Cardiovascular Action of Oxytocin

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Rec Date: October 29, 2014; **Acc Date:** October 30, 2014; **Pub Date:** November 04, 2014

Editorial

Oxytocin, a neuropeptide that participates in mammalian birth and lactation, is produced primarily in the hypothalamus. Oxytocin, acting in the central nervous system, plays an important role in a variety of complex social behaviors in mammals. Recent studies have suggested that oxytocin is endowed with plerotropic effects on cardiovascular system, intrinsic oxytocin system is critical for maintenance of cardiovascular homeostasis [1,2]. It has also been proposed that oxytocin may work directly on vascular cells to slow the progression of pathophysiological processes involved in cardiovascular diseases [3].

Oxytocin is synthesized and released in the heart and the

baseline blood pressure, an enhanced baroreflex gain and an enhanced pressor response to oxytocin [24], while overexpression of oxytocin receptors in the hypothalamic paraventricular nucleus increased baroreceptor reflex sensitivity and buffers blood pressure variability in conscious rats [25], suggesting that endogenous oxytocin in central neural system functions as a vasopressor peptide to enhance pressor response in normal condition. However, oxytocin knockout mice also exhibited an increased pressor response to chronic stress, suggesting that oxytocin has an inhibitory effect of stress-induced pressor response [26], because it has been proposed that oxytocin may act as an anti-stress hormone with regards to the cardiovascular axis [27]. Inhibitory effects of pressor response to chronic stress may be related to anti-stress effect of oxytocin. Blood volume is essential for blood pressure, particular for long-term regulation of blood pressure. It has been observed that isotonic volume expansion by intra-atrial injection of isotonic saline induced a rapid increase in plasma oxytocin and ANP concentrations and a concomitant decrease in plasma vasopressin concentration, and that oxytocin (I.P.) injected caused a significant increase in urinary osmolality, natriuresis and plasma ANP level [28]. Because it is known that loss of blood volume stimulates release of vasopressin from hypothalamus-pituitary, which decreases release of ANP from atria. It has been hypothesized that volume-expansion stimulates release of neuropeptide oxytocin from hypothalamus-pituitary into blood, which circulated to the atria to stimulate ANP release and promote natriuresis [28]. Therefore, a

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