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HISTAMINE AND RELAXIN: AN INTRIGUING CONNECTION

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Histamine is released during cardiac anaphylaxis and ischemic-reperfusion injury, producing severe arrhythmias, cardio-depressive effects and coronary spasm.
Relaxin (RLX), formerly known for its effects on reproduction and pregnancy, has been shown to be a pleiotropic hormone, targeting numerous non-reproductive organs. Relaxin, at 10 ng/ml, promotes dilation of blood vessels in guinea pig isolated heart; inhibits the release of histamine; depresses aggregation of platelets induced by thrombin from 82 to 40 % of maximum (p B 0.001) and
their release from megakaryocytes and contributes to the regulation of fluid balance. Experimental studies performed in vascular and blood cell in vitro and in animal models of vascular dysfunction as well as pioneer clinical observations, have provided evidence that RLX prevents and/or improves cardiovascular diseases, such as ischemia–reperfusion and heart failure. Concerning the mechanisms of action of relaxin and histamine, stimulation of nitric oxide (NO) generation, with consequent rise in intracellular cyclic GMP (from 4 to 28 pg/mg of protein) production has been demonstrated to occur in the target cells and organs.
Dimaprit, a histamine H2 receptor agonist, decreases the amount of histamine released during the first 5 min of cardiac anaphylaxis from 4.2 to 1.4 lg/g of tissue and controls the positive inotropic and chronotropic responses of the isolated heart taken from actively sensitized guinea pigs, an effect mediated by NO-cGMP pathway.

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