# SUPERMINERN

S

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### INTRODUCTION

- Superantigens (SAGs) are class of microbial antigens (viral, bacterial or mycoplasma proteins).
- They non specifically activate large number of T cells and as a result they cause cytokine strom.
- SAGs bind simultaneously to specific V $\beta$  regions of T cell receptors and to the  $\alpha$  chain of MHC class II molecules.
- SAGs requires no antigen processing.

## **DIFFERENCES**

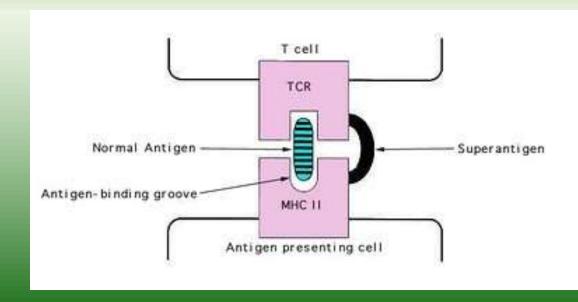
### Antigens

- Specific activation of T cells
- Binds to particular epitope on the TCR
- Activate 0.01-0.001% of T cells

### Superantigens

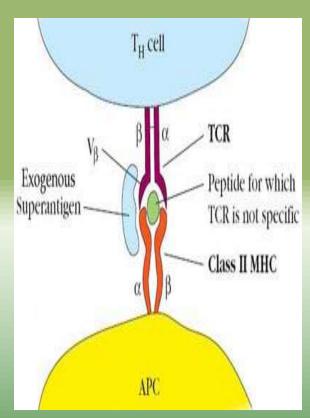
Non specific activation of T cells Binds to  $V\beta$  chain of TCR

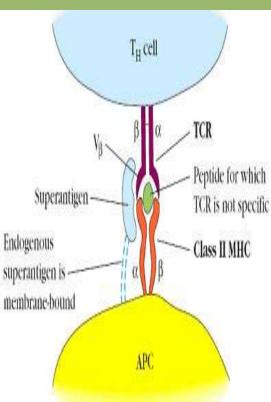
Activate 20-30% of T cells



### TYPES OF SUPERANTIGENS

- **Exogenous SAGs:** soluble proteins secreted by bacteria and a variety of exotoxins (e.g., SE, SPE, and TSST).
- Endogenous SAGs: cell membrane protein encoded certain viruses that infect mammalian cells (e.g., MMTV, and EBV).
- B-cell SAGs: These SAGs stimulate predominantly B cells. Formation of immune complexes.





### SUPERANTIGEN TOXINS

#### Staphylococcal SAg

Staphylococcal enterotoxin A, B, C, D, E, G, H, I, J,(most studied A, B and TSST1), TSST-1 [staphylococcal enterotoxin F]
. Staphylococcal protein A (SpA), [B-cell Superantigen]

#### Streptococcal SAg

Streptococcal pyrogenic exotoxins (SPE): SPE-A, SPE-B, SPE-C, SPE-D, SPE-F, -derived Superantigen)
SPE-G, SPE-H, SPE-J, SMEZ, Mitogenic factor (MF), SSA

- •Mycoplasma arthritidis Sag- MAM (mycoplasma arthritidis
- •Human liver sialoprotein- Protein Fv (B-cell SAg)
- •EB Virus HERV-K<sub>1</sub>8 env
- •HIV HIV-gp120 (B-cell SAg).
- •Yersinia pseudotuberculosis-YPM, Yersinia enterocolitis?
- •Peptostreptococcus magnus- Protein L (B-cell SAg)
- Rabies?

# STRUCTURE OF SUPERANTIGEN

- Three dimensional
- Tightly packed
- Binding regions

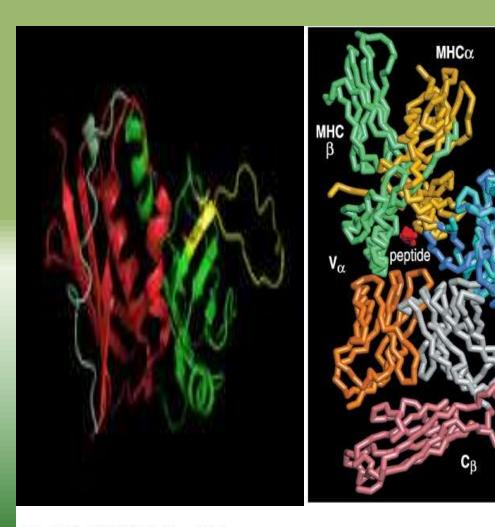
