Rats, mice, and even turkeys have been selectively inbred to alter blood pressure. The primary use of many of these models has been to study the inheritance of specific traits rather than the physiology of blood pressure control. However, blood pressure control has been studied in two inbred strains of hypertensive rats in addition to the Okamoto and Dahl strains.

The New Zealand strain was developed in the late 1950's and was thus the first strain of genetically hypertensive rats. But, it has not been studied widely outside of New Zealand and the cause of the hypertension remains unknown. Plasma renin activity and sodium metabolism appear to be normal. Some indications of sympathetic overactivity have been found.

The Bianchi or Milan strain of genetically hypertensive rats was developed by inbreeding in Italy in the late 1960's. It has primarily been studied by the originators of the strain, who first demonstrated the possibility of renal cross-transplantation in the rat. It was then demonstrated that the chronic level of blood pressure after renal cross-transplantation was determined by the kidney, not the recipient.

Selective inbreeding has been employed in attempts to alter blood pressure in mice (837), rats, rabbits (11), chickens (897), turkeys (543), and other species. These studies have shown: firstly that pressure can be altered by inbreeding, secondly that pressure is under polygenic control, and thirdly that Mendelian dominance is not observed in blood pressure changes of genetic origin. A comprehensive review has been provided by Schlager (836).

The physiological basis of blood pressure increases and decreases has not been explored in most of these species.
But, in addition to the Okamoto and Dahl strains of rats, two other strains have been studied from a physiological viewpoint and are discussed further below.

THE NEW ZEALAND STRAIN OF GENETIC HYPERTENSIVE RATS

New Zealand hypertensive rats are often described as having "genetic" hypertension to help distinguish them from Okamoto rats with "spontaneous" hypertension. The New Zealand strain was originally developed in the laboratory of Professor Smirk and in 1958 it was the first strain of spontaneously hypertensive rats to be described (879). At first the incidence of hypertension was reported to be less than 100 percent but continued inbreeding has apparently improved this incidence to the full 100 percent level. The strain has remained primarily in New Zealand and has been studied extensively by investigators in Dunedin. Recently, Simpson and colleagues (866) have summarized these investigations.

Hemodynamics

Cardiac output has been reported to be normal in this model (134). Renal blood flow may be reduced a little bit (130).

Plasma volume, extracellular fluid volume, and total body water are normal (370) in young New Zealand rats that are in the process of becoming hypertensive. Plasma and extracellular fluid volume are low (369), (569), (568) in adult animals. Hematocrit is increased (369), (569). These data offer no support for the idea that volume expansion is playing a role in the genesis of the hypertension. But, the observation that venous compliance is decreased (863) complicates interpretation of the volume data.

Hormones

Plasma renin activity is a little low in this model (368), (569). No other hormones have been implicated.

Neural factors

Evidence for a nervous component in this genetic model