
بسم الله الرحمن الرحيم

Small Group Discussion (SGD)
1st year MBBS, Batch 50 (2023)
Physiology of Smooth Muscle Contraction

Dr. Muhammad Usman

Date:-15th may2023

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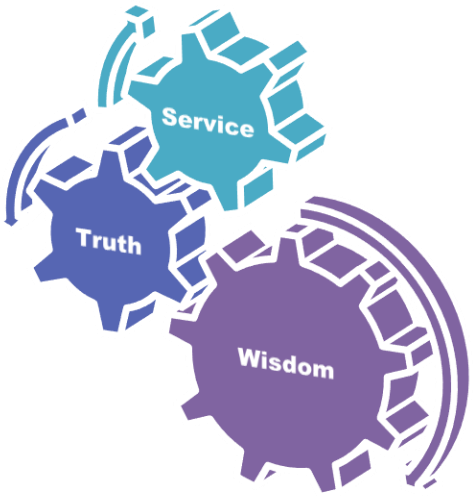
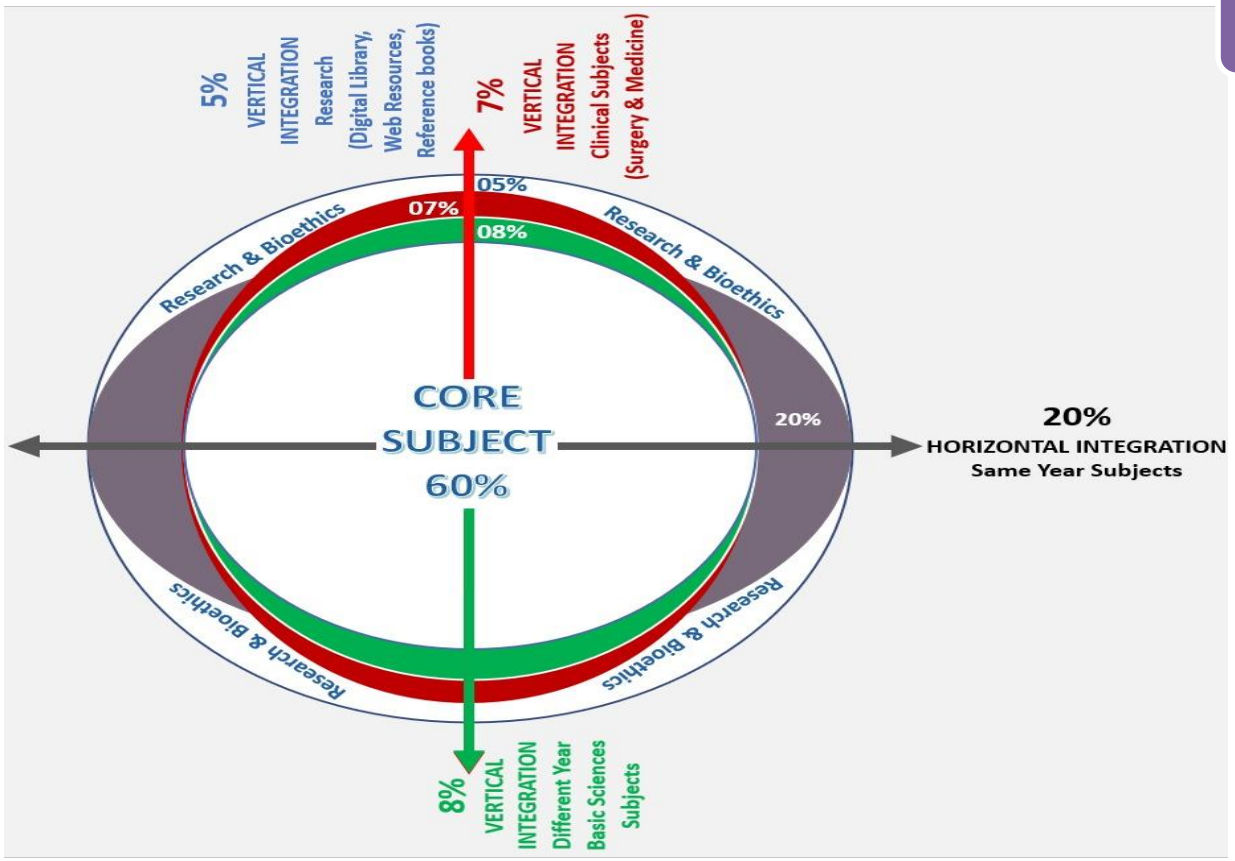
Motto

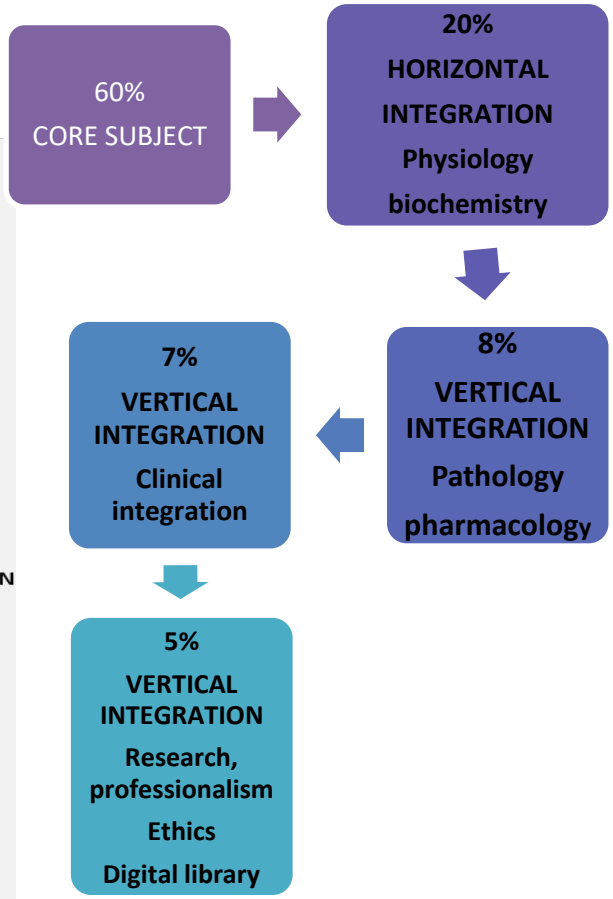


Vision; The Dream/Tomorrow

- To impart evidence based research oriented medical education
- To provide best possible patient care
- To inculcate the values of mutual respect and ethical practice of medicine

