Introduction

More than one third of adults in the United States have elevated cholesterol levels and are at increased risk for developing cardiovascular disease (1). To lower cholesterol, a class of drugs called statins are most commonly prescribed. Statins are very effective in lowering LDL cholesterol levels by up to 40%, but not without side effects. Approximately 25 million Americans use statins, and between 5-22% report some form of musculoskeletal pain, known as myalgia (1,2).

The most widely accepted theory as to why statin-induced myalgia occurs is that while the key enzyme in the HMG-CoA reductase pathway, HMG-CoA reductase, is inhibited, an important pathway is affected upstream (Figure 1). Not only is the production of cholesterol reduced, but the production of an essential protein in our bodies called ubiquinone is reduced as well. Ubiquinone is used in the electron transport chain in the mitochondria for production of ATP. When ubiquinone function is impaired, energy production is inefficient and metabolism is impaired (3). Historological evidence of mitochondrial dysfunction has been reported, with lipid accumulation and ragged red fibers identified in type I skeletal muscle fibers of statin users (3). These are the fibers that are most prominently activated during daily activities such as walking as well as endurance type exercises.

It is well known that statin use can lead to exercise intolerance, but little is known about their effects on athletes, specifically Masters swimmers whose primary form of exercise is non-weight bearing. This study aimed to assess the effect of statins on Masters swimmers between the ages of 55 and 75. We assessed their overall cardiorespiratory fitness and endurance in a VO2peak treadmill test, as well as substrate metabolism based on respiratory exchange ratio (RER) and blood lactate measures. These measures were taken during two separate sessions within a 10-week period. We hypothesized that the Masters swimmers who were on statins would have impaired fat metabolism, decreased aerobic endurance, and muscle symptom questionnaire was completed by each subject.

Methods & Materials

Subjects: 16 participants, 55-75 years of age 7 statin users and 9 non-statins users(Statin: 64.86 ± 2.38, 5 males and 2 females; Non-statin: 62.67 ± 2.02, 5 males and 3 females). All were Masters swimmers who train with minimal weight-bearing, leg-based exercises. Since statin-users are listed in Table 1. Written informed consent was obtained from all participants, and the study was approved by the Hamline University Institutional Review Board. A medical history, activity, and muscle symptom questionnaire were completed by each participant.

Experimental Design: Participants performed the following tests during two separate visits to the lab:

Statistical analysis: Continuous variables were analyzed using independent one-tailed t-tests, and ordinal variables were analyzed using the Mann-Whitney U test. Effect size was determined using an online effect size calculator (6). Differences were considered significant at p<0.05. Data were expressed as mean ± SE.

Session 1:

- **Fasted Blood Lipids, Triglycerides, and Glucose** were measured during session 1 using an Alere Cholesterol LDX System.

- **Resting Electrocardiogram (ECG)**: A resting ECG was recorded to screen for any heart abnormalities. The ECG was analyzed by a cardiologist before conducting the VO2peak test in session 2.

- **Fasted Respiratory Exchange Ratio (RER)**: A ten-minute sample of expired breath was collected and analyzed using a calibrated TrueOne 2400 Metabolic Measurement System (ParvoMedics).

- **Heart Rate**: Participants walked at a brisk pace while the incline was increased by 2% every 2 minutes until volitional exhaustion was reached. Oxygen consumption was measured using a calibrated TrueOne 2400 Metabolic Measurement System (ParvoMedics). E

- **Rating of Perceived Exertion (RPE)**: During the VO2peak test, participants were asked to give an RPE number between 1 and 10 once per minute.

- **Heart Rate**: During the VO2peak test, participants walked at a brisk pace while the incline was increased by 2% every 2 minutes until volitional exhaustion was reached. Oxygen consumption was measured using a calibrated TrueOne 2400 Metabolic Measurement System (ParvoMedics). E

- **Blood Lactate Levels**: Blood lactate levels were taken before and immediately following the VO2peak test using a LactatePlus device (Nova Biomedical).

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Results

<table>
<thead>
<tr>
<th>Total Cholesterol and LDL levels are significantly lower in the Statins group compared to the Non-Statins group.</th>
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<tbody>
<tr>
<td>Table 1. Participant characteristics at visit 1</td>
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<tr>
<td>Age (y)</td>
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<tr>
<td>Height (cm)</td>
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<tr>
<td>Weight (kg)</td>
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<td>BMI (kg/m²)</td>
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<td>Systolic blood pressure</td>
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<td>Diastolic blood pressure</td>
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<td>Resting heart rate (bpm)</td>
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<td>Metabolic Profile</td>
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<td>Glucose (mg/dL)</td>
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<td>Total cholesterol</td>
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<td>LDL</td>
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<td>Triglycerides</td>
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Values are mean ± SE. Significant differences 1, p<0.05.

Although there was no significant difference in VO2Peak or RER, a trend towards significance can be seen in Treadmill Endurance Time.

Conclusions

While no differences in VO2peak or RER were found between the two groups, a trend towards significance was found for treadmill endurance. We conclude that statin therapy may not significantly inhibit peak oxygen consumption, leg exercise endurance, nor fasting RER in this group of 16 participants. However, based on the moderate to large effect sizes, more research is warranted to further examine the differences between these measures in statin and non-statin users. It is possible that the training status and mode of exercise of these Masters swimmers had a protective effect against statin-induced muscle dysfunction. Further research should target a larger cohort of Masters athletes experiencing muscle pain or weakness in order to detect possible intolerance to statin therapy.

Literature Cited


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