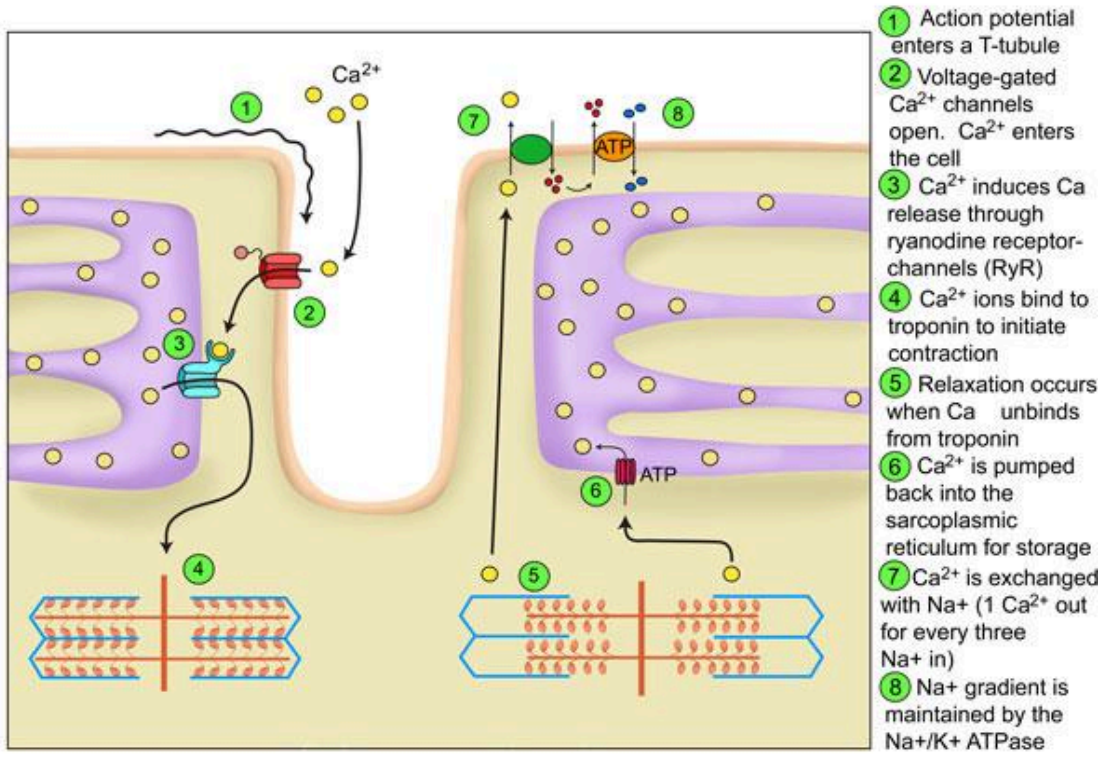
1.3.2

Cardiac Excitation Coupling



Cardiac Excitation Coupling.
BYU-Idaho image: Created by Becky T Fall 2018

Now that you know how action potentials are generated and conducted in the heart, we need to study how these action potentials lead to contraction of the cardiac muscle cells. We have already explained that cardiac muscle is very much like skeletal muscle and that the mechanism of contraction is the same in both muscle types. Therefore, the key to cardiac muscle contraction is also calcium. As with skeletal muscle, calcium is stored intracellularly in the sarcoplasmic reticulum (SR). However, unlike skeletal muscle, cardiac muscle also relies on extracellular calcium for proper functioning. This means that the extracellular level of calcium is very important for heart muscle function.

Intracellular stores of calcium in the well-developed sarcoplasmic reticulum can help skeletal muscle buffer against blood calcium level changes, but heart muscle does not have this advantage. The heart muscle sarcoplasmic reticulum is much less developed and heart muscle fibers are more sensitive to extracellular calcium level oscillations. In fact, small changes in blood calcium levels can cause heart arrhythmias.

As might be expected, there are some important ion channels that mediate the entry of calcium. The first is a voltage-gated calcium channel found in the membranes of the T-tubules (L-Type Calcium Channels). These are the same channels discussed earlier that are responsible for the plateau phase of the action potential. When the action potential descends into the T-tubules these channels open, allowing calcium to diffuse into the cell. This calcium then binds to calcium release channels found in the membranes of the SR (these channels are also known as ryanodine receptors, RyR, channels). The binding of calcium to these channels causes them to open allowing calcium to diffuse out of the SR and bind to troponin initiating contraction. Thus, extracellular calcium triggers the release of sarcoplasmic reticular calcium. This process is referred to as calcium-induced, calcium release. Once the signal ends, a calcium ATPase in the membranes of the SR pumps the calcium back into the SR and contraction ends. Since some of the calcium that is now in the cell came from the extracellular fluid, a Na^{+}-Ca^{2+} exchanger (secondary active transport – 3 Na^{+} to 1 Ca^{++}) moves calcium from the cytoplasm back to the extracellular fluid. As will be discussed later, the strength of the muscular contraction in the heart is dependent on the amount of calcium that enters, hence, there are mechanisms to regulate how much calcium enters the cells.