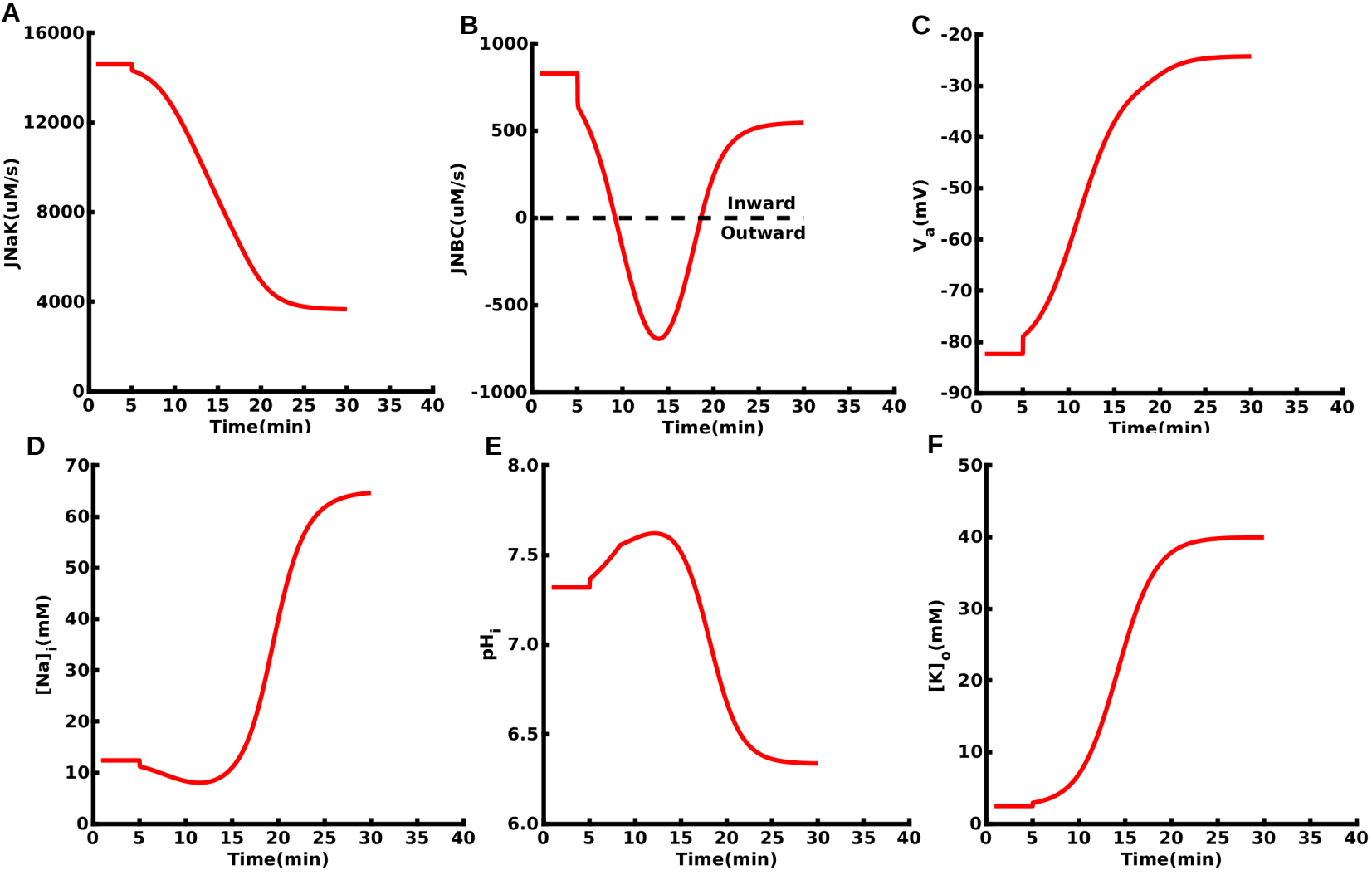
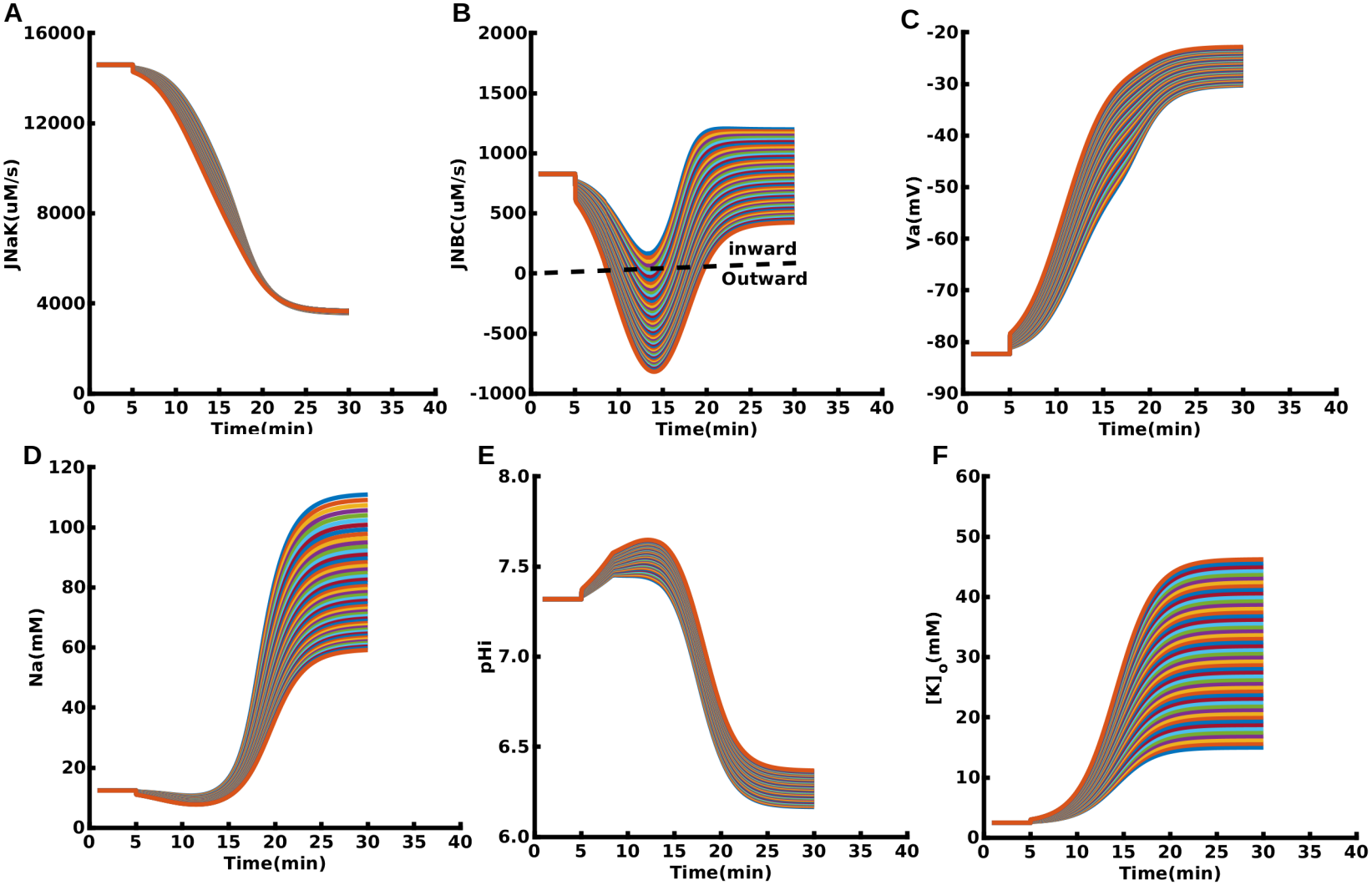
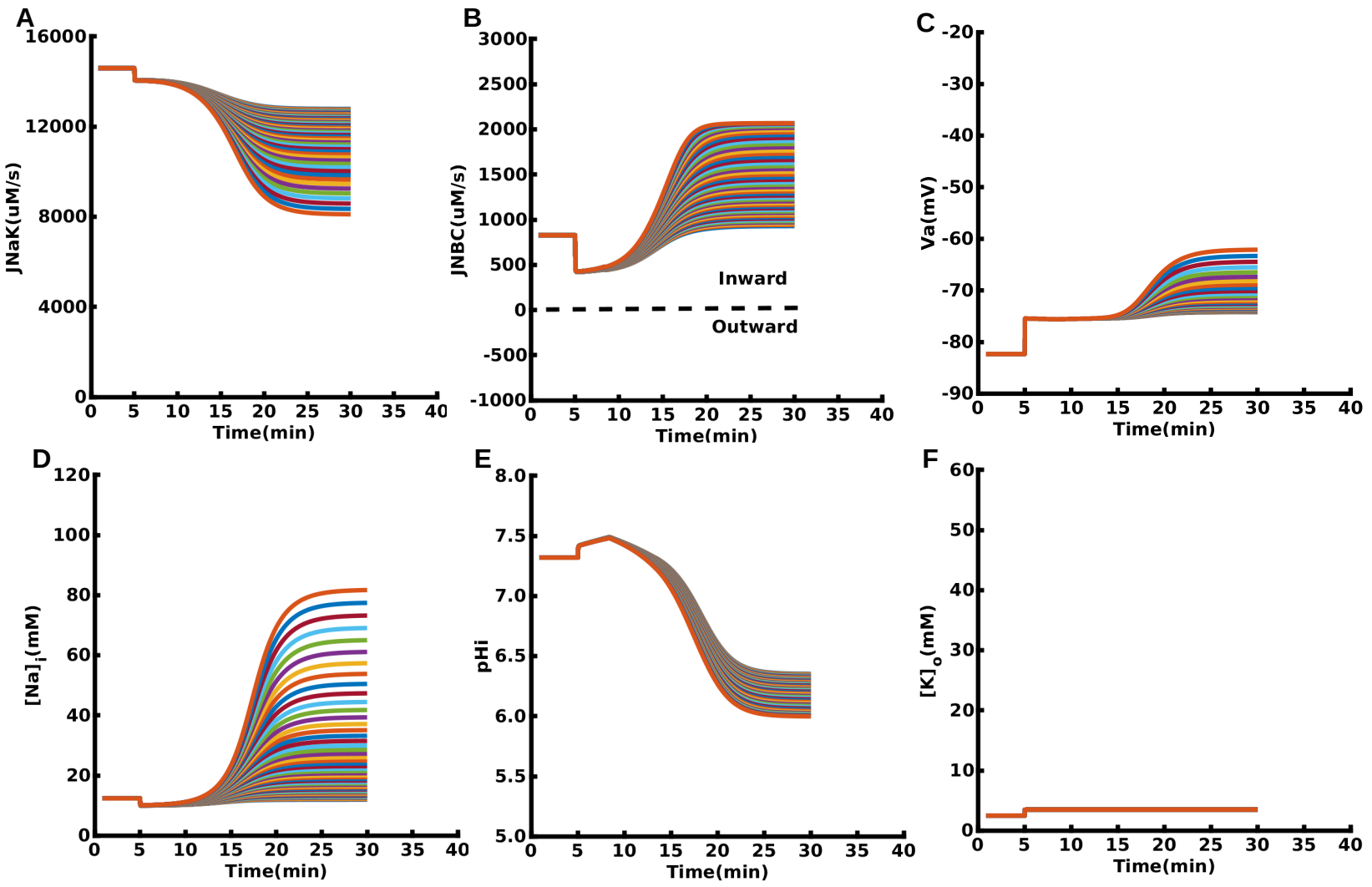
