uniform increment of only 25–35 above the pretraining level in all groups. (2) The absolute rates of insulin-mediated glucose metabolisdclearance achieved posttraining remained well below those in age-matched controls (Figure 2). These findings imply that, regardless of the level of insulin resistance present prior to exercise, the effect of exercise is non-specific and will result in a comparable, small increase in insulin sensitivity. Hence, the improvement in insulin-mediated glucose elimination after training was of same magnitude in control subjects (who manifest normal insulin sensitivity) and in the obesekliabetic groups (who manifest severe insulin resistance).

EXTREME VERSUS CHRONIC EXERCISE: ACTION MECHANISM

The mechanisms by which acute and chronic exercise promote glucose elimination in healthy and diabetic people will be the main topic of the remaining section of this review. The significance of differentiating between the effects of acute versus chronic exercise has been highlighted by recent studies. The former are transient and vary depending on the kind, level, and length of exercise. On the other hand, frequent exercise is linked to long-term adaptations that often last a while after the exercise is discontinued. For these benefits, three training sessions (30–60 minutes each) must be completed over the course of at least 4-6 weeks. It is crucial to first look at the hormonal and substrate changes that take place after an acute bout of exercise in order to completely understand the mechanisms underlying the boost in glucose utilisation following any training programme. Here, they will be briefly discussed. The reader is directed to Bjorkman's article in the current issue for a more in-depth analysis of this topic.

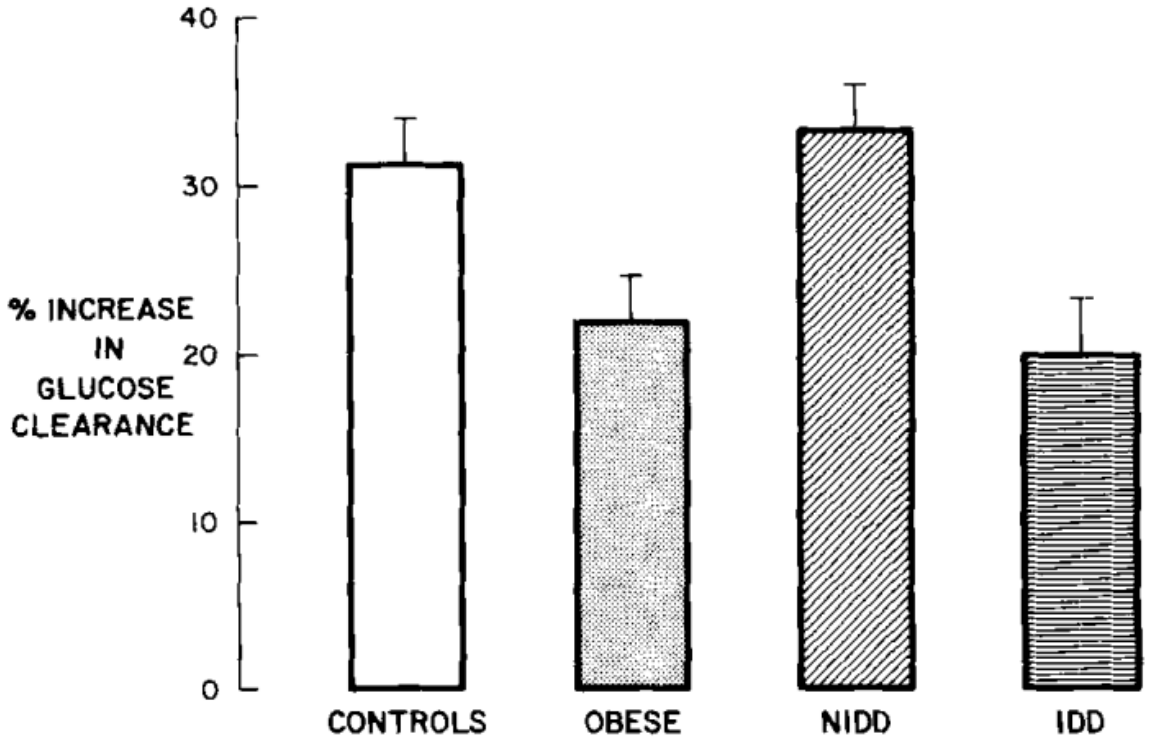

Figure 2 shows the increase in insulin-mediated glucose clearance during euglycemic insulin clamp studies conducted before and after a 6-week physical training programme in the control, nondiabetic obese, non-insulin-dependent diabetic, and insulin-dependent diabetic groups. The increase is expressed as a percentage above baseline. None of the four groups' incremental answers showed any discernible differences.

SUMMARY

In conclusion, a substantial body of research suggests that physical fitness is a key factor in determining insulin sensitivity and total glucose tolerance. Physical training throughout time, both acute and chronic, is linked to increased disposal of a glucose load. In contrast, a decline in glucose tolerance is brought on by inactivity. Muscle is the main tissue whose increased glucose elimination occurs after exercise. The improvement in glucose tolerance following a short period of exercise is primarily brought on by improved glucose transport and increased glycogen synthesis. Numerous factors, including increased muscle mass, increased muscle blood flow and capillary area, increased mitochondrial oxidative enzyme capacity, and activation of the glucose transport system, appear to be responsible for the beneficial effects of chronic physical training on glucose metabolism. The specific significance of exercise in the management of diabetes patients is still unknown, despite the well-documented effects of training on glucose metabolism. Acute exercise has been found to be a beneficial adjunct in establishing adequate glycemic control in people with insulin dependant (type I) diabetes. Acute exercise's potential to improve glucose control in non-insulin-dependent (type 1) diabetes patients hasn't gotten much attention, though. It is yet unknown how long-term physical exercise affects the management of both insulin-dependent (type I) and non-insulin-dependent (type 11) diabetic patients.

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Acknowledgements

I appreciate the participation of teachers, principals and the administrative staff of the schools for their kind assistance and showing their interest to complete this research efficiently. Also I am thankful to the tribal ministry of the state and ethical committee of the University for granting me permission to accomplish this study.

Conflict of Interest

The research is done with authentic data and field work hence, the author's professional judgment about research and report writing is not compromised.

How to cite this article: Damor J.D (2013). Fitness Workouts and Insulin Sensitivity. *International Journal of Indian Psychology*, *1*(1), 96-101. DIP: 18.01.013/20130101, DOI: 10.25215/0101.013