Effects of non-insulin-dependent diabetes on oxygen consumption during treadmill exercise

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ABSTRACT

REGENSTEINER, J. G., J. SIPPEL, E. T. McFARLING, E. E. WOLFEL, and W. R. HIATT. Effects of non-insulin-dependent diabetes on oxygen consumption during treadmill exercise. *Med. Sci. Sports Exerc.*, Vol. 27, No. 5, pp. 661–667, 1995. Persons with non-insulin-dependent diabetes mellitus (NIDDM), in the absence of complications, have a decreased exercise performance compared with nondiabetic subjects. However, degree of impairment and factors associated with reduced exercise performance have not been fully characterized. Maximal exercise performance using a graded treadmill protocol was assessed in 10 sedentary persons with uncomplicated NIDDM (aged 51 ± 7) and 10 healthy age- and activity-matched controls. Potential correlates of exercise performance measured included fasting and post-exercise glucose concentrations and fasting insulin concentration, hemoglobin A_1C, hematocrit, and whole blood viscosity. At maximal exercise, diabetic persons had a 24% lower maximal walking time and 20% lower maximal $\dot{V}O_2$ than controls (both $P < 0.05$), while hemodynamic measures did not differ between groups. During graded exercise, at work loads below the maximal one, the relationship between $\dot{V}O_2$ and work load was significantly lower in persons with NIDDM than controls by an average of 16%. No correlations were found between peak exercise performance and any of the potential correlates of exercise performance measured. We conclude that persons with NIDDM have an impaired peak exercise performance not associated with degree of glycemic control. The reduced rate of increase in oxygen consumption during increasing submaximal work loads in NIDDM suggests that limitations in oxygen delivery may impair exercise performance in otherwise healthy persons with diabetes.

EXERCISE TEST, HEMATOCRIT, GLUCOSE, BLOOD VISCOSITY

Some, although not all, previous studies have suggested that persons with uncomplicated non-insulin-dependent diabetes mellitus (NIDDM) have an impaired peak exercise performance compared with healthy age-matched controls (10,11,25,26). However, the range of impairment in peak exercise performance is large, varying from 0% to 28% (4,10–12,25,26). Factors that may affect the validity of this finding include the type of exercise protocol used, reproducibility of the exercise testing methods, and selection of appropriate age and activity-matched controls for comparison.

In diabetic individuals, there are several potential pathogenic mechanisms that may be associated with a decreased capacity for exercise. Previous studies have evaluated the effects of hyperglycemia on exercise performance (15,26) but associations have not been found between hemoglobin A_1C (HbA_1C_1) or fasting serum glucose concentration and exercise performance (15,26, 27). Alternatively, alterations in oxygen delivery in subjects with NIDDM may play a role, in that exercising cardiac output is reduced compared with normal subjects in some studies of uncomplicated diabetes (23). In addition, whole blood viscosity is increased in NIDDM (13,14), which may also affect oxygen delivery and exercise performance.

We hypothesized that persons with NIDDM would have a reproducible impairment in peak exercise performance as compared with age- and activity-matched controls. The submaximal $\dot{V}O_2$ response to graded exercise was measured to evaluate the relationship between submaximal work loads and oxygen consumption. We also measured fasting glucose level, fasting insulin level, whole blood viscosity, and other factors that could influence exercise performance in NIDDM. To accomplish these goals, we compared 10 age- and activity-matched persons with uncomplicated NIDDM to 10 healthy nondiabetic controls and found maximal exercise performance and submaximal $\dot{V}O_2$ response to graded exercise to be impaired in diabetic persons compared with healthy controls.

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TABLE 1. Characteristics of control subjects and persons with NIDDM.

	Control	NIDDM
N	10	10
Age (yr)	51 ± 7	50 ± 7
Gender (males/females)	6/4	6/4
Disease duration (yr)	—	6.7 ± 6.8
Weight (kg)	82.9 ± 17.4	90.7 ± 14.2
Body mass index ($kg \cdot m^{-2}$)	27 ± 4	30 ± 5
Habitual physical activity (MET hours·wk^{-1})	189 ± 55	203 ± 53

Values are mean ± SD. No significant differences were found between control and diabetic subjects.

