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Astrocytes are critical regulators of neural and neurovascular network communication. Potassium transport is a central mechanism behind their many functions. Astrocytes encircle synapses with their distal processes, which express two potassium pumps (Na-K and NKCC) and an inward rectifying potassium channel (Kir), while the vessel-adjacent endfeet express Kir and BK potassium channels. We provide a model of the entire neurovascular unit (synaptic region, astrocytes, and arteriole) that simulates potassium movement between the synaptic space, astrocyte intracellular space, and perivascular space. Our model reproduces several phenomena observed experimentally: (1) functional hyperemia, in which neural activity triggers astrocytic potassium release at the perivascular endfoot, inducing arteriole dilation; (2) K+ undershoot in the synaptic space following periods of neural activity, and (3) neural-induced astrocyte hyperpolarization during Kir blockade. Our results suggest that the dynamics of functional hyperemia are governed by astrocytic Kir for the fast response and BK for the long term. The model supports the hypothesis that K+ undershoot is caused by excessive astrocytic uptake through Na-K and NKCC pumps, while the effect is balanced by Kir. We address parametric uncertainty in the model using high dimensional stochastic sensitivity analysis, and reveal the possible limitations of the model.