Professor Umar Model of Integrated Lecture

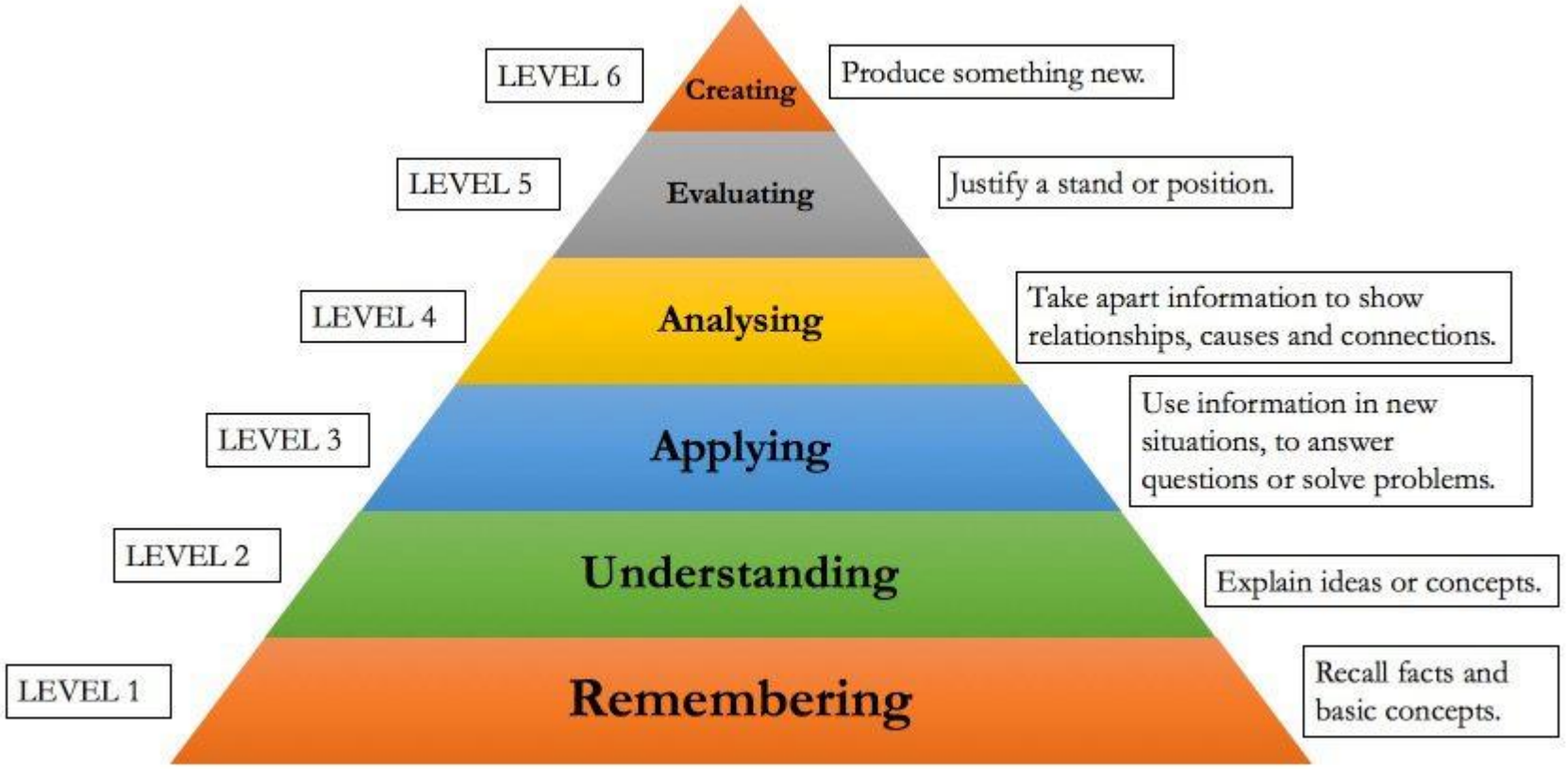




BLOOM'S TAXONOMY : DOMAINS OF LEARNING

Sr. #	Domain of learning	Abbreviation	Levels of the domain	Meaning
1	cognition	C	C1	Recall / Remembering
2			C2	Understanding
3			C3	Applying / Problem solving
4	Psychomotor	P	P1	Imitation / copying
5			P2	Manipulation / Follows instructions
6			P3	Precision / Can perform accurately
7	Attitude	A	A1	Receiving / Learning
8			A2	Respond / Starts responding to the learned attitude
9			A3	Valuing / starts behaving according to the learned attitude