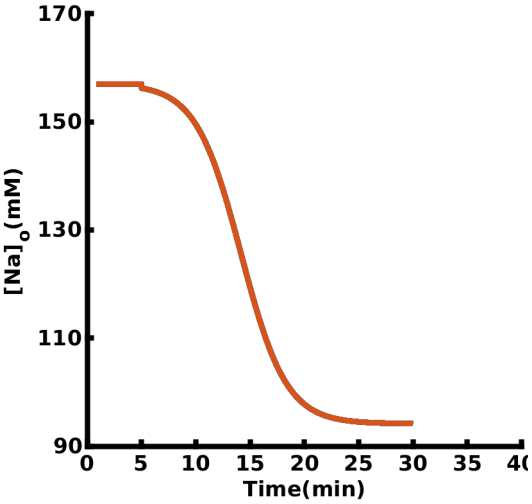
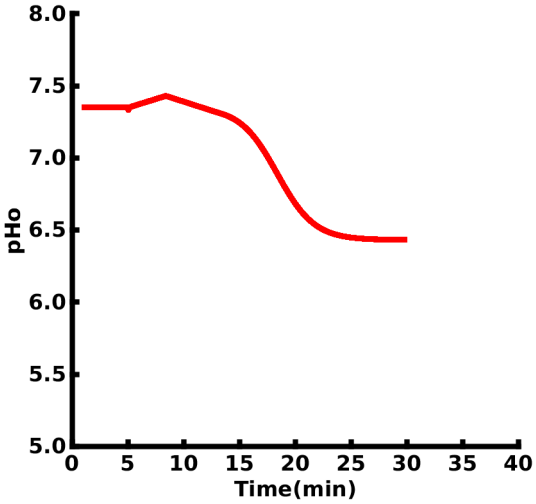