METHODS

Subjects. Six men and four women with NIDDM were recruited and compared with 10 normal controls matched for age, sex, weight, and level of habitual activity (Table 1). No subject was more than 35 lbs over ideal body weight by standard tables. Two diabetic but no control subjects smoked. Participants had not been hospitalized in the last 6 months. The study was approved by the University of Colorado School of Medicine Human Subjects Committee, and written informed consent was obtained from all enrolled subjects.

Presence of NIDDM was documented by chart review which confirmed the diagnosis and type of treatment for NIDDM. Persons with NIDDM were included in the study if their diabetes was treated by diet ($N = 1$) or oral agents ($N = 9$), but not if they were treated with insulin, because these subjects tend to have more advanced disease. Other than oral agents, diabetic subjects were taking no other medicines. No person with diabetes having a fasting serum glucose over 250 $mg \cdot dl^{-1}$ on therapy was included.

Before the first exercise test, absence of comorbid conditions was confirmed by history, physical examination, and laboratory testing. Diabetic persons with clinically evident distal symmetrical neuropathy were excluded from further study, by evaluation of symptoms (numbness, paresthesia) and signs (elicited by vibration, pinprick, light touch, ankle jerks), because of possible effects on exercise performance (6). Persons with autonomic dysfunction (>20 mm fall in upright BP without a change in heart rate) were excluded.

Persons with NIDDM were excluded if they had evidence of ischemic heart disease by history or abnormal resting or exercise electrocardiogram (EKG) (≥1 mm ST segment depression). Persons with angina or any other cardiac or pulmonary symptoms potentially limiting exercise performance were excluded as well. Presence of systolic blood pressure >190 at rest or >250 with exercise or diastolic pressure >95 at rest or >105 with exercise was also grounds for exclusion. Subjects were excluded who had peripheral arterial disease as evaluated by resting and postexercise measurements of ankle brachial indices as previously described (9).

Subjects with proteinuria (urine protein > 200 $mg \cdot dl^{-1}$) or a creatinine ≥ 2.0 $mg \cdot dl^{-1}$, suggestive of renal disease, were excluded. Renal disease was grounds for exclusion since it can alter exercise performance (3,17).

Control subjects were screened identically to diabetic subjects but had a normal history and physical examination. These subjects were taking no medications and had a normal blood glucose level and hemoglobin A_1C.

Habitual physical activity level. All participants were sedentary, reporting that they did not exercise on a regular basis. Potential participants were excluded if they reported exercising for at least 20 min, more than two times a week. Sedentary behavior was confirmed by a questionnaire asking about physical activity levels over the previous week (24). Subjects were asked in a series of questions to itemize their time (reporting specific activities) into work, leisure, and housework categories (23). Questionnaire results were calculated in metabolic equivalents (MET) where 1 MET equals resting oxygen consumption (approximately 3.5 $ml \cdot kg^{-1} \cdot min^{-1}$). Only persons exercising at a level <260 MET $h \cdot wk^{-1}$ were accepted into the study as meeting the requirement for sedentary behavior (as determined in a previous study (21)).

Treadmill testing. In all participants, a graded treadmill protocol (modified Naughton) was performed to exhaustion after an overnight fast (2). The exercise test began at 2 mph, 3.5% grade, and the grade was increased by 3.5%, or the speed increased every 2 min (first grade is increased, then speed and so on) to peak exertion, using the same protocol for all patients. Because using the treadmill, workload cannot be calculated since treadmill walking is a weight-bearing exercise, time on the treadmill at increased grade and/or speed is used as a surrogate for increasing workload. Rates of oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) were measured, breath-by-breath, at rest, and during treadmill exercise, using an Ametek metabolic system (Ametek Thermox, Pittsburgh, PA). Oxygen consumption was normalized on a per kilogram (kg) basis. However, since persons with NIDDM tended to weigh more than control subjects (difference not statistically significant), $\dot{V}O_2$ was also normalized to height and body mass index (BMI), as well as being presented as an absolute value ($l \cdot min^{-1}$). Arm blood pressure (by auscultation) and heart rate (by 12-lead electrocardiogram) were obtained every minute during exercise. Cardiac status was monitored throughout the treadmill test by 12-lead EKG. In all subjects, maximal exercise performance was characterized by the longest walking time and the highest oxygen consumption attained during the treadmill test. Maximal oxygen uptake ($\dot{V}O_{2max}$) was defined in the present study as $\dot{V}O_2$ remaining unchanged or increasing less than 1 $ml \cdot kg^{-1} \cdot min^{-1}$ for 30 s or more despite an increment in work load (30). Maximal respiratory exchange ratio was calculated as $\dot{V}CO_2/\dot{V}O_2$. The submaximal $\dot{V}O_2$ values