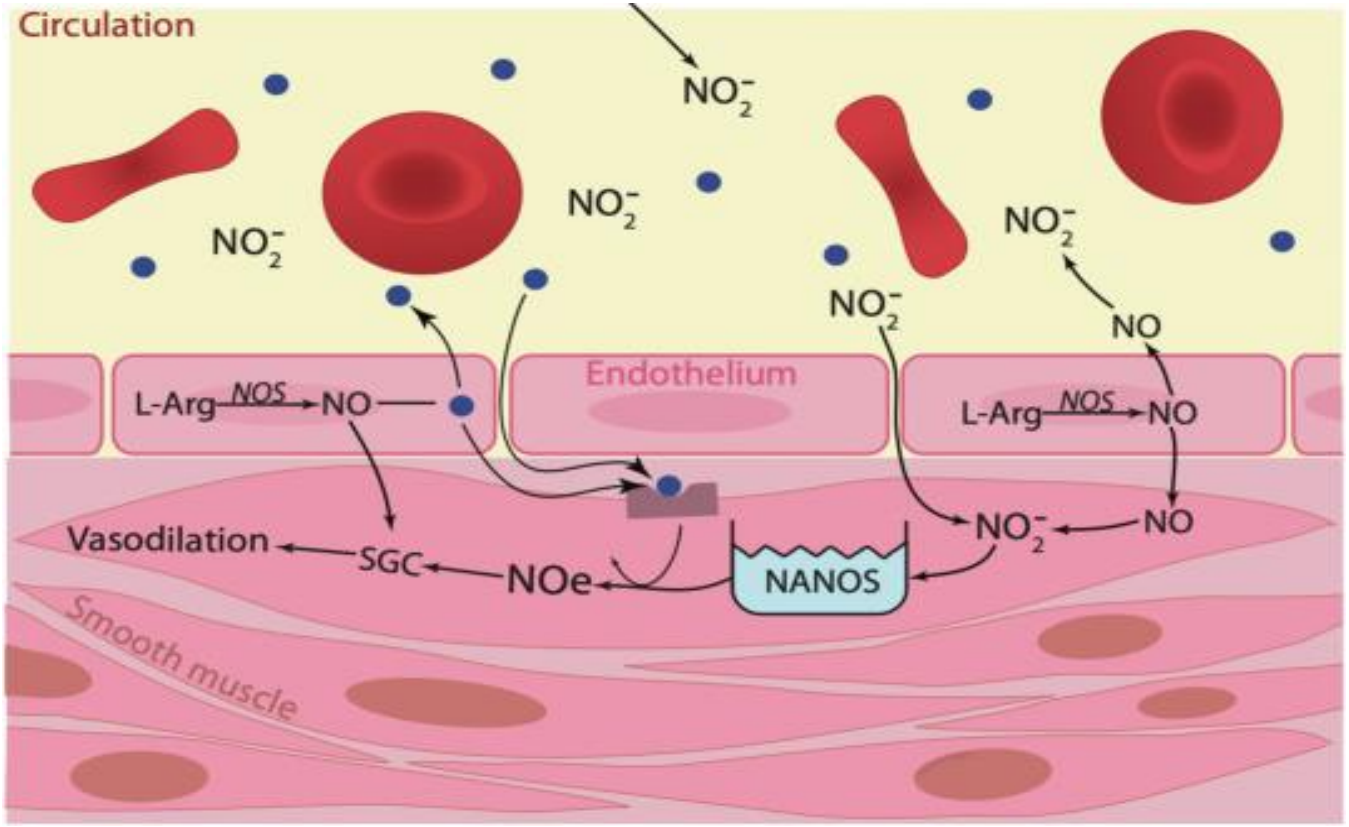
BLOOM'S TAXONOMY OF THE COGNITIVE DOMAIN



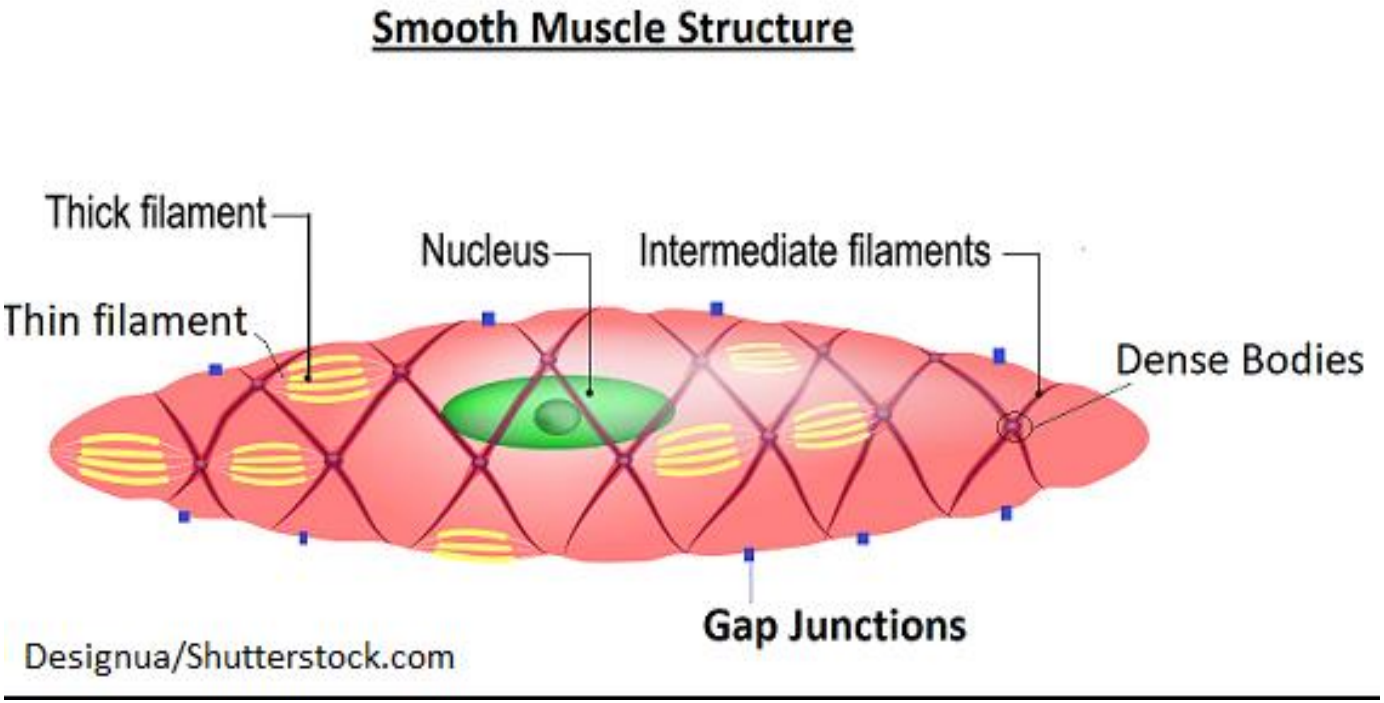
LEARNING OBJECTIVES

Sr. #	Learning Objective	Domain of Learning
1	To understand the structure, function, and regulation of smooth muscle.	C2
2	To Discuss the role of smooth muscle in various physiological processes.	C2
3	Explain the molecular and cellular mechanisms of smooth muscle contraction.	C2
4	Understand the role of calcium signaling, cross-bridge cycling, and regulatory factors in smooth muscle contraction.	C2
5	Identify the factors influencing smooth muscle tone and relaxation.	C4

Horizontal integration
(With Biochemistry)



Horizontal integration
(With Anatomy)



