



Autonomic Nervous System: Chapter 3. Cotransmission in the autonomic nervous system (Handbook of Clinical Neurology)

Geoffrey Burnstock

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After some early hints, cotransmission was proposed in 1976 and then “chemical coding” later established for sympathetic nerves (noradrenaline/norepinephrine, adenosine 5'-triphosphate (ATP), and neuropeptide Y), parasympathetic nerves (acetylcholine, ATP, and vasoactive intestinal polypeptide (VIP)), enteric nonadrenergic, noncholinergic inhibitory nerves (ATP, nitric oxide, and VIP), and sensory-motor nerves (calcitonin gene-related peptide, substance P, and ATP). ATP is a primitive signaling molecule that has been retained as a cotransmitter in most, if not all, nerve types in both the peripheral and central nervous systems. Neuropeptides coreleased with small molecule neurotransmitters in autonomic nerves do not usually act as cotransmitters but rather as prejunctonal neuromodulators or trophic factors. Autonomic cotransmission offers subtle, local variation in physiological control mechanisms, rather than the dominance of inflexible central control mechanisms envisaged earlier. The variety of information imparted by a single neuron then greatly increases the sophistication and complexity of local control mechanisms. Cotransmitter composition shows considerable plasticity in development and aging, in pathophysiological conditions and following trauma or surgery. For example, ATP appears to become a more prominent cotransmitter in inflammatory and stress conditions.

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