shown were expressed as 30-s averages. After an initial screening treadmill test in all subjects, subsequent tests were performed on three occasions in persons with NIDDM (one test per week for three consecutive weeks) and on one occasion in normal controls. During all testing, subjects were allowed to lightly rest their hands on the treadmill bar for balance only, but not to grip the bar.

Blood collection and preparation. Blood lactate concentrations were measured only during the first maximal treadmill test (after the screening test) in all subjects. For the measurement of blood lactate concentration, a 20-g intravenous catheter was placed in a forearm vein, with a three-way stopcock to facilitate blood drawing, and patency was maintained with heparinized saline. For each sample, 50 μl of blood was withdrawn and immediately deproteinized in 3% perchloric acid, and then stored on ice. Blood for measurement of lactate concentration was drawn at rest, every minute during exercise, as well as 5 min after exercise. Blood for measurement of fasting serum glucose and insulin concentrations was drawn at rest before each test, and blood for measurement of post-exercise glucose concentrations was drawn immediately after exercise. Samples for whole blood viscosity (10 ml) were obtained without stasis, from the IV site before each treadmill test.

Assay methods. Lactate was assayed by a lactate dehydrogenase method (22). Serum insulin concentrations were measured by radioimmunoassay (19,31). Serum glucose concentration was measured by the glucose oxidase method (16). Whole blood viscosity was determined using a cone plate viscometer (Brookfield Engineering Laboratory, Inc, Stoughton, MA) (7), which has a precise rotating torque meter that can be driven at different speeds of rotation (shear rates) and that senses the resistance the sample imposes upon a flat surface (the plate). The viscometer permits the rapid analysis of small samples of blood under direct observation and obtains absolute values of shear stress. Readings from the viscometer are converted into viscosity (in centipoise) by applying the equation: Viscosity (centipoise) = (shear stress/shear rate) $\times$ 100. Viscosity measurements were obtained at shear rates of 5.75, 11.5, 23, 46, 115, and 230 s^{-1}.

Lactate threshold. The lactate threshold was determined as the point at which blood lactate concentration began to progressively increase (Fig. 1). Individual lactate thresholds are shown in Figure 1 for a representative diabetic patient and a healthy control.

Statistical analysis. A within-subjects analysis of variance was used for within-subjects comparisons, and a paired *t*-test was used for between-subjects comparisons. Reproducibility was determined from the standard deviation of the three repeated measurements within subjects (SD rep). This standard deviation describes the variability of the replicates for that measurement. A coefficient of variation was derived from the standard deviation of the replicates divided by the mean response value for each measurement. Correlations between normally distributed variables were determined by the Pearson's product moment correlation coefficient (r). Multivariate regression techniques were used to assess whether a variety of factors were associated with exercise performance. Values are given as mean $\pm$ SD and considered significant when $P < 0.05$, in a two-tailed test.