Core concept

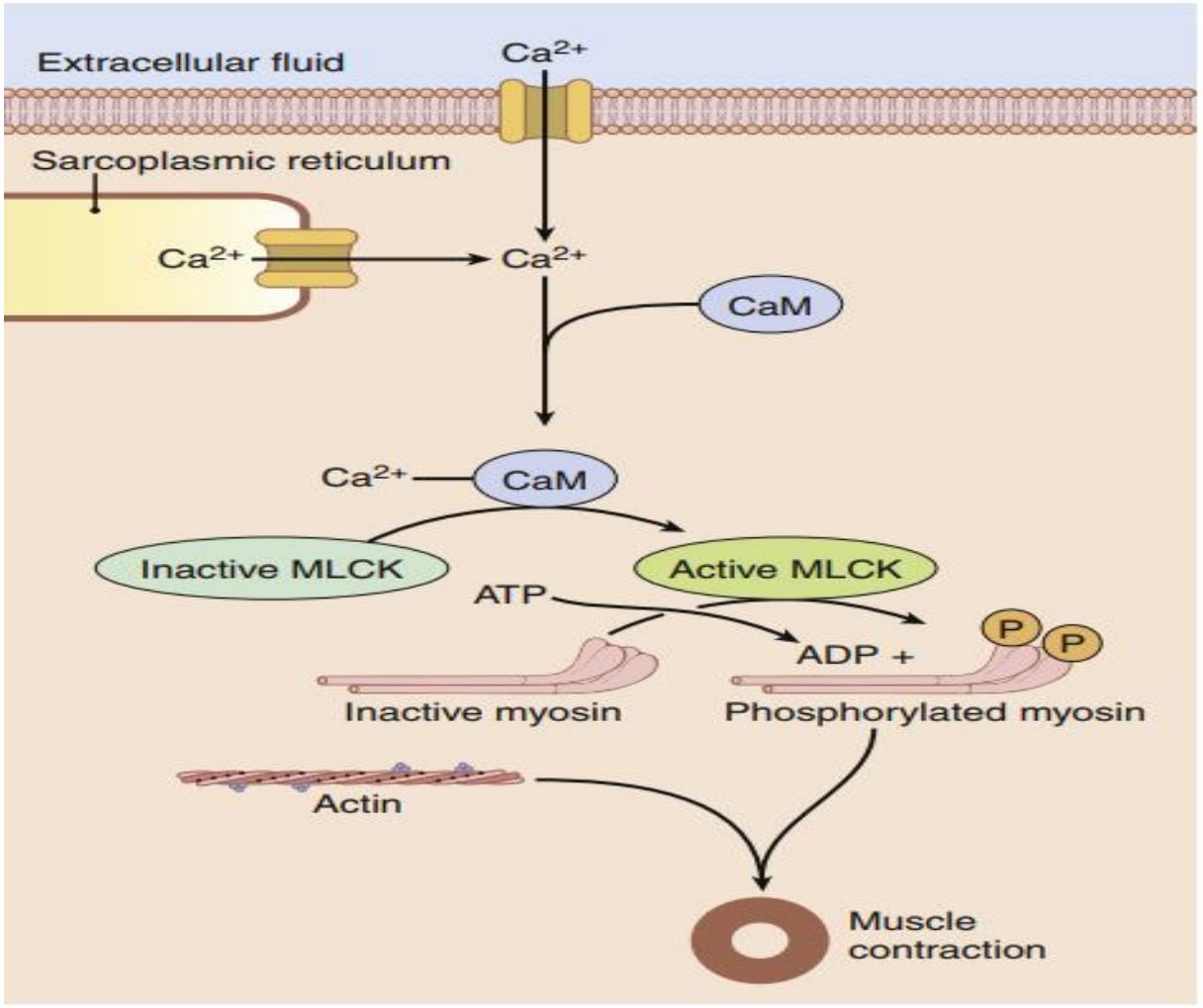
Physiology of Smooth Muscle

- Smooth muscle is considered to be much more primitive than either cardiac or skeletal muscle.
- Muscle striations are not visible in smooth muscle, so the sarcomere relationship of myosin to actin does not exists in smooth muscle.
- However, per cross sectional area smooth muscle is as strong as skeletal muscle and smooth muscle is highly resistant to fatigue.

Smooth Muscle Contractile Mechanism

- Contractive Process in Smooth Muscle
  - 1. Chemical basis for smooth muscle contraction
    - a. Contains actin and myosin filaments similar in structure and interaction to skeletal muscle
    - b. No troponin complex - mechanism for contraction is different
    - c. Calcium influx activates the contractile process
    - d. ATP provides energy for contraction

Smooth Muscle Contractile Mechanism



Smooth Muscle Contractile Mechanism

-2. Physical basis for smooth muscle contraction

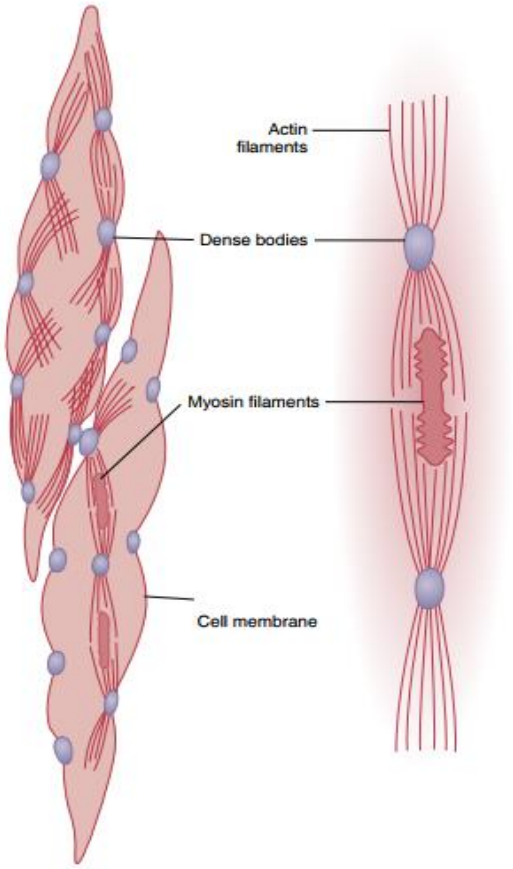
- A. Smooth muscle is not striated
- b. Dense bodies - attachment for actin fibers - some attach to cell membrane others located throughout the cytoplasm - dense bodies in one cell may join with a dense body in the adjacent cell
- c. Few myosin fibers are located in the actin fibers (15:1 ratio of actin to myosin)

Smooth Muscle Contractile Mechanism

- 3. Comparison of smooth muscle contraction with skeletal muscle contraction
  - a. Starts slower and lasts longer than striated muscle fiber, smooth muscle has a prolonged contraction - up to hours to days
  - b. Can shorten and stretch to a greater extent than striated muscle
  - c. Contraction is initiated by calcium influx into the sarcoplasm (Outside Calcium)

