INTRODUCTION

It is well known that thyroid hormones are necessary for the normal functions of the most tissues. In mammals, including humans, thyroid hormones are essential for the normal growth and development. In the adult animals, thyroid hormones exert their actions on the metabolic rate of the whole body, consequently almost all tissues participate on the increased calorigenesis through the elevation of the metabolisms of carbohydrates, proteins, lipids, nucleic acids, and ions. Since thyroid hormones affect many physiological processes and are required for the permissive actions of other hormones, it is not surprising that thyroid hormone deficiency may result in a wide variety of physiological and clinical disturbances, with abnormalities in functional and consequent clinical manifestations involving virtually every organ systems.

Thyroid deficiency affects the biochemical and mechanical properties of skeletal contractile function as well. Several studies have reported a clinical association between hypothyroidism and myopathy. Muscle weakness, fatigue and slow movement are the common observed symtomps in hypothyroid patients. The various contractile defects of skeletal muscle are also observed in hypothyroid state. The slow relaxation of skeletal muscle has been a well known clinical finding in hypothyroidism for many years. Reduction in muscle tension has been found in skeletal muscle obtained from hypothyroid rats. In addition, it has been reported that hypothyroidism frequently leads to the reduction in fatigue resistance of the skeletal muscle.

Biochemical evidence demonstrates that a reduction in mitochondrial activity resulting from the reduced content of mitochondria and associated

oxidative enzymes is a hallmark feature of hypothyroid muscle. Thus, the oxidative metabolism of skeletal muscle may be less efficient in hypothyroidism. This is in consistent with the reports that the maximal oxygen consumption ($\dot{V}o_2$ max) is depressed in this thyroid deficient state. Furthermore, previous studies in hypothyroid skeletal muscles illustrated a reduction in the myosin ATPase activity and impairment in the transport activity of sarcoplasmic reticulum. These biochemical alterations may contribute to the abnormalities of the contractile properties of hypothyroid muscle.

It is well known that regularly performed endurance training induces major adaptations in skeletal muscle including an increase in the mitochondrial content and respiratory capacity of the muscle fibers. Interestingly, previous studies have been reported that the reduction in oxidative enzymes and oxidative capacity in skeletal muscle of hypothyroid rats can be normalized by endurance training. This highlights the hypothesis that the impairments of muscle functions as a consequence of hypothyroidism may be altered or improved by endurance training.

Hypothyroidism is one of the most common endocrinopathies in adults. Although this condition can be clinically treated by thyroxine administration, some remained abnormalities in contractile properties of skeletal muscles are recognized as the important problems. Therefore, the purpose of this study was to ascertain the effects of exercise on the contractile properties of hypothyroid skeletal muscles. Since there were different abnormalities in contractile functions of hypothyroid muscles, depending on the muscle fiber types, the responses of fast and slow - twitch muscles were separately investigated.

The study was performed in the rats which were induced hypothyroidism by administration of antithyroid drug. The soleus muscle which contains primarily slow - twitch fibers and a small numbers of fast - twitch fibers, and the plantaris muscle which normally composes of fast - twitch fibers and a small proportion of slow - twitch fibers were studied. The model of exercise performed was swimming training.