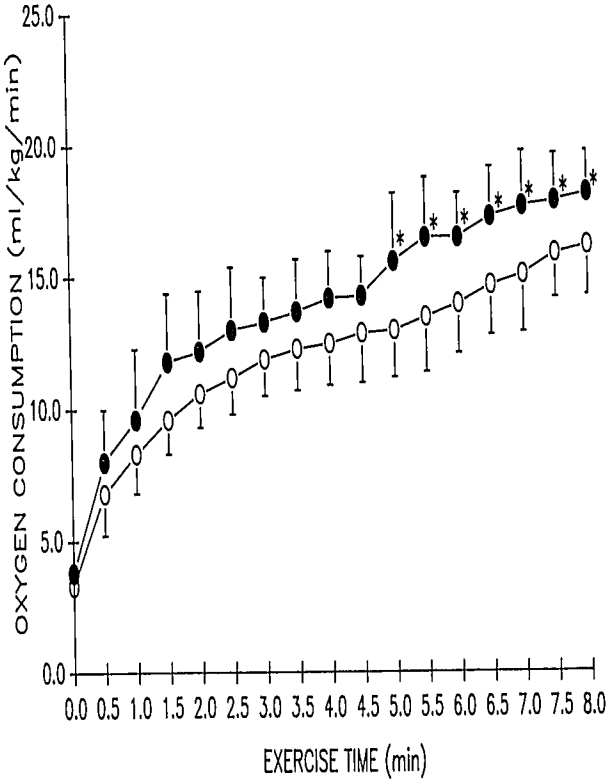

Figure 1—Representative lactate thresholds for one diabetic and one control subject. Lines through data points represent regression of the data below and above the lactate threshold. The inflection point for each subject is considered to be the threshold (as indicated by the *arrow*). The $\dot{V}O_2$ at that minute of exercise was identified as the $\dot{V}O_2$ of the threshold.

RESULTS

Subject characteristics. By design, persons with NIDDM were well-matched to nondiabetic controls in terms of age, sex, and level of habitual physical activity (Table 1). Subjects reported having had NIDDM for 6.7 yr (range was 0.5–19.0 yr). Subjects with NIDDM and controls did not differ significantly as to weight and body mass indices. However, both groups exceeded ideal body weight (as derived from standard tables). Persons with NIDDM were 25 $\pm$ 16% and controls were 18 $\pm$ 14% over ideal body weight (P = NS between groups).

Glucose and insulin concentrations, HbA_1C, hematocrit, and blood viscosity. Fasting serum glucose concentration was predictably higher in persons

TABLE 2. Assessment of fasting and post-exercise glucose and fasting insulin concentrations, HbA_1C, hematocrit, and viscosity in persons with NIDDM and controls.

	Control	NIDDM
Fasting serum glucose ($mg \cdot dl^{-1}$)	90 ± 9	188 ± 68*
Post-exercise serum glucose ($mg \cdot dl^{-1}$)	96.8 ± 19.0	188 ± 76
Fasting serum insulin ($\mu U \cdot ml^{-1}$)	10.7 ± 3.7	15.7 ± 7.5
Hemoglobin A_1C (%)	5.7 ± 0.4	10.1 ± 3.0*
Hematologic factors		
Hematocrit (%)	46.4 ± 3.7	48.4 ± 3.4
Hemoglobin ($gm \cdot dl^{-1}$)	15.3 ± 1.1	15.2 ± 1.7
Viscosity		
Viscosity (shear rate of 230 s^{-1}) (centipoise)	4.6 ± 0.5	5.0 ± 0.5*
Viscosity (shear rate of 115 s^{-1}) (centipoise)	5.1 ± 0.4	5.6 ± 0.5*
Viscosity (shear rate of 46 s^{-1}) (centipoise)	6.6 ± 0.8	7.5 ± 0.9*
Viscosity (shear rate of 23 s^{-1}) (centipoise)	8.5 ± 1.7	9.9 ± 1.1*
Viscosity (shear rate of 11.5 s^{-1}) (centipoise)	11.1 ± 3.1	12.0 ± 1.8
Viscosity (shear rate of 5.75 s^{-1}) (centipoise)	13.8 ± 5.6	14.5 ± 2.0

Variables shown were all measured during the first maximal exercise test for both diabetic and control subjects.
* $P < 0.05$ difference between means for persons with NIDDM and control subjects.

TABLE 3. Maximal exercise performance in control and NIDDM subjects.

