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Memory affects alternans under voltage- and calcium- mediated conditions in a fractional-order cardiomyocyte model

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The electrical behavior of cardiomyocytes is typically approximated with ideal resistor-capacitor circuit networks. However, non-ideal circuit components may more accurately model excitable cell properties. These non-ideal circuit components are governed by fractional-order dynamics and impart capacitive memory effects that contribute a history dependence to the transmembrane potential. Our prior work revealed that in a minimal model driven solely by voltage instabilities, membrane capacitive memory acts to shorten the action potential duration (APD) and suppresses alternans, a beat to beat alternation in the APD. We investigate here the effect of memory in a model driven by both intracellular calcium cycling as well as transmembrane voltage dynamics. Two parameterizations were studied corresponding to concordant calcium driven and voltage driven alternans mechanisms. Simulations for fractional orders ranging from 1 to 0.85 were performed with cycle lengths (CL) between 200 and 600 milliseconds. Consistent with our recent work, APD was shortened at smaller fractional orders. Capacitive memory also decreased peak intracellular calcium concentrations per beat. Membrane capacitive memory suppressed alternans in the calcium driven at decreasing fractional orders. However, intermediate fractional orders between 0.9 and 0.95 promoted alternans in the voltage driven parameterization. Our results suggest that membrane capacitive memory plays a role in both suppression and formation of alternans under both voltage and calcium driven mechanisms.