Commentary: Chromaffin Cell Plasticity in Stress, Inflammation, Development, and Disease

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Chromaffin cells secrete catecholamine in response to acetylcholine stimulation released from splanchnic neurons. Various hormone and peptides co-released with catecholamine and acetylcholine have been shown to cause paracrine or autocrine regulation of stimulus–secretion coupling in chromaffin cells in normal conditions and during stress. In this meeting, several talks focused on this subject in three different sessions chaired by *Youssef Anouar* and *Corey Smith and Kathrin Engisch*. Several papers presented focused on the responses induced by pituitary adenylate cyclase activating polypeptide (PACAP), a neuropeptide co-released with acetylcholine from presynaptic terminals. *Lee Eiden* (USA) gave two talks: first, he reported a thorough microarray analysis of the transcriptomes induced by TNF and PACAP, respectively. Secondly, he reported the genes that were upregulated during restraint stress and hypoglycemia in adrenal medulla. Among them was galanin, a peptide implicated in the modulation of catecholamine release. The galanin transcript, as well as transcripts encoding catecholamine synthesizing enzymes (TH and PNMT) were upregulated by PACAP via a PKA-independent, ERK1/2-dependent pathway, during stress. It appears that PACAP regulates catecholamine secretion at different levels. *S.-A. Chan* (USA), from *Corey Smith's* group, reported that PACAP stimulates a phosphorylation-dependent recruitment of T-type Ca^{2+} channels leading to an increase of catecholamine secretion. *Eiden* together with *Smith* have presented

their work in a review on PACAP as a regulator of stimulus–secretion–synthesis coupling at the sympathoadrenal synapse during stress (below). *Stephen Bunn* (New Zealand) highlighted the importance of bidirectional signaling between the immune and neuroendocrine systems. He found that treatment of chromaffin cells with the cytokine IL-6 activated the ERK1/2 signal transduction pathway and induced significant changes in gene transcription. An extensive review of this work implicating the physiological significance of cytokine signaling on chromaffin cells during stress is presented by *Bunn* and *Eiden* (below).

In addition to the classical model used for the study of the molecular mechanisms involved in stimulus-secretion coupling, chromaffin cells have also been used as a model for the study of cell survival and differentiation. *Youssef Anouar* (France) provided new evidence to support that PACAP-regulated selenoprotein T expression is involved in chromaffin cell survival during development. *Vladimir Vukicevic* (Germany) reported the isolation and characterization of the progenitors from adrenal medulla. He demonstrated that these cells can self-renew and differentiate into cells with characteristics of dopaminergic neurons. A review of the regulation of the progenitor state of chromaffin cells is presented below. Indeed, the presence of such progenitor/stem cells in the adrenal medulla is very exciting and raises the possibility of a potential source of dopaminergic neurons for treating neurodegenerative diseases such as Parkinson's disease.

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