	Control	NIDDM
Duration (min)	18.7 ± 3.7	14.2 ± 5.4*
Oxygen consumption measurements		
Maximal $\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	27.2 ± 5.3	21.5 ± 5.8*
Maximal $\dot{V}O_2$ ($ml \cdot BMI^{-1} \cdot min^{-1}$)	84.3 ± 17.4	63.5 ± 18.5*
Maximal $\dot{V}O_2$ ($ml \cdot cm^{-1} \cdot min^{-1}$)	1324.6 ± 302.5	1100.9 ± 275.7*
Maximal $\dot{V}O_2$ ($l \cdot min^{-1}$)	2.3 ± 0.6	1.9 ± 0.5*
Maximal RER	1.07 ± 0.05	1.10 ± 0.10
Maximal heart rate ($beats \cdot min^{-1}$)	161 ± 13	153 ± 18
Maximal systolic blood pressure (mm Hg)	191 ± 23	199 ± 17
Maximal diastolic blood pressure (mm Hg)	89 ± 7	95 ± 8

$\dot{V}O_2$, Oxygen consumption; RER, respiratory exchange ratio.
Results are presented from the first maximal exercise test for diabetic persons and the single maximal test for controls.
* $P < 0.05$ difference between persons with NIDDM and control subjects.

with diabetes than controls (Table 2) as was post-exercise glucose concentration. HbA_1C percentage was higher in diabetic than control subjects. Hematocrit was in the normal range (for an altitude of 5,280 ft) for both diabetic and control subjects (42%–54% for males and 40%–49% for females; normal values established in the Clinical Chemistry Laboratory of the University of Colorado). Hematocrit (and also hemoglobin) did not differ between diabetic and control subjects (Table 2). Whole blood viscosity was measured for six shear rates. Diabetic subjects had a higher whole blood viscosity than nondiabetics for shear rates of 230, 115, 46, and 23 (Table 2, all $P < 0.05$). For shear rates of 11.5 and 5.75, differences between groups were not significant (Table 2).

Maximal treadmill exercise performance. All subjects reached $\dot{V}O_{2max}$, defined as $\dot{V}O_2$ remaining unchanged or increasing less than 1 $ml \cdot kg^{-1} \cdot min^{-1}$ for 30 s or more despite an increment in work load (30). In addition, all subjects at peak exercise had a respiratory exchange ratio greater than 1.0 and in addition, cited exhaustion as their reason for stopping exercise.

Maximal walking time was 24% less in persons with NIDDM as compared with controls ($P < 0.05$) (Table 3). Maximal oxygen consumption was 21% less in persons with NIDDM than controls when normalized to weight in kilograms (Table 3). Importantly (because of the nonsignificant weight difference between persons with NIDDM and controls), $\dot{V}O_2$ was also less when normalized to BMI or height, as well as when not normalized to BMI, height, or weight (i.e., in $l \cdot min^{-1}$; all $P < 0.05$) (Table 3).

In spite of the lower exercise performance of diabetic subjects in terms of maximal walking time and $\dot{V}O_2$, the maximal respiratory exchange ratio, maximal heart rate, and maximal systolic and diastolic blood pressures did not differ between persons with diabetes and controls, suggesting that the exercise effort was similar between groups.

To allow an estimation of the reproducibility of performance variables, we conducted three maximal exercise tests in diabetic subjects over a 3-wk period. Maximal walking time varied less than 1 min and maximal $\dot{V}O_2$ varied less than 2 $ml \cdot kg^{-1} \cdot min^{-1}$ over repeat testing. Coefficients of variation were 7% for maximal walking time and 5% for maximal VO_2. Measurements of maximal heart rate, blood pressure, and RER were also highly reproducible. Coefficients of variation were 3% for maximal heart rate and for maximal RER, 6% for maximal systolic blood pressure and 2% for maximal diastolic blood pressure.

Submaximal VO_2 response to graded exercise. In addition to evaluating maximal exercise performance, we compared $\dot{V}O_2$ values between groups at each submaximal work load for which there was complete data for all twenty subjects in the study (through minute 8). Oxygen consumption per work load was significantly lower for minutes 5–8 in diabetic persons compared with controls (Fig. 2) and tended to be lower at all work loads below 5 min. The differences between groups per half minute averaged 16% over minutes 5–8. These differences remained whether $\dot{V}O_2$ was normalized to weight in kilograms, height in centimeters, or BMI, or presented as an absolute value ($ml \cdot min^{-1}$).