Smooth Muscle Contractile Mechanism

- D. Sarcoplasmic reticulum in smooth muscle is sparse - 3 to 5 % of cell volume
- e. Calcium flows into sarcoplasm from extracellular fluid
- f. No T-tubules in smooth muscle - Therefore, calcium movement is slow
- g. Smooth muscle tone - occurs due to the slow movement of calcium from the cell
- h. Smooth muscle has less ATPase activity and therefore, less degradation of ATP



Smooth Muscle Contractile Mechanism

- l. Smooth muscle only needs 1/10 to 1/300 of the energy that skeletal muscle requires
- j. Only one ATP is required per contraction cycle no matter how long it lasts
- k. Smooth muscle reaches full contraction about 1/2 second after stimulation
- l. Contractile force reaches maximum within 1 - 2 seconds after stimulation

Smooth Muscle Contractile Mechanism

- M. Rate of contraction is 30 times slower than skeletal muscle
- n. Contractions can last from 0.2 to 30 seconds
- o. Smooth muscle force of contraction can be approximately 2X that of skeletal muscle
- p. Smooth muscle can shorten to a greater degree than skeletal muscle reduces lumen of organs to almost zero

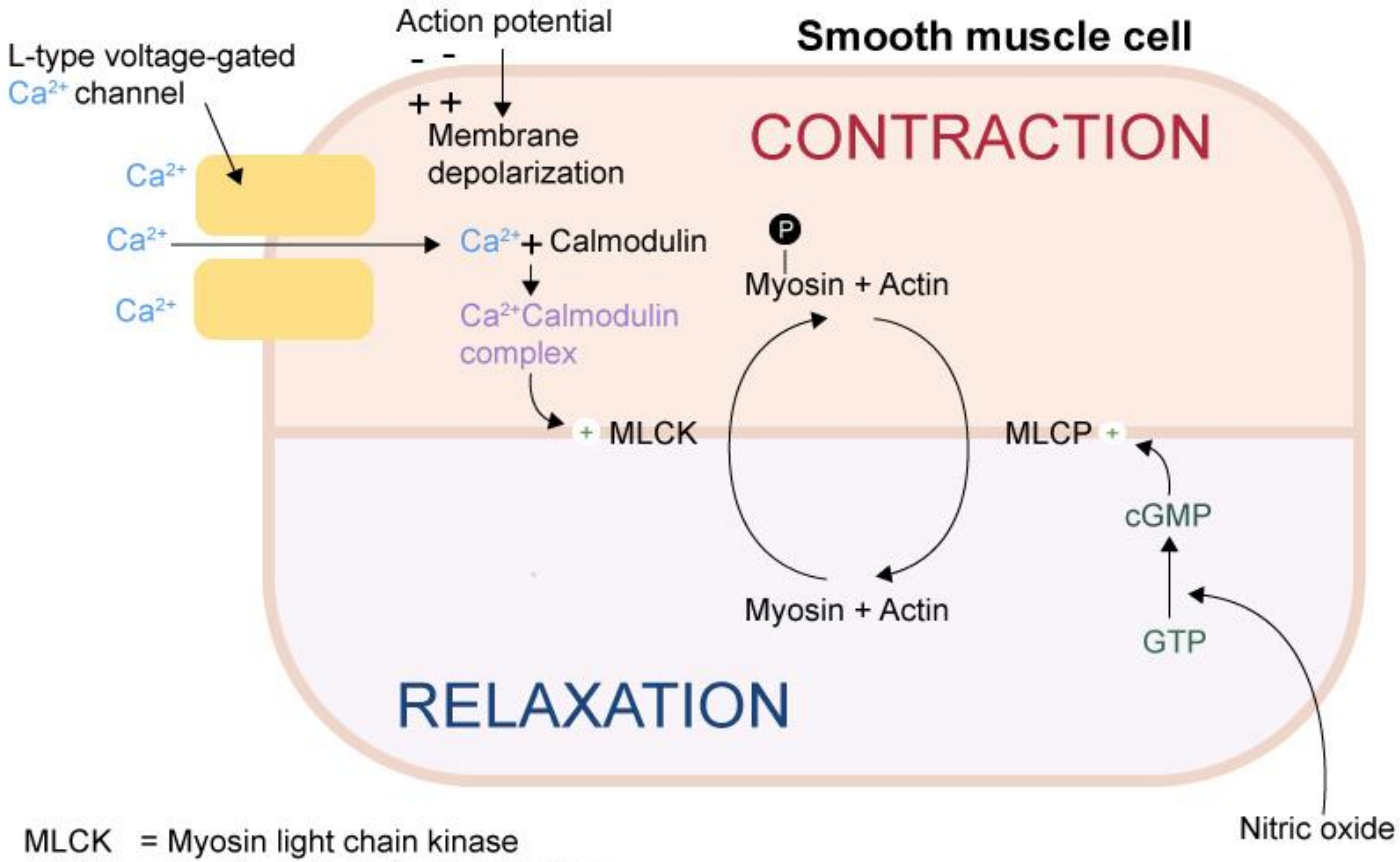
Smooth Muscle
Contractile Mechanism

- 4. “Latch Mechanism - prolonged holding in smooth muscle
  - a. After contraction is initiated, less stimulus and energy are needed to maintain the contraction (Energy conservation)
  - b. Can maintain prolonged tonic contractions for hours with little energy and little excitatory signal from nerves or hormones

Smooth Muscle Contractile Mechanism

- C. Smooth muscle can undergo great changes in length and still retain the ability to contract effectively
- d. This response allows vessels and hollow organs to change size but maintain the pressure within the structure at a constant level (Probably related to the “latch mechanism”)

