











Summary of The simulation of intense ISCHEMIA

The strength of ischemia was kept the same for all simulations.

- Slide 1: This slide presents the control simulations. In this scenario, there is a significant influx of Na+ ions, a rise in extracellular K+ (K+o), strong acidification, and depolarization. We observe three phases in the NBCe1 flux. The first phase is influenced by extracellular pH (pHo), the second phase is affected by K+o, and the third phase is primarily driven by the intense Na+ load and depolarization.
- Slide 2: In this slide, we apply the same level of inhibition to the pump while keeping K+o at its resting state. We observe that NBCe1 operates in an inward mode, leading to a strong influx of Na+ ions, a decrease in depolarization compared to the control, and significant acidification.
- Slide 3: Here, pHo was maintained at its resting level, and neither Na+ nor K+o changed compared to the control. However, the first phase observed in NBCe1 flux during the control case disappears in this scenario. The cell becomes more alkaline compared to the control case.
- Slide 4: In this slide, the membrane potential of astrocytes was kept at rest. NBCe1 operates in an inward mode, resulting in a decrease in extracellular K+ (Ko) compared to the control, an increase in Na+ load, and increased acidification.
- Slide 5: With resting intracellular K+ (Ki), Ko decreases compared to the control, Na+ load increases compared to the control, depolarization decreases, and NBCe1 operates in reverse mode, leading to depolarization.
- Slide 6: At rest, intracellular Na+ (Nai), there is a significant rise in Ko, increased depolarization, alkalization in the beginning, and a distinct reversal and depolarization.
- Conclusion: In summary, during intense ischemia, the mode of operation of NBCe1 depends on extracellular K+ concentrations. Low Ko leads to NBCe1 operating in an inward mode, while high Ko results in NBCe1 operating in reverse mode, along with strong depolarizations.