

## AMBULATORY BLOOD PRESSURE AND HEART RATE RESPONSES TO VEGETARIAN MEALS

S.E.G. SCIARRONE, M.T. STRAHAN, L.J. BEILIN, V. BURKE, P. ROGERS  
and I.L. ROUSE

Lactoovovegetarians have been reported to have lower blood pressures than meat eaters in both cross-sectional populations (Sacks et al. 1974; Rouse et al. 1983a) and randomised controlled intervention trials (Margetts et al. 1986; Rouse et al. 1983b). The mechanisms accounting for these blood pressure differences are unclear and the aim was to investigate these. It has been postulated that carbohydrate intake activates the sympathetic nervous system via post-prandial increases in glucose and insulin (Landsberg and Young 1983). Given the lower glycaemic index of a vegetarian diet compared to an omnivorous diet (Jenkins et al. 1981) it was hypothesised that reduced levels of post-prandial glucose and insulin following a vegetarian meal would lead to a reduced level of sympathetic activity and therefore blood pressure and heart rate compared to that seen after consuming an omnivorous meal. It was proposed that these effects may contribute to the lower blood pressure of lactovovegetarians overall.

A group of 20 normotensive, meat-eating males were matched for age and body mass index and randomly allocated to either an omnivorous or vegetarian diet for six weeks after two weeks of baseline measurements. Day-time blood pressure and heart rate responses to "standard" vegetarian and control meals were measured using ambulatory blood pressure monitors periodically throughout the intervention period. Plasma samples were taken approximately one hour after "standard" breakfasts for the determination of catecholamines, glucose and insulin levels.

Multiple regression analysis showed systolic blood pressure to be lower in the vegetarian group throughout intervention (coef.=-1.27, std err=0.45,  $P<0.01$ ) but this was unrelated to the time of the estimated post-prandial peak in plasma glucose. Diastolic blood pressure showed a post-prandial fall in both diet groups (coef.=-0.76, std err=0.16,  $P<0.01$ ) but a preprandial rise which was attenuated by consumption of the vegetarian diet (coef.=0.72, std err=0.16,  $p<0.01$ ). The vegetarian group had a lower heart rate throughout intervention (coef.=-3.59, std err=0.85  $P<0.01$ ). Both the vegetarian and the control group showed comparable post-prandial increases in heart rate (coef.=1.24, std err=0.27,  $P<0.01$ ).

No evidence of reduced sympathetic nervous system activity from plasma catecholamines or differences in plasma glucose and insulin could be found following ingestion of a vegetarian breakfast compared to a control breakfast. With respect to plasma levels this may have been a consequence of the timing of blood sampling and further studies are required in which plasma glucose, insulin and noradrenaline are monitored more closely throughout the day in conjunction with frequent monitoring of blood pressure and heart rate changes under controlled conditions. Consequently whether or not the lower heart rate and the blood pressure response to meals of the vegetarians can be attributed to a reduction in sympathetic nervous stimulation via a reduced (or delayed) blood glucose and insulin response to vegetarian meals could not be determined from this study.

In conclusion, ambulatory blood pressures are reduced on a lactovovegetarian diet in association with lower heart rates, suggesting a central nervous or cardiac mechanism. There was no clear evidence for an effect on blood pressure related to the lower glycaemic index of a lactovovegetarian diet although a pre-prandial effect on diastolic blood pressure was observed and this needs to be investigated further.

JENKINS, D.J.A., WOLEVER, T.M.S., TAYLOR, R.H., et al. (1981). *Am. J. Clin. Nutr.* 34: 362.

LANDSBERG, L., YOUNG, J.B. (1983). *Am. J. Clin. Nutr.* 38:1018.

MARGETTS, S.M., BEILIN, L.J., VANDONGEN R., ARMSTRONG, S.K. (1986). *Br.Mcd.J.* 293: 1468.

ROUSE, I.L., ARMSTRONG, B.K and BEILIN, L. (1983a). *J. Hypertens.* 1: 65.

ROUSE, I.L., BEILIN L.J., ARMSTRONG B.K and VANDONGEN R. (1983b). *Lancet* 1: 5.

SACKS, F.M., ROSNER, B. and KASS, E.H. (1974). *Am. J. Epidemiol.* 100: 390.