LINDSEY MORAIS and MORGAN BRIDI, Department of Neuroscience, West Virginia University, WV, 26505. Stress-evoked activation of corticotropin-releasing factor neurons in primary sensory cortex.

Stress profoundly alters neural circuit function, yet the activity of stress-responsive neuropeptide systems within primary sensory cortices remains poorly understood. Corticotropin-releasing factor (CRF) is a central regulator of the stress response and is expressed in cortical neurons, but its functional recruitment in sensory cortical circuits during acute stress has not been well characterized. The objective of this study was to determine how acute stress modulates activity in CRF-expressing neurons compared with excitatory neurons in the primary sensory cortex. To address this, fiber photometry was used to measure calcium activity *in vivo* in CRF+ neurons and CaMKII+ excitatory neurons within the primary auditory (A1) and visual (V1) cortices. CRF-ires-cre and CaMKII-cre mice were injected with a Cre-dependent jGCaMP8m viral construct and implanted with optical fibers targeting A1 or V1. Neural activity was recorded during acute stress paradigms including tail restraint, tube restraint, and forced swim. Photometry signals were processed using ΔF/F normalization and event-aligned analyses. Acute stress produced robust increases in calcium activity in both neuronal populations at stress onset. CaMKII+ excitatory neurons exhibited a rapid transient increase in fluorescence that quickly returned toward baseline. In contrast, CRF+ neurons displayed a rapid and sustained increase in activity that persisted throughout the stress exposure. These results indicate that CRF-expressing neurons in the primary sensory cortex show sustained activation during acute stress, suggesting a potential role for CRF signaling in modulation sensory cortical processing under stress conditions.