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Do adrenal neuroendocrine and steroidal cells express the GluA2 ionotropic glutamate receptor?

GluA2 is an ionotropic receptor that is activated by glutamate, an excitatory neurotransmitter. It is widely expressed in neurons in the central nervous system where it mediates the fast component of synaptic transmission. Several studies have also reported the presence of mRNA for multiple glutamate receptors (including GluA2) in the rodent adrenal gland. This is unexpected because glutamate is not thought to be a neurotransmitter in the peripheral (efferent) nervous system. However, the presence of mRNA for GluA2 does not necessarily imply the presence of the GluA2 protein. In order to confirm the presence and location of GluA2 in the adrenal gland, we implemented immunohistochemistry by staining for GluA2 in the mouse adrenal.

Adrenal cryosections were prepared from adult male and female mice, then incubated overnight with anti-GluA2 monoclonal antibodies. Immunoreactivity was revealed using HRP-coupled secondary antibodies and DAB. The adrenal gland contains an outer cortex which secretes steroidal hormones and an inner medulla which releases the catecholamine hormones, adrenaline and noradrenaline during the fight-or-flight response. GluA2 immunoreactivity was observed in the cortex and in the neuroendocrine chromaffin cells in the medulla. However, there was a thin band of unstained cells located between the medulla and the cortex. We hypothesized that the thin band might have been the remnants of the X-zone. The latter is a transient zone in the mouse adrenal that is thought to correspond to the human fetal zone. Previous work in mice has shown the X-zone develops after birth, then is substantially lost by P21 in male mice and after pregnancy in female mice. The function of the X-zone is not known.

To clarify whether X-zone cells lack GluA2 expression, we are now co-staining the adrenal gland for both GluA2 and a marker of the X-zone, AKR1C1. Our preliminary results are thus generally consistent with previous studies localizing GluA2 within the adrenal but suggest that its expression is selectively down-regulated in the x-zone. In future experiments, directly measuring the consequence of GluA2 activation may provide a direct way to determine the role of this receptor in adrenal function. We hypothesize that GluA2 activation would lead to the release of glucocorticoid and catecholamine hormones during the stress response.