PATHOPHYSIOLOGY OF ASTHMA

FLAME LECTURE: 24

MISTRY 6.25.19

RISK FACTORS

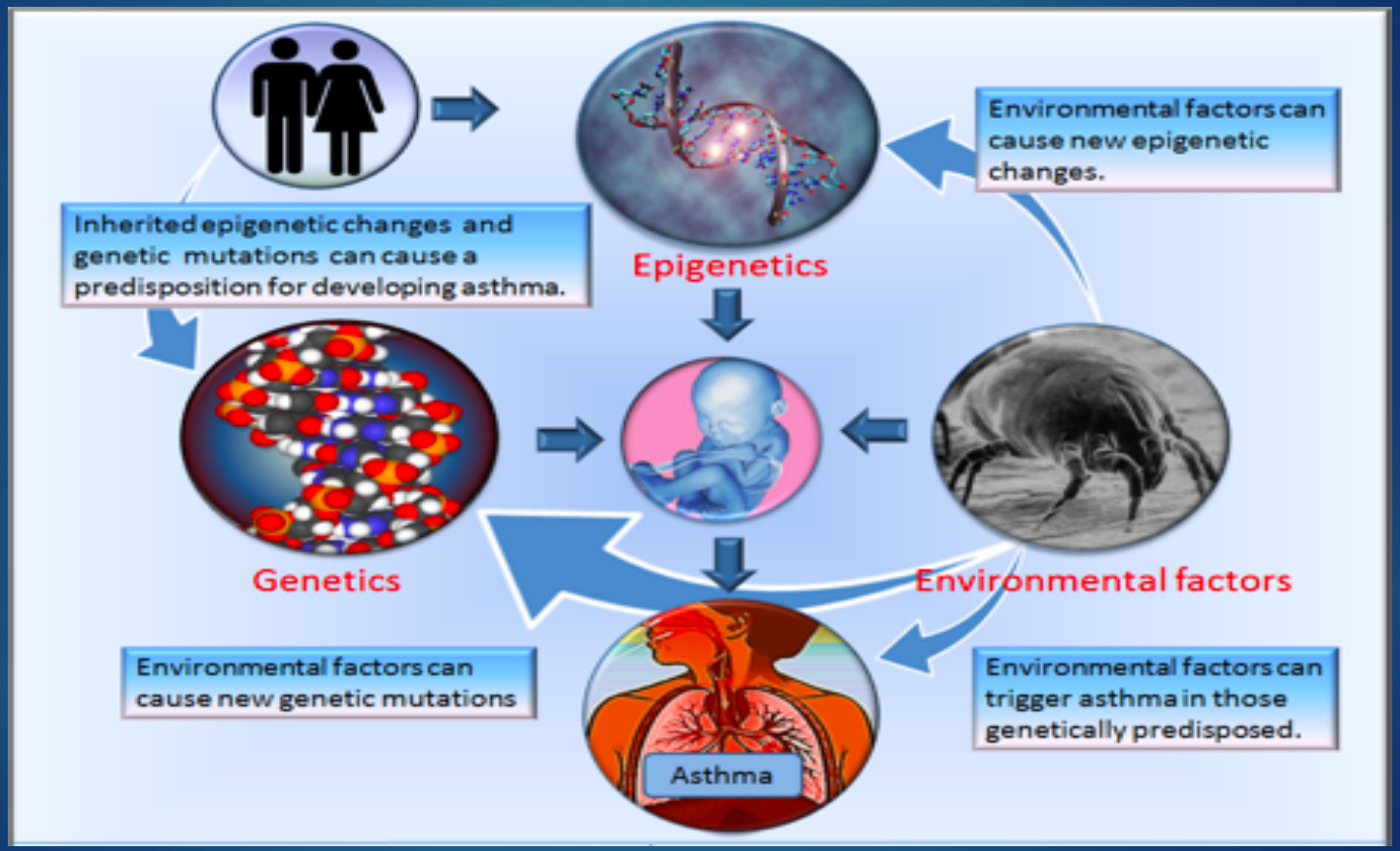
Some individuals have a heightened immune response to common environmental allergens (atopy)

Thus, they are predisposed to develop specific IgE antibodies directed against these allergens