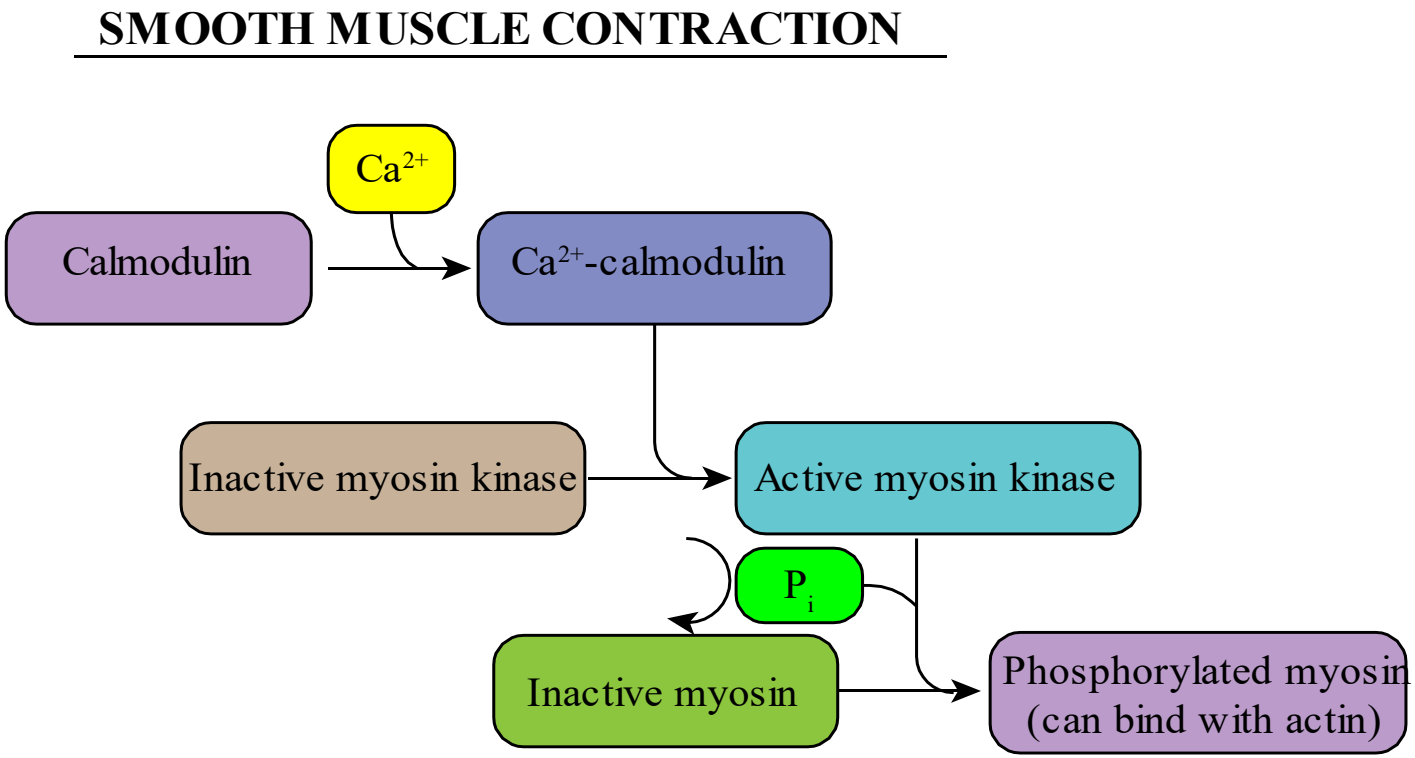
Smooth Muscle Contractile Mechanism



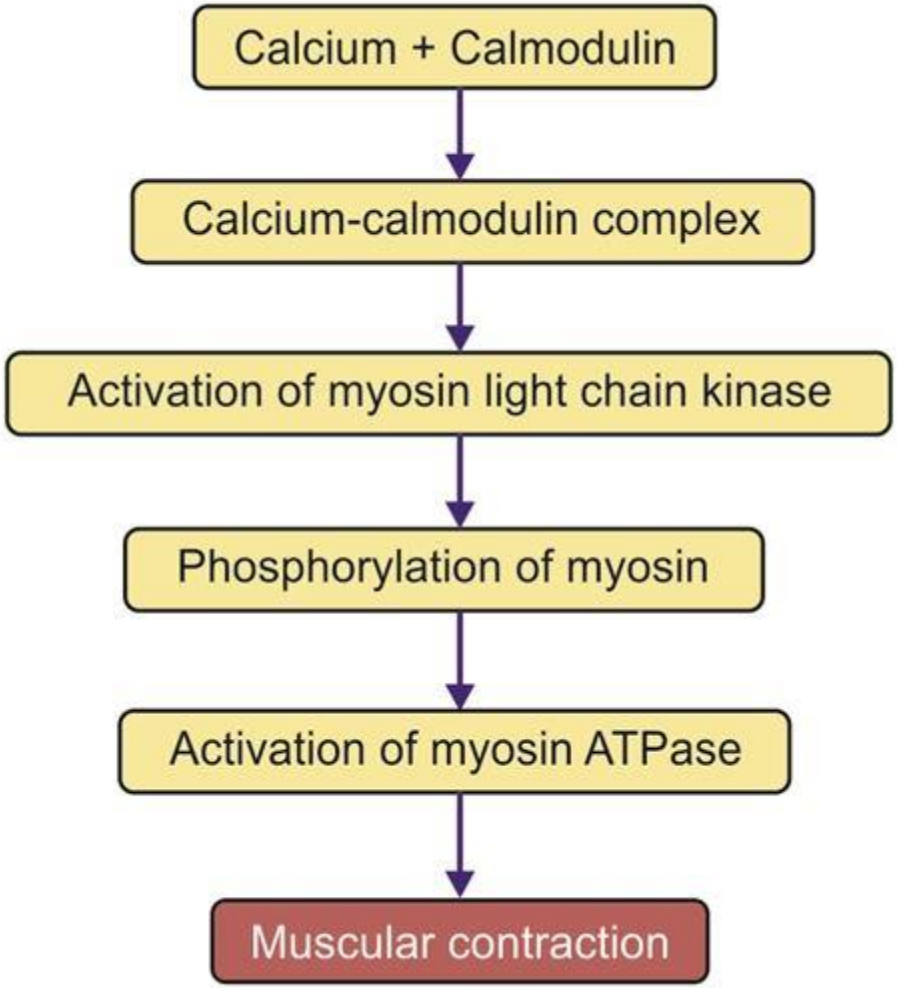
MLCK = Myosin light chain kinase
MLCP = Myosin light chain phosphatase
GTP = Guanosine triphosphate
cGMP = Cyclic guanosine monophosphate

