Neuromodulators, such as acetylcholine (ACh), control information processing in neural microcircuits by regulating neuronal and synaptic physiology. Computational models and simulations enable predictions on the potential role of ACh in reconfiguring network activity. As a prelude into investigating how the cellular and synaptic effects of ACh collectively influence emergent network dynamics, we developed a data-driven framework incorporating phenomenological models of the physiology of cholinergic modulation of neocortical cells and synapses. The first-draft models were integrated into a biologically detailed tissue model of neocortical microcircuitry to investigate the effects of levels of ACh on diverse neuron types and synapses, and consequently on emergent network activity. Preliminary simulations from the framework, which was not tuned to reproduce any specific ACh-induced network effects, not only corroborate the long-standing notion that ACh desynchronizes spontaneous network activity, but also predict that a dose-dependent activation of ACh gives rise to a spectrum of neocortical network activity. We show that low levels of ACh, such as during non-rapid eye movement (nREM) sleep, drive microcircuit activity into slow oscillations and network synchrony, whereas high ACh concentrations, such as during wakefulness and REM sleep, govern fast oscillations and network asynchrony. In addition, spontaneous network activity modulated by ACh levels shape spike-time cross-correlations across distinct neuronal populations in strikingly different ways. These effects are likely due to the regulation of neurons and synapses caused by increasing levels of ACh, which enhances cellular excitability and decreases the efficacy of local synaptic transmission. We conclude by discussing future directions to refine the biological accuracy of the framework, which will extend its utility and foster the development of hypotheses to investigate the role of neuromodulators in neural information processing.