A
JNaK(uM/s)
16000
12000
8000
4000
0
0 5 10 15 20 25 30 35 40
Time(min)
B
JNBC(uM/s)
1000
500
0
-500
-1000
Inward
Outward
0 5 10 15 20 25 30 35 40
Time(min)
C
V_a(mV)
-20
-30
-40
-50
-60
-70
-80
-90
0 5 10 15 20 25 30 35 40
Time(min)
D
$[Na]_i$(mM)
70
60
50
40
30
20
10
0
0 5 10 15 20 25 30 35 40
Time(min)
E
pH_i
8.0
7.5
7.0
6.5
6.0
0 5 10 15 20 25 30 35 40
Time(min)
F
$[K]_o$(mM)
50
40
30
20
10
0
0 5 10 15 20 25 30 35 40
Time(min)


A
JNaK(uM/s)
16000
12000
8000
4000
0
0 5 10 15 20 25 30 35 40
Time(min)
B
JNBC(uM/s)
2000
1500
1000
500
0
-500
-1000
inward
Outward
0 5 10 15 20 25 30 35 40
Time(min)
C
Va(mV)
-20
-30
-40
-50
-60
-70
-80
-90
0 5 10 15 20 25 30 35 40
Time(min)
D
Na(mM)
120
100
80
60
40
20
0
0 5 10 15 20 25 30 35 40
Time(min)
E
pHi
8.0
7.5
7.0
6.5
6.0
0 5 10 15 20 25 30 35 40
Time(min)
F
[K]o(mM)
60
50
40
30
20
10
0
0 5 10 15 20 25 30 35 40
Time(min)