Lucy Liu

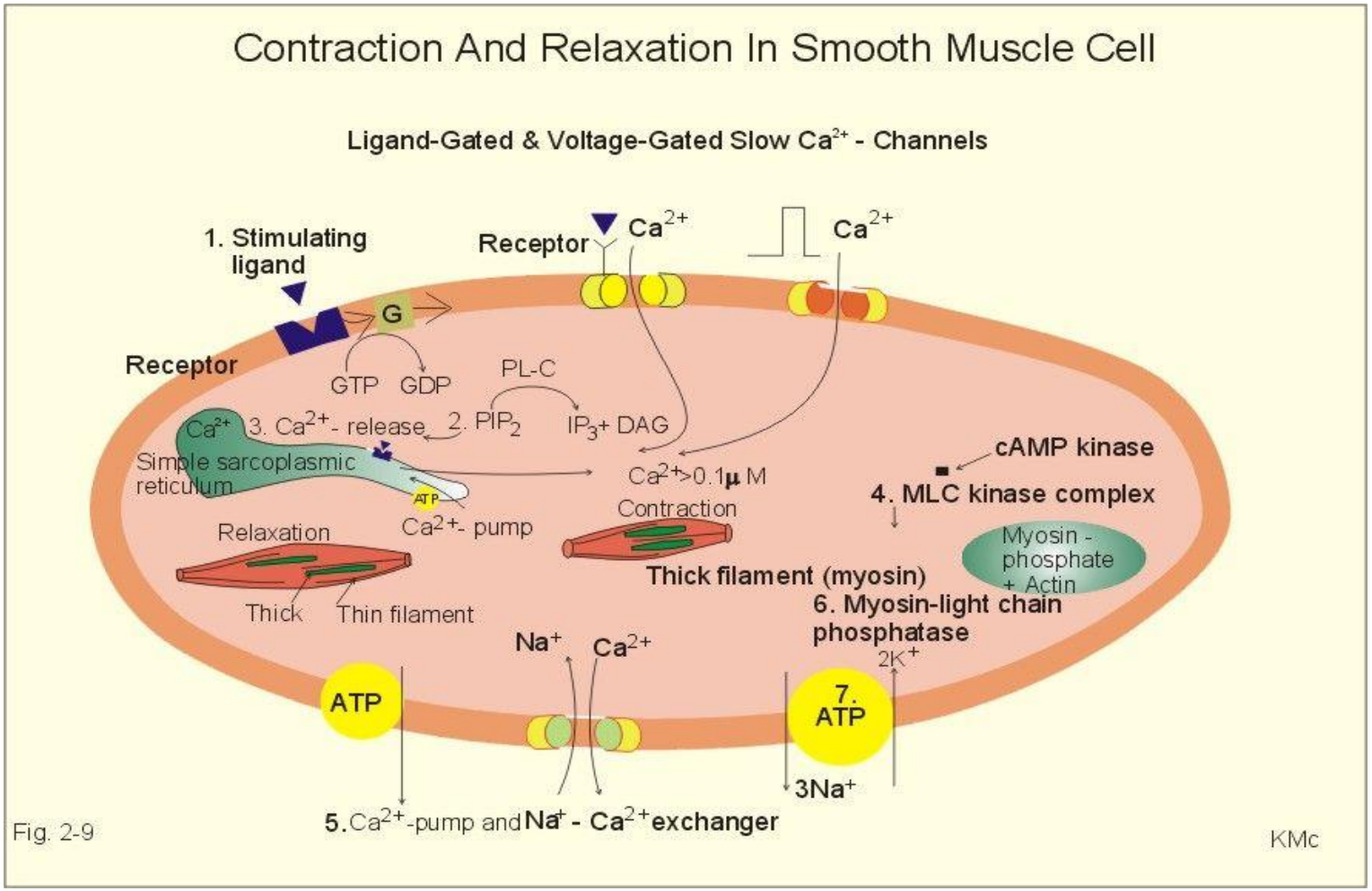
Smooth Muscle Contractile Mechanism



Smooth Muscle Contractile Mechanism



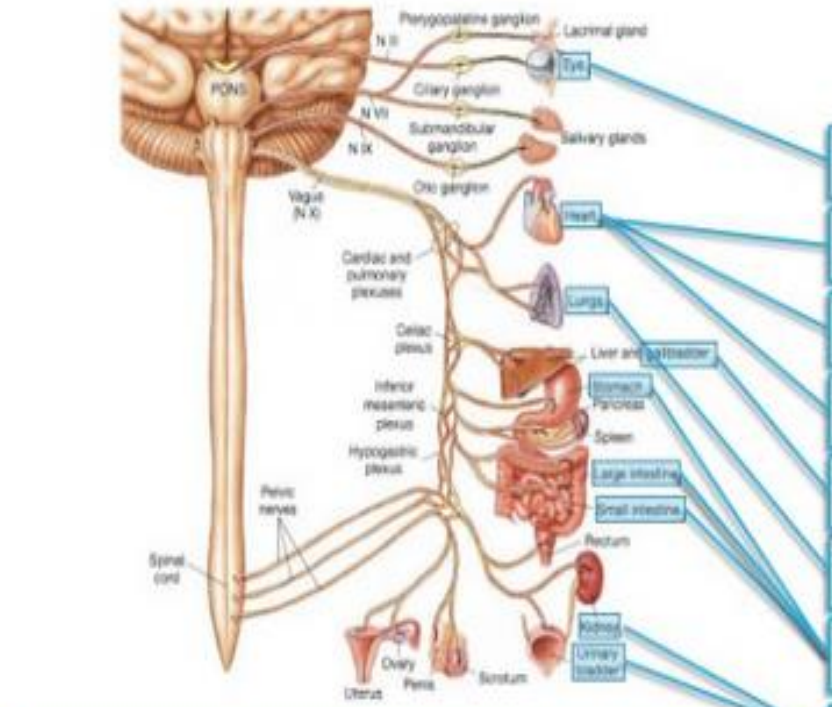
Smooth Muscle Contractile Mechanism



Vertical integration

(With Clinical and Para-clinical Sciences)

Multisystemic Smooth Muscle Disorder Syndrome



Symptoms arise from disfunction of SMC innervated by autonomic nerve fibers

- Congenital Mydriasis, Aniridia
- PDA, APW, PAH,
- Aortic Aneurysm and Dissection
- Low Blood Pressure, Dysautonomia
- Gallbladder Stones
- Lung Disease, Asthma
- Gut dysmotility, Malrotation, Prune-Belly
- Atonic bladder, Hydronephrosis

BIOMEDICAL ETHICS (Lesson of The Day)

