

# **INTRODUCTION AND AIM OF THE WORK**

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Diabetes which is a major risk factor for arterioatherosclerosis mimicks an accelerated aging, at least as far as thickening of basement membranes, fibronectin and collagen biosynthesis are concerned (*Labat et al.*, 1991).

Thickening of the capillary basement membrane is a characteristic feature of diabetes and is considered to cause diabetic microangiopathy. Laminin, a glycoprotein in the basement membrane and type III procollagen peptide are increased in diabetic microangiopathy, suggesting that progressive changes in diabetic microangiopathy occur with their synthesis (*Okazaki et al.*, 1988). These appear to be good non-invasive markers for measuring basement membrane metabolism and type III collagen accumulation (*Okazaki et al.*, 1988).

Fibronectin is a high molecular weight  $\alpha_2$  glycoprotein produced by vascular endothelium. Its peculiar role in the structure of connective tissue together with its wide involvement in coagulative dynamics justified the increasing interest for fibronectin in the pathogenesis of diabetic vascular sequelae (*Nardelli et al.*, 1987).

The aim of the present work is to through more light on some factors involved in vascular complications in diabetes mellitus, namely plasma fibronectin, serum procollagen III and serum laminin in diabetics with normal fundus and in diabetics with retinopathy in a trial to clarify their possible role.