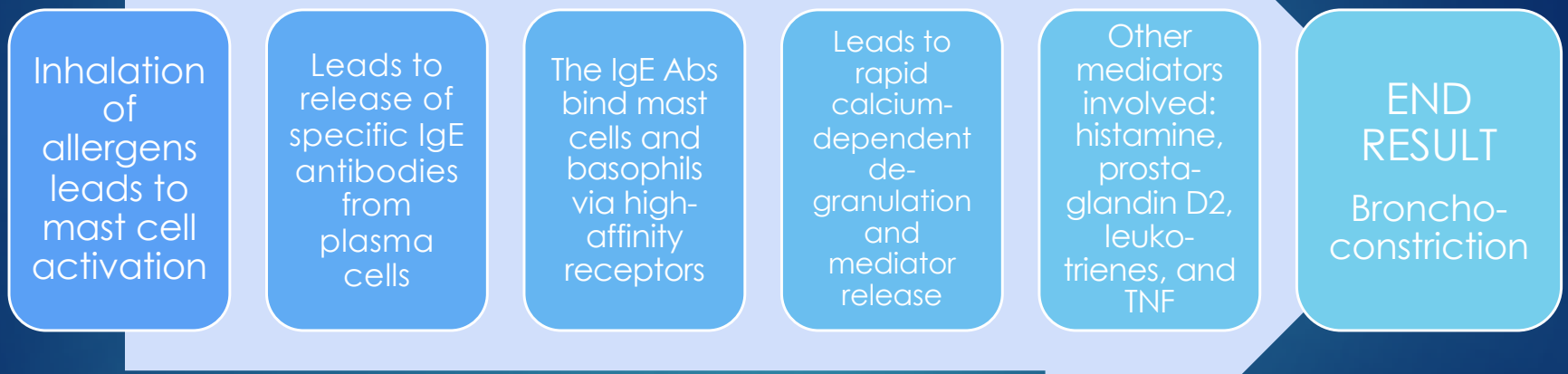
Others may have intrinsic abnormalities in airway smooth muscle function, thus, airway remodeling is more likely to occur in response to injury/inflammation

Further, interactions between epithelial and mesenchymal cells can modulate and add to the effects of airway inflammation

