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Hormonal Heart-Mind Connections: Clinical and Research Implications

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Oxytocin (OX) is an 8-amino acid peptide that is secreted in greatest concentration from the pituitary. It is best known for its roles in parturition and milk letdown reflex in nursing mothers, but males have OX in as great quantities in the brain and pituitary as females and have equal receptors. Recent discoveries have established OX as an essential hormone to facilitate prosocial behaviors such as trust, empathy, compassion, and generosity. It was shown to be deficient in children who spent their early years in orphanages with little human contact and has been shown to promote social engagement in autistic children. It is released by nurturing behavior such as massage, singing, and dog-owner bonding as well as exercise, sex, and fatty and sweet foods. This study looks at the OX system and demonstrates CNS-cardiac connections and their implications.

There is an extensive OX receptor network in multiple organs in the body, including the brain, hypothalamus, pituitary, uterus, breast, and kidney, with the heart demonstrating a concentration of both receptors and an OX synthetic system second only in size to the hypothalamus. The role of OX in the heart has yet to be established. OX is secreted in a pulsatile manner and has a plasma $t_{_{1/2}}$ of 10 to 15 minutes.

^{11°} OX causes release of atrial natriuretic peptide (ANP), which has been shown to account for most cardiovascular effects of OX. ANP has been shown to suppress adrenocorticotropic hormone secretion and turn off the sympathetic stress response. Additionally, it has been shown to have significant antiinflammatory effects, promote myocardial revascularization and vascular endothelialization, lower heart rate, prevent endothelial disruption, antagonize the effects of aldosterone and renin on the myocardium and cardiac vasculature, decrease myocardial and endothelial fibrosis, limit cellular damage and post-myocardial infarction death, and decrease reperfusion injury.

The biobehavioral psychophysiologic implications of these connections for disease prevention, and postincident survival in patients who are post-MI, poststent and post-CABG are vast. Some questions: Does heart rate variability training increase OX and ANP? Does change in these hormones predict survival? Does caregiver compassionate behavior influence ANP and OX secretion and survival? What relationship do OX and ANP have with telomerase and telomere length, which are known stress markers and survival predictors? Can peptide analogues be used therapeutically? Can prosocial behavior training of patients and significant others change OX and ANP levels and influence survival? Are one-time measurements or salivary and urine measurements reliable for assessment?