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Decreased Numbers of Somatostatin-Expressing Neurons in the Amygdala of Subjects with Bipolar Disorder or Schizophrenia: Relationship to Circadian Rhythms

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Abstract:

**Background:** Growing evidence points to a key role for somatostatin (SST) in schizophrenia (SZ) and bipolar disorder (BD). In the amygdala, neurons expressing SST play an important role in the regulation of anxiety, often comorbid in these disorders. We tested the hypothesis that SST-immunoreactive (IR) neurons are decreased in the amygdala of subjects with SZ and BD. Evidence for circadian SST expression in the amygdala and disrupted circadian rhythms and rhythmic peaks of anxiety in BD suggest a disruption of rhythmic expression of SST in this disorder.

**Methods:** Amygdala sections from 12 SZ, 15 BD, and 15 control subjects were processed for immunocytochemistry for SST and neuropeptide Y (NPY), a neuropeptide partially co-expressed in SST-IR neurons. Total numbers ($N_t$) of IR neurons were measured. Time of death (TOD) was used to test associations with circadian rhythms.

**Results:** SST-IR neurons were decreased in the lateral amygdala nucleus in BD ($N_t$, $p=0.003$) and SZ ($N_t$, $p=0.02$). In normal controls, $N_t$ of SST-IR neurons varied according to TOD. This pattern was altered in BD, characterized by decreases of SST-IR neurons selectively in subjects with TOD corresponding to the day (06:00-17:59). Numbers of NPY-IR neurons were not affected.

**Conclusions:** Decreased SST-IR neurons in the amygdala of SZ and BD, interpreted here as decreased SST expression, may disrupt responses to fear and anxiety regulation in these subjects. In BD, our findings raise the possibility that morning peaks of anxiety depend on a disruption of circadian regulation of SST expression in the amygdala.

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