A
JNaK(uM/s)
16000
12000
8000
4000
0
0 5 10 15 20 25 30 35 40
Time(min)
B
JNBC(uM/s)
3000
2500
2000
1500
1000
500
0
-500
-1000
Inward
Outward
0 5 10 15 20 25 30 35 40
Time(min)
C
Va(mV)
-20
-30
-40
-50
-60
-70
-80
-90
0 5 10 15 20 25 30 35 40
Time(min)
D
[Na]i(mM)
120
100
80
60
40
20
0
0 5 10 15 20 25 30 35 40
Time(min)
E
pHi
8.0
7.5
7.0
6.5
6.0
5.5
5.0
0 5 10 15 20 25 30 35 40
Time(min)
F
[K]o(mM)
60
50
40
30
20
10
0
0 5 10 15 20 25 30 35 40
Time(min)


170
150
130
110
90
[Na]o(mM)
0
5
10
15
20
25
30
35
40
Time(min)


8.0
7.5
7.0
6.5
6.0
5.5
5.0
pHo
0
5
10
15
20
25
30
35
40
Time(min)

Summary Of The Slides For Effects of NBCe1 in Severe Ischemia

Different color represent different simulation.

Slide 1:
To study severe ischemia, the sodium-potassium pump was stressed for 20 minutes. In Slide 1, we observed an increase in Na+ levels, intense acidification, reverse activity of NBCe1, and significant intracellular depolarization.

Slide 2:
In Slide 2, we manipulated potassium bath concentrations (increasing the Kobath amplitude parameter by 0.25 for each simulation) to vary extracellular potassium levels, ranging from approximately 10 mM to 46 mM. As the extracellular potassium concentration (K+) increased, we noticed a corresponding increase in the reverse behavior of NBCe1, membrane depolarization, and alkalization.

