Lipid profile, plasma cortisol, and fasting glucose levels of young adult normotensive males with and without a family history of hypertension

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Normotensive individuals, in their forties, with a family history of hypertension have been shown to possess elevated circulating lipid[1] and plasma cortisol levels[2]. Cortisol has been shown to interact with sympathetic and renal function and influence blood pressure[3], whereas hyperlipidemia is associated with hypertension[4]. It is not clear, however, if the elevated levels of lipids and cortisol found in older normotensive individuals with a family history of hypertension (aged 40 years and over) are also present in the young. Consequently, the study aim was to examine lipid (total cholesterol, triglyceride, high and low density lipoprotein), fasting glucose, and plasma cortisol in a young normotensive adult population aged between 18 and 25 years. Blood (50 ml) was sampled (8-10 am) from a cannulated forearm vein, in male subjects with (n=50, +FH) and without (n=14, -FH) a family history of hypertension, after they underwent an overnight fast and 24 hour abstinence from caffeine-containing products and alcohol. FH was defined as having at least one parent or grandparent taking medication for hypertension. The average systolic blood pressure in the +FH group, was significantly higher (124 ± 1 mmHg) than for the –FH group (117 ± 3 mmHg, $P<0.05$). Plasma cortisol was higher in +FH (382 ± 22 nmol/L), compared with –FH subjects (370 ± 53) but the difference was not statistically significant. No significant difference was found in any of the following: lipid profile parameters, body mass index, resting heart rate, diastolic and mean arterial pressures and fasting glucose. It is concluded that unlike their older counterparts, young adult hypertensive offspring at the end of their second decade, display a normal profile for circulating blood lipids, plasma cortisol and fasting glucose.

(1) Hopkins PN et al (1996) Curr Opin Lipid 7: 241-253.
(2) al'Absi M, Arnett DK (2000) Biomed Pharmacother 54: 234-244.
(3) Fredrikson M et al (1991) Psychophysiol 28: 656-664.
(4) Neutel JM et al (1992) American Heart J 124: 435-440.