Lactate thresholds were clearly identified for all subjects. The absolute $\dot{V}O_2$ at the lactate threshold was lower in diabetic as compared with control subjects (13.5 ± 1.9 vs 16.0 ± 4.1 $ml \cdot kg^{-1} \cdot min^{-1}$, $P < 0.05$). However, when expressed as a percent of maximal $\dot{V}O_2$, the groups did not differ. In addition, at any minute of exercise, there were more diabetic persons exercising above the lactate threshold than controls. For instance, at 5 min of exercise, six subjects with NIDDM were above their lactate threshold in contrast to three control subjects.

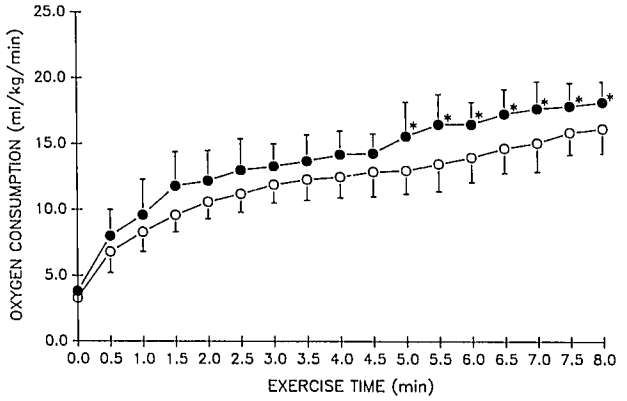

Figure 2—Oxygen consumption during submaximal exercise in diabetic (*open circles*) and control (*closed circles*) subjects. The diabetic subjects had a lower oxygen consumption than the nondiabetic subjects. *$P < 0.05$ between time points.

Predictors of exercise performance. As has been described for normal subjects (1), in persons with diabetes, age was inversely related to maximal $\dot{V}O_2$ ($y = -0.50x + 46.83$, $r = -0.64$, $P < 0.05$). In addition, weight, body mass index, and years of disease (diagnosed) were not related to exercise performance in diabetic persons.

There was no correlation between fasting insulin concentration, fasting or post-exercise glucose concentration, or percent HbA1C with any measure of maximal exercise performance in diabetic subjects. Hematocrit, although in the normal range, correlated with maximal walking time ($r = 0.67$, $P < 0.05$) and maximal $\dot{V}O_2$ ($r = 0.66$, $P < 0.05$) in diabetic persons only. Similar results were observed with hemoglobin where the correlation with maximal walking time was $r = 0.62$, and with maximal $\dot{V}O_2$ was $r = 0.60$, (both $P < 0.05$). However, whole blood viscosity did not correlate with exercise performance at any of six shear rates.

DISCUSSION

We found that maximal oxygen consumption was reproducibly lower in complication-free persons with NIDDM as compared with age and activity-matched controls. Not only was maximal exercise performance lower in NIDDM, but $\dot{V}O_2$ was also lower at submaximal work loads compared with control subjects. Finally, maximal exercise performance in persons with NIDDM was not associated with glucose or insulin concentrations, whole blood viscosity, or other clinical factors.

Since the population studied had a degree of obesity that may have been greater in the persons with diabetes, we normalized $\dot{V}O_2$ not only to kilograms of body weight, but also by height and BMI as well as presenting it as an absolute value (i.e., in $l \cdot min^{-1}$), at maximal and during submaximal exercise. We found that regardless of the way in which $\dot{V}O_2$ was normalized, oxygen consumption was consistently lower in persons with diabetes than controls at maximal exercise and during submaximal exercise. Importantly, if the difference in $\dot{V}O_{2max}$ between the two groups could be accounted for by differences in weight, we would expect that the NIDDM participants would have a *higher* absolute value rather than a lower one especially since treadmill exercise is weight bearing (29).

Because the presence and degree of exercise impairment reported in persons with diabetes differed between previous studies, it was important to establish the reproducibility of the finding to reliably determine the magnitude of any decrease in maximal performance in diabetic persons. Three replicates of the exercise test (in diabetic persons only) were performed and all measures of exercise performance were found to be highly reproducible. Intrasubject reproducibility of exercise performance in the NIDDM population could not be assumed since difficulty in obtaining a reproducible performance has been observed in other disease states (5,20). Nonreproducibility between tests in diseased groups may be due in part to the testing methods employed (30). For instance, when a diseased or even deconditioned person is tested using a protocol that increases workload very rapidly, the person may be forced to terminate exercise quickly, whereas a protocol that increases work load more slowly may allow for a better performance. Therefore, we selected a protocol that was appropriate to both a diseased population and a sedentary healthy one and were able to achieve a reproducible maximal exercise performance.

