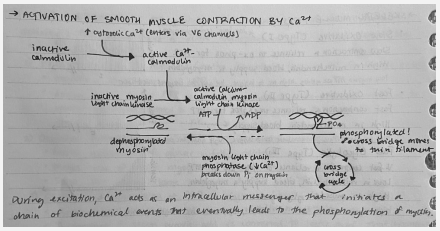
Smooth Muscle Features

Location: Walls of hollow organs and tubes, structures that change in volume. (ex-intestines).

Non-striated.
Uninuclear.
Spindle-shaped cells (dia = 2-10 µm, l = 50-400 µm).
Divide lifelong.
Thick filaments = myosin.
Thin filaments = actin, anchored to the plasma membrane or dense bodies.
Filaments are not organized into myofibrils, no sarcomeres.
No troponin, calmodulin instead!
Tropomyosin present.
Contract by sliding-filament mechanism.
No T-tubules.
Sarcoplasmic reticulum present.

Activation of Smooth Muscle Contraction by Ca+2



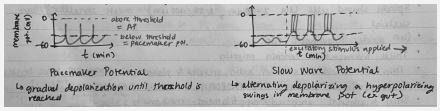
Single Unit Smooth Muscle

Cells respond to stimuli as a single unit due to connections via gap junctions = functional syncytium.
Capable of generating pacemaker activity.
Spontaneous AP from pacemaker SMC can propogate to non-pacemaker SMC due to gap junctions.
Experience pacemaker potential and slow-wave potential.
Ex- GI tract walls, reproductive tract walls, urinary tract walls, walls of small blood vessels.

Multi Unit Smooth Muscle

SMCs that are activated by neuronal input.
Cells respond to stimuli independently and contain few gap junctions.
Ex- Walls of large blood vessels, large airways to lungs, muscles of the eye that adjust the lens, iris of the eye, base of hair follicles.

Pacemaker vs. Slow-Wave Potential



Excitation/Contraction Coupling in SMCs

Self or neuron excitation leads to Ca2+ entry from the extracellular space via VG channels.

Ca2+ entry triggers internal release of Ca2+ from SR.
Ca2+ binds to calmodulin in cytosol.
Ca2+-calmodulin complex activates light chain myosin kinase (phosphorylates light chain of myosin).
Phosphorylated myosin light chain binds to actin = activated cross-bridges.
Removal of Ca2+ desphosphorylates myosin, dissociating it from actin.
Gap junctions allow rapid spread of excitation between connected cells.
Contraction strength is directly proportional to cytosolic [Ca2+].