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Ketamine anesthesia induces rampant excitation and broadband power increase in granular input layers of the rodent primary auditory cortex

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Research on sensory encoding has built its foundation on animal models under anesthesia and must now transfer its gained insight into awake experimentation. Many of the studies seeking to bridge the gap between these two contrasting states have done so at a single- or multi-unit level rather than in at a population or current flow level. In this study, local field potentials and current source density (CSD) analysis were used to provide a mesoscopic measure of cortical microcircuitry in consideration of spatial and temporal synaptic activity. CSD profiles of primary auditory cortex (ACx) of ketamine-anesthetized Mongolian gerbils (Meriones unguiculatus) were compared before and after cortical silencing with muscimol and further compared with CSD data from awake recordings in passively listening animals. Cortical silencing with muscimol showed significant reduction of tone-evoked activity across all cortical layers. Residual sink activity was only found in lemniscal thalamocortical input layers IV and Vb/VIa. In comparison to awake recordings, our main finding in the anesthetized ACx was a gain enhancement in early granular sink activity in layers III/IV. This indicates a differential recruitment of the recurrent microcircuitry in granular input layers under ketamine anesthesia and found a sharper columnar frequency tuning under anesthesia mainly due to granular gain enhancement after best-frequency stimulation. Also, we observed that in awake ACx the columnar activity is less stimulus-onset-locked, as compared to the anesthetized state. Due to this, we performed continuous wavelet analysis and discovered that the broadband stimulus time-locking occurred significantly under ketamine anesthesia only in granular input layers. We suppose that distinct differences between layer-dependent tuning properties and broadband power increase are due to a differential recruitment of the interacting excitatory and inhibitory microcircuitry between the two global brain states.