We found that the absolute oxygen consumption was lower, not only at maximal, but also at submaximal work loads in diabetic as compared with control subjects, a finding not previously observed. In healthy individuals, with increasingly progressive work, there is a predictable relationship between change in workload and change in $\dot{V}O_2$ (this rate of increase in $\dot{V}O_2$ is independent of $\dot{V}O_{2max}$ in normal untrained subjects) (8,18). In subjects with cardiovascular diseases such as congestive heart failure or peripheral arterial disease, there is a smaller increase in $\dot{V}O_2$ than expected with increasing submaximal workloads (8). The reduced rate of circulatory adjustment to an increase in workload is interpreted as reflecting the impaired oxygen delivery that is a characteristic of these disease states (8). Thus, the finding that the submaximal $\dot{V}O_2$ of diabetic subjects were lower than those of controls at submaximal work loads suggests the possibility that impaired oxygen delivery may limit exercise performance even in persons with uncomplicated NIDDM. In support of this concept, it has been observed that during exercise, persons with diabetes have an impaired cardiac output response to increasing workloads compared with nondiabetics, even when persons with autonomic dysfunction were excluded (23). However, in

subsequent studies, measurements of oxygen delivery and oxygen extraction need to be performed to fully evaluate the present findings.

Whole blood viscosity has previously been observed to be higher in diabetic than nondiabetic individuals (13,14). In the present study, although viscosity was higher in diabetic than nondiabetic individuals for four of six shear rates, there was no correlation between viscosity and exercise performance. In contrast, hematocrit, though in the normal range, was positively correlated with exercise performance in diabetic subjects. This finding suggests that a higher hematocrit may be beneficial in diabetes. Alternatively, persons with slightly higher hematocrits may have been generally healthier than those with lower hematocrits, even though indetectably so by our screening techniques. However, years of disease (diagnosed) did not correlate with exercise performance which would argue against differences in health status playing an important role in the diabetic individuals studied. Explanation of the finding of a relationship between hematocrit and exercise performance will require further study.

The lactate threshold is defined as the point at which the blood lactate concentration began to progressively increase. Previous studies have shown that when an individual performs exercise at a workload above his/her lactate threshold, fatigue occurs over a short time forcing the cessation of exercise (28). In contrast, exercise at workloads below the lactate threshold can be sustained for long periods of time (28). Thus, if the lactate threshold occurs at a lower absolute $\dot{V}O_2$, there are greater limitations on exercise performance. In the present study, we found that the lactate threshold occurred at a lower absolute $\dot{V}O_2$ in diabetic compared with control patients. In addition, not only was the $\dot{V}O_2$ at the lactate threshold lower in diabetic than control patients, but we also found that for any minute of exercise, there were more diabetic persons exercising above the lactate threshold than controls. For instance, at 5 min of exercise, six subjects with NIDDM were above their lactate threshold in contrast to three control subjects. This finding is further evidence that diabetic patients will have a more limited ability to sustain moderate exercise workloads than nondiabetics.

We found no correlations between exercise performance and fasting or post-exercise serum glucose concentrations, fasting insulin concentrations or HbA_1C in diabetic patients. It is important to note that correlations have not previously been found between any marker of glucose metabolism and exercise performance (15,27).

Most of the literature pertaining to exercise performance in diabetic persons is concerned with the metabolic consequences of exercise, either acute or chronic, rather than the functional ability to exercise *per se*. The importance of considering functional performance is that in persons with a chronic disease, the physical ability to carry out activities tends to decrease more dramatically than in nondiseased persons over time. This may be especially true for diabetic subjects who develop one of the common complications of diabetes, for instance, heart disease or peripheral arterial disease. Preservation of function thus assumes an even greater role than with nondiseased persons. Further studies should explore the mechanisms by which exercise performance is impaired in persons with NIDDM including evaluating more directly whether aspects of oxygen delivery are at least in part responsible for the impaired exercise performance in NIDDM.

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