Key Words: Angiotensin II, Collagen, Cardiac Fibroblasts

P-182
ANGIOTENSIN II-INDUCED STIMULATION OF COLLAGEN SECRETION AND PRODUCTION IN CARDIAC FIBROBLASTS

The possible contributions of the angiotensin receptor subtypes 1 (AT₁) and 2 (AT₂) to angiotensin II (Ang II)-induced changes in collagen secretion and production were studied using the specific angiotensin AT₁-receptor and AT₂-receptor antagonists telmisartan and P-186, respectively.

Cardiac fibroblasts (from normal male adult rats) from passage 2 were cultured to confluence and incubated in the presence of 10⁻⁴ to 10⁻⁶ M Ang II in serum-free Dulbecco’s MEM medium for 24 hours. Collagen production and secretion were assayed by [³H]-Proline incorporation: non-collagen production and secretion were also calculated.

Ang II dose-dependently increased collagen secretion and production in rat adult cardiac fibroblasts in culture. Non-collagen secretion and production were also concentration-dependently increased by Ang II. Addition of 100 nmol/l Ang II increased (p < 0.01) collagen secretion and production by 75±6 (SEM) % and 113±23%, respectively. Pretreatment of cardiac fibroblasts with telmisartan completely blocked the Ang II-induced increase in collagen secretion (p < 0.001) and production (p < 0.01) and in non-collagen secretion (p < 0.01) and production (p < 0.01). P-186 had no effect on the Ang II-induced increase in collagen secretion and production. Addition of telmisartan and P-186 did not affect collagen secretion and production in basal cardiac fibroblasts.

Our data demonstrate that the effects of Ang II on collagen secretion and production in adult rat cardiac fibroblasts in culture are AT₁-receptor mediated, since they were abolished by the specific AT₁-receptor antagonist, telmisartan, but not by the specific AT₂-receptor antagonist, P-186.

Key Words: Angiotensin II, Collagen, Cardiac Fibroblasts

P-183
STIMULATION OF COLLAGEN GEL CONTRACTION BY ANGIOTENSIN II (1-8) AND ANGIOTENSIN II (2-8) IN CARDIAC FIBROBLASTS

The aim of the present study was to investigate whether collagen gel contraction can be induced by cardiac fibroblasts in serum-free conditions and whether angiotensin II (1-8) and its fragments angiotensin II (2-8), II (1-7), II (3-8), II (4-8) or II (1-4) can stimulate this contraction in serum-free conditions.

Cardiac fibroblasts (from normal male adult rats) were cultured to confluence and added to a hydrated collagen gel in a Dulbecco’s Modified Eagle’s Medium without fetal bovine serum for 1, 2 or 3 days. Angiotensin II (1-8) stimulated dose-dependently the contraction of collagen mediated by cardiac fibroblasts after 1, 2 or 3 days of incubation. Telmisartan completely blocked the angiotensin II (1-8)-induced collagen contraction by cardiac fibroblasts while P-186 and des-Asp¹-Ile⁸-angiotensin II had no effect. Angiotensin II (2-8) also stimulated the contraction of collagen mediated by cardiac fibroblasts after 1, 2 or 3 days of incubation. This contraction was completely blocked by des-Asp¹-Ile⁸-angiotensin II and telmisartan. Angiotensin II (3-8), (4-8), (5-8), (1-7) and (1-4) did however not affect the collagen gel contraction by cardiac fibroblasts.

Angiotensin II (1-8) and (2-8) stimulate the collagen contraction through specific angiotensin II subtype receptors.

Key Words: Angiotensins, Collagen, Cardiac Fibroblasts

P-184
IMPACT OF AMBULATORY BLOOD PRESSURE CRITERIA FOR WHITE COAT HYPERTENSION ON CARDIAC STRUCTURE IN A BIRACIAL POPULATION IN CONNECTICUT
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Medicare approved the use of ambulatory blood pressure monitoring (ABPM) and indicated that it “will be covered for those with suspected white-coat hypertension” (WCH). The document recommended a 24-hour average of <135/85 mmHg be used to define WCH when office BP was >140/90 mmHg. We examined the effects on cardiac structural parameters of 3 levels of 24-hour BP to define WCH in 62 never treated hypertensive (HTN) and 30 normotensive (NTN) control subjects from a biracial population in Connecticut. Cardiac structural parameters in patients with WCH from a biracial population in Connecticut approach those in normotensive subjects when 24-hour ABPM level is <130/80 mmHg. Lower levels of ABPM are preferred for the diagnosis of WCH until prospective randomized trials with clinical endpoints are completed.

<table>
<thead>
<tr>
<th>Variable</th>
<th>NTN</th>
<th>WCH (A)</th>
<th>WCH (B)</th>
<th>WCH (C)</th>
<th>HTN*</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>30</td>
<td>8</td>
<td>18</td>
<td>29</td>
<td>32</td>
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<tr>
<td>Age, yrs</td>
<td>51</td>
<td>53</td>
<td>50</td>
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<td>49</td>
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<td>BMI, kg/m²</td>
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<td>29.8</td>
<td>29.8</td>
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</tr>
<tr>
<td>24-h BP, mmHg</td>
<td>121/71</td>
<td>125/76</td>
<td>129/77</td>
<td>130/80</td>
<td>148/91</td>
</tr>
</tbody>
</table>

A: 24-h BP < 130/80, B: 24-h BP < 135/85, C: 24-h BP < 140/90, * 24-h BP > 140/90

Key Words: Ventricular Hypertrophy, Ambulatory Blood Pressure, Hypertension

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