Slide 3:
In Slide 3, with Ko adjusted to 3.5 mM, NKA pump inhibition was increased in each simulation by decreasing the pump flux by 0.74% of its resting value in each simulation. It showed that NBCe1 reversibility vanished, and the membrane depolarization decreased compared to the same value of Na+ load as in Slide 1. This suggests that a greater Na+ load is required to achieve the same level of depolarization as in Slide 1.
Slide4: Extracellular Nao and pHo consider for slide 1 to 3

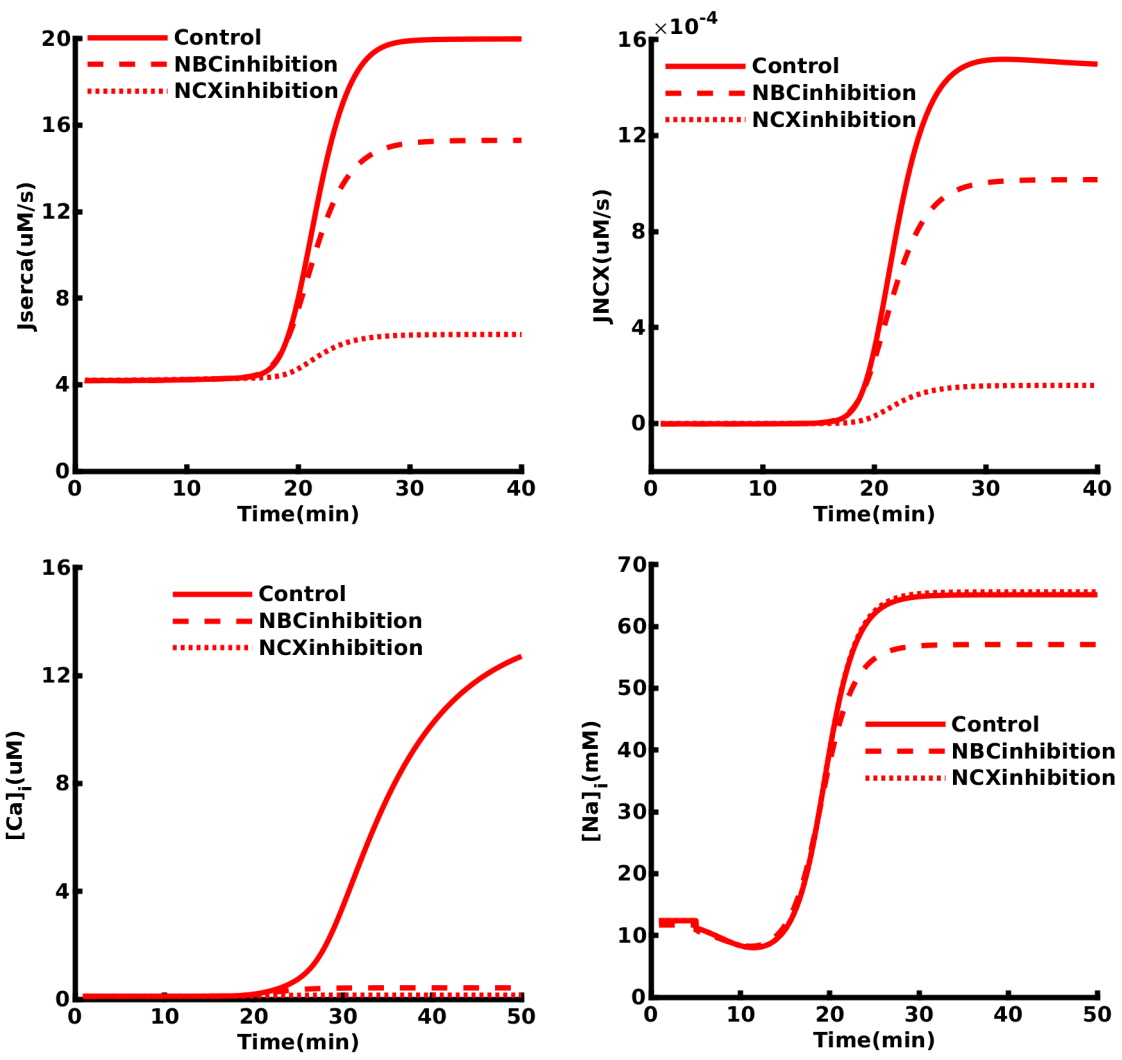


Fig. The calcium signaling, which originates after 20 minutes, reveals that the Sodium-Calcium Exchanger (NCX) operates in reverse mode when the Sodium-Bicarbonate Co-transporter (NBC) operates in inward mode. The dampening of calcium signaling observed during NBC inhibition indicates that NBC contributes to the reversal of NCX. The weak increase in sodium ions (Na+) during NCX inhibition suggests that NCX is indeed operating in reverse mode. In our model, we only consider three calcium pathways: SERCA, TRPV, and NCX. This omission is due to the absence of glutamate in our model, which precludes the consideration of glutamate receptors. We extended the simulation duration to observe calcium stability over a longer period.