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Possible cause of taurine-deficient cardiomyopathy: potentiation of angiotensin II action.

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Abstract

Taurine, an amino acid that exhibits anti-angiotensin II and osmoregulatory activity, is found in very high concentration in the heart. When the intracellular content of taurine is dramatically reduced, the heart develops contractile defects and undergoes an eccentric form of hypertrophy. The development of myocyte hypertrophy has been largely attributed to angiotensin II, whose growth properties are antagonized by taurine. Overt heart failure is usually associated with myocyte death, including death due to angiotensin II-induced apoptosis. However, the effect of taurine deficiency on angiotensin II-induced apoptosis has not been examined. To investigate this effect, taurine-deficient cells, produced by incubating rat neonatal cardiomyocytes with medium containing the taurine transport inhibitor, beta-alanine, were exposed to angiotensin II. The peptide increased terminal deoxynucleotidyl transferase-mediated deoxyuridine triphosphate nick end-labeling (TUNEL) staining and caspase 9 activation more in the taurine-deficient than the normal cell. Angiotensin II also promoted the translocation of protein kinase C (PKC)epsilon and PKCdelta, the expression of Bax, and the activation of c-Jun N-terminal kinase (JNK), effects that were greater in the taurine-deficient cell. However, the data ruled out a role for extracellular signal-related kinase (ERK), Bad, and p38 mitogen-activated protein kinase in the beta-alanine-angiotensin II interaction. Because PKC and JNK affect the expression and phosphorylation state of certain Bcl-2 family members, they appear to contribute to the potentiation of angiotensin II-induced apoptosis by taurine deficiency.

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