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| Title | Angiotensin-converting enzyme genotypes in hypertensive patients with left ventricular hypertrophy |
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| Citation | The 5th Medical Research Conference, Hong Kong, China, January 2000, v. 22 n. Supp 2, p. 35 |
| Issued Date | 2000 |
| URL | http://hdl.handle.net/10722/46872 |
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## S-CH-2

### ANGIOTENSIN-CONVERTING ENZYME GENOTYPES IN HYPERTENSIVE PATIENTS WITH LEFT VENTRICULAR HYPERTROPHY

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**Objective:** Hypertension, left ventricular hypertrophy (LVH) and angiotensin-converting enzyme homozygous deletional (DD) genotype are all cardiovascular risk factors which, if present together, may confer especially high risk. We and others previously reported a decrease with age in the prevalence of the DD genotype in hypertensive patients, which may be due to decreased survival. In this study, we hypothesise that there might be fewer patients with the DD genotype among patients with hypertensive LVH.
**Methods:** 70 hypertensive patients and 102 normal healthy controls were studied. LVH was determined by echocardiography in hypertensive patients. DNA was extracted from leucocytes and amplified by PCR using specific primers. Insertion (I) or deletion (D) alleles were identified after electrophoresis of PCR products.
**Results:** The left ventricular mass index (LVMI) in hypertensive patients with DD, ID and II genotypes were (mean ± SE) 111±6, 115±5 and 136±8 g/m$^2$ respectively (p = 0.03).

| | n | DD | ID | II | D | I |
|---|---|---|---|---|---|---|
| Normal controls | 102 | 22% | 32% | 46% | 0.38 | 0.62 |
| Hypertensives | 70 | 14% | 37% | 49% | 0.33 | 0.67 |
| without LVH | 33 | 24% | 36% | 39% | 0.42 | 0.58 |
| with LVH | 37 | 5%* | 38% | 57% | 0.24** | 0.76 |

*$\chi^2$ = 4.4, p = 0.04**$\chi^2$ = 5.2, p = 0.02

**Conclusion:** The lower LVMI in hypertensive DD patients and the lower prevalence of the DD genotype in hypertensive patients with LVH are consistent with increased cardiovascular mortality in patients with hypertension, LVH and DD genotype. The striking mortality reductions observed in the HOPE study may be related to the ability of ACEI to address all of these risk factors.

## S-CH-3

### PLASMA BRADYKININ LEVEL AND ANGIOTENSIN-CONVERTING ENZYME GENE POLYMORPHISM IN HYPERTENSIVE PATIENTS

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**Introduction:** Bradykinin is a vasodilator which may be involved in the pathophysiology of hypertension. Its degradation is mediated by angiotensin converting enzyme (ACE). An insertion/deletion polymorphism of the ACE gene is a marker of ACE activity. We previously observed for the first time that plasma bradykinin level is related to the ACE genotype. Our aim was to confirm this novel finding in a larger group of patients.
**Methods:** 52 patients with essential hypertension (21 men, 31 women; mean age 50, range 24-75) and 6 normal subjects (3 men, 3 women; mean age 31, range 23-37) were studied with informed consent. Venous blood (3 ml) was drawn from a forearm vein and put into pre-chilled polypropylene tubes containing protease inhibitors. These samples were promptly centrifuged and the plasma stored at -40°C. Plasma bradykinin was measured by radioimmunoassay. DNA was extracted from the buffy coat and amplified by polymerase chain reaction (PCR) using specific primers. The insertion and deletion alleles were identified as separate bands after electrophoresis of PCR products. **Results:** Mean plasma bradykinin level was 89.1±7.9 pmol/l in hypertensive patients and 11.6± 1.4 pmol/L in normal subjects (p < 0.001). Untreated hypertensive patients had higher levels than those on treatment (136.5±26.6 vs. 82.9±7.9 pmol/L, p = 0.03). Plasma bradykinin level correlated with ACE genotype (r = 0.30, p = 0.03) but was not related to age or gender. The levels in patients with the DD, ID and II genotypes were 33.7± 12.2, 89.2± 12.2 and 100.4± 11.3 pmol/L respectively (p = 0.004). **Conclusions:** These results in a new series of patients confirm our previous finding that the insertional genotype is associated with higher plasma bradykinin level in hypertensive patients. The higher level of bradykinin in hypertension may be a consequence of raised blood pressure as antihypertensive treatment appears to reduce it. Further studies are needed to clarify the role of bradykinin in cardiovascular diseases in man.