

CHAPTER 1. PREFACE

Obesity has long been recognized as an important risk factor for a large array of diseases. Epidemiological studies have shown the relationships between obesity and common diseases, such as hypertension, dyslipidemia and NIDDM (1-4). In Japanese, cardiovascular and cerebrovascular diseases have been main factors for mortality for many years (5). An increase in these vascular diseases and mortality may be related with obesity-related hypertension (6).

The possible mechanisms of obesity-related hypertension were previously addressed: 1) Deranged kidney functions such as renal hemodynamics and tubular reabsorption (7). 2) Insulin resistance and compensatory hyperinsulinemia (8, 9). and 3) Increased SNA (7). Increased SNA especially causes renal vasoconstriction and increase in tubular sodium reabsorption in kidney, increase in cardiac output in heart, and vasoconstriction in vascular bed (9). In addition, obesity-related hormones, such as *insulin and leptin*, are capable to increase SNA (10, 11). Therefore, the elevation of SNA would be a principal candidate for the cause of an obesity-related hypertension.

By the way, dietary constituents have changed from "*traditional high-carbohydrate diet*" to "*Western style high-fat diet*" in Japan. As the dietary style was changed, prevalence of obesity was increased in recent years. Thus, relevant obesity-related health problems are greatly associated with FAT. In animal studies, FAT is often used in order to induce dietary obesity and FAT-induced obese hypertensive animals are a model of dietary obesity-related BP elevation.

It is interest to investigate whether increased SNA contributes to FAT-related BP elevation, and what kinds of sympathetic activating factors relate to FAT-related BP elevation.