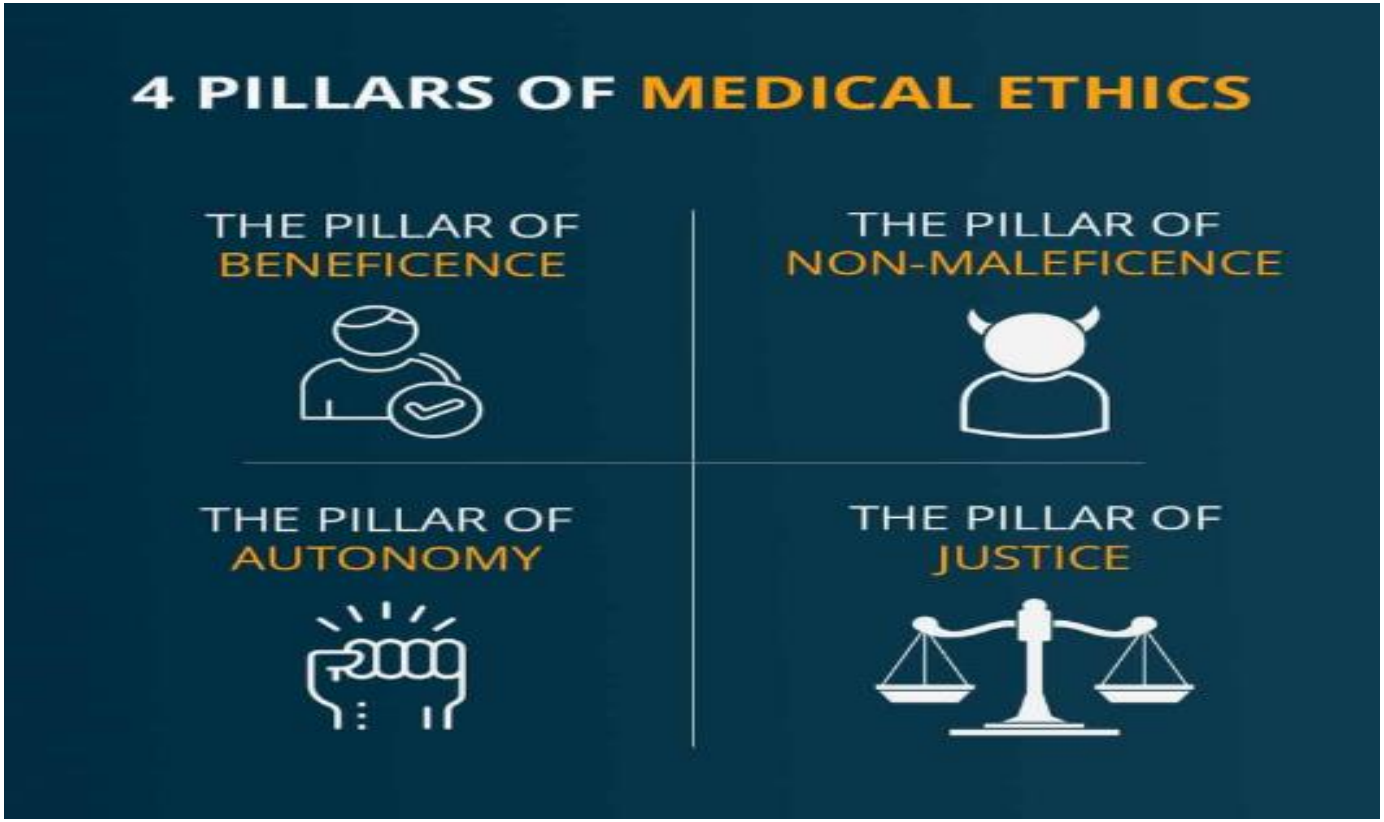
- Core biomedical ethical principles are fundamental guidelines that shape and govern ethical decision-making and behavior.

- These principles provide a framework for individuals and organizations to determine what is morally right or wrong in various contexts.

Beneficence

- The principle of beneficence is the obligation of physician to act for the **benefit of the patient** and supports a number of moral rules to protect and defend the right of others, prevent harm, remove conditions that will cause harm, help persons with disabilities, and rescue persons in danger.
- It is worth emphasizing that, the language here is one of positive requirements. The principle calls for not just avoiding harm, but also to benefit patients and to promote their welfare.

- The core pillars/principles of medical ethical include:



Brainstorming/Recall About Topic Under Consideration

MCQ 1:

In excitation-contraction of smooth muscle, calcium binds to what protein after influx into the cytoplasm?

A. Calmodulin
B. Myosin light chains
C. Troponin
D. Tropomyosin
E. Protein kinase A

Key

Correct Answer: A

Brainstorming/Recall

MCQ 1:

The role of myosin light-chain protein in smooth muscle is what?

A. Bind to calcium ions to initiate excitation-contraction coupling
B. Phosphorylate cross-bridges, thus driving them to bind with the thin filament
C. Split ATP to provide the energy for the power stroke of the cross-bridge cycle
D. Dephosphorylate myosin light-chains of the cross-bridge, thus relaxing the muscle
E. Pump calcium from the cytosol back into the sarcoplasmic reticulum

Key

Correct Answer: B

Suggested research article

Journal of Asthma and Allergy

Dovepress
open access to scientific and medical research

Open Access Full Text Article

REVIEW

Targeting Airway Smooth Muscle Hypertrophy in Asthma: An Approach Whose Time Has Come

Anne Chetty
Heber C Nielsen

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Abstract: Airway smooth muscle (ASM) cell dysfunction is an important component of several obstructive pulmonary diseases, particularly asthma. External stimuli such as allergens, dust, air pollutants, and change in environmental temperatures provoke ASM cell hypertrophy, proliferation, and migration without adequate mechanistic controls. ASM cells can switch between quiescent, migratory, and proliferative phenotypes in response to extracellular matrix proteins, growth factors, and other soluble mediators. While some aspects of airway hypertrophy and remodeling could have beneficial effects, in many cases these contribute to a clinical phenotype of difficult to control asthma. In this review, we discuss the factors responsible for ASM hypertrophy and proliferation in asthma, focusing on cytokines, growth factors, and ion transporters, and discuss existing and potential approaches that specifically target ASM hypertrophy to reduce the ASM mass and improve asthma symptoms. The goal of this review is to highlight strategies that appear ready for translational investigations to improve asthma therapy.

Keywords: airway smooth muscle cells, hypertrophy, proliferation, airway remodeling

https://doi.org/10.2147/JAA.S280247

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Thank You!