GENETICS/EPIGENITICS ALSO PLAY A ROLE

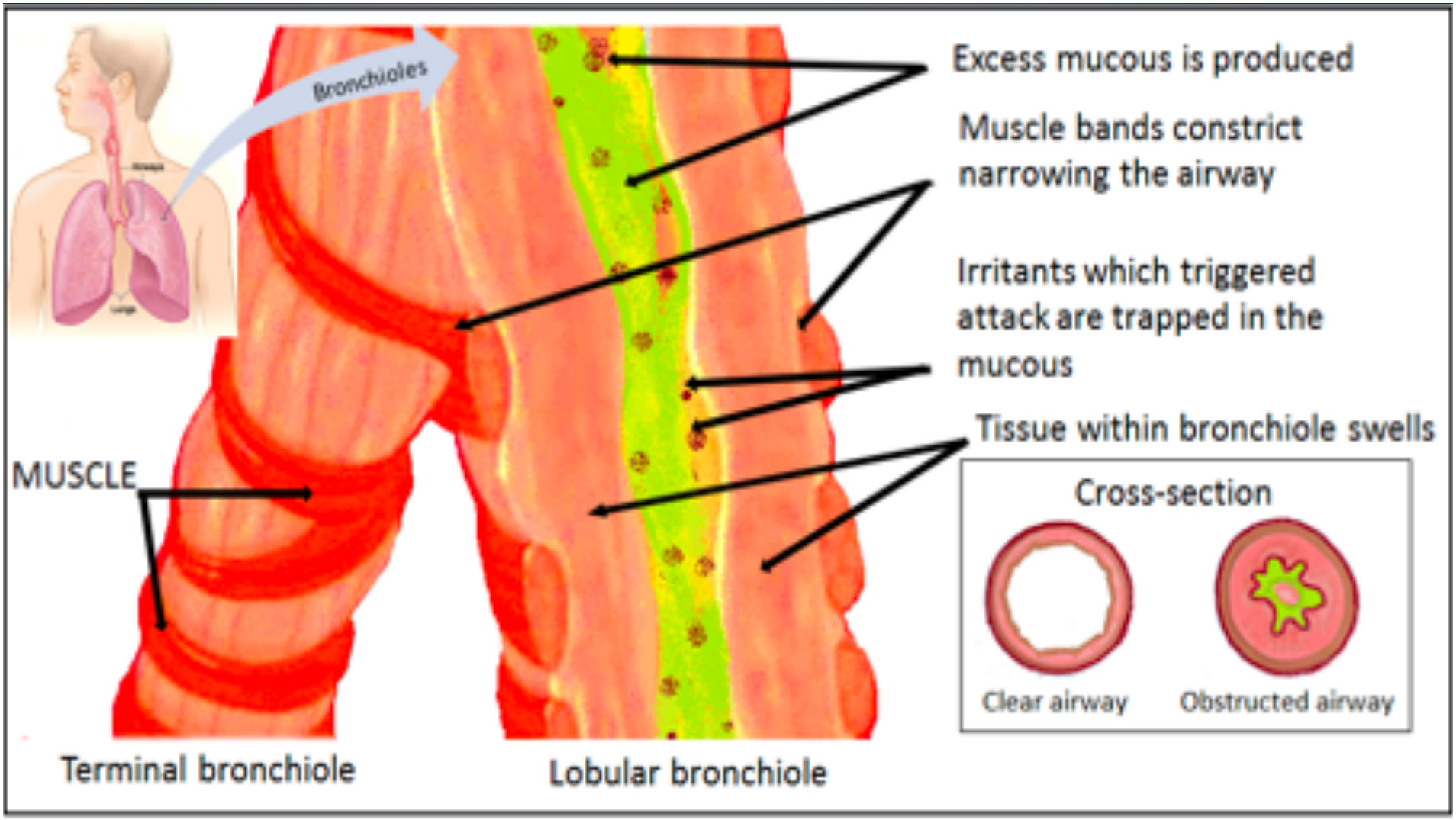


EARLY PHASE PATHOPHYSIOLOGY
MAST CELL ACTIVATION



LATE PHASE PATHOPHYSIOLOGY MORE INFLAMMATION

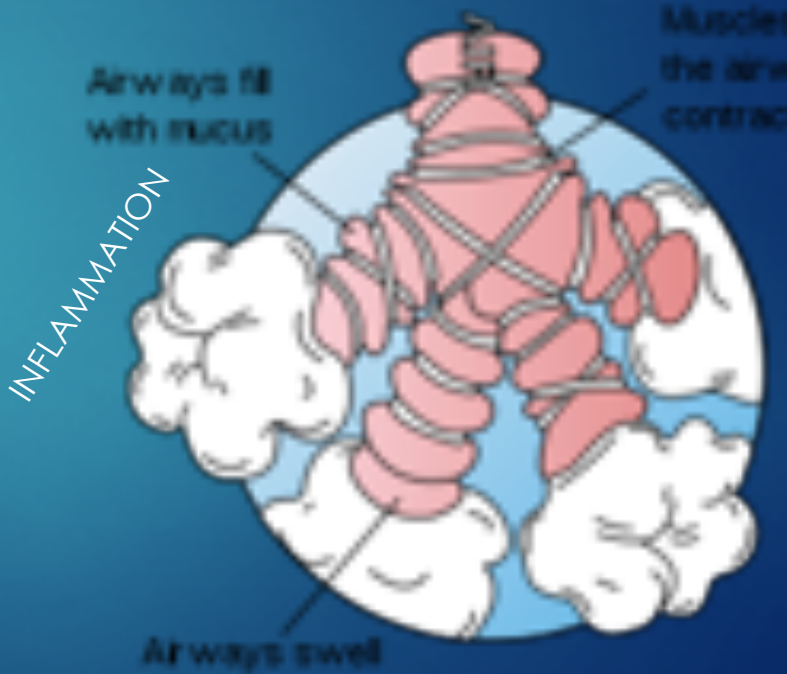
- Continued influx of inflammatory cells
  - Monocytes, dendritic cells, neutrophils, T-lymphocytes, eosinophils, and basophils
- This results in further airway smooth muscle contraction and bronchoconstriction even several hours later
- Edema and various cells cause thickening of the airway and plugging of airways with mucus or cellular debris (as seen on the next slide)


Bronchioles
Excess mucous is produced
Muscle bands constrict narrowing the airway
Irritants which triggered attack are trapped in the mucous
Tissue within bronchiole swells
MUSCLE
Cross-section
Clear airway
Obstructed airway
Terminal bronchiole
Lobular bronchiole

OR SHOWN ANOTHER WAY…

NORMAL AIRWAYS

DURING AN ASTHMA ATTACK



LONG-TERM PATHOPHYSIOLOGY

- Long-term inflammation leads to airway remodeling which includes:
  - Damage or loss of normal airway epithelium
  - Increased number of mucous-producing goblet cells
  - Thickening of the basement membrane
  - Increased airway smooth muscle mass

IMPORTANT LINKS / RESOURCES

- https://www.uptodate.com/contents/pathogenesis-of-asthma?search=asthma%20pathophysiology&source=search_result&selectedTitle=1~150&usage_type=default&display_rank=1#H1
- http://sphweb.bumc.bu.edu/otlt/MPH-Modules/PH/RespiratoryHealth/RespiratoryHealth6.html
- http://www.nlm.nih.gov/medlineplus/magazine/issues/fall11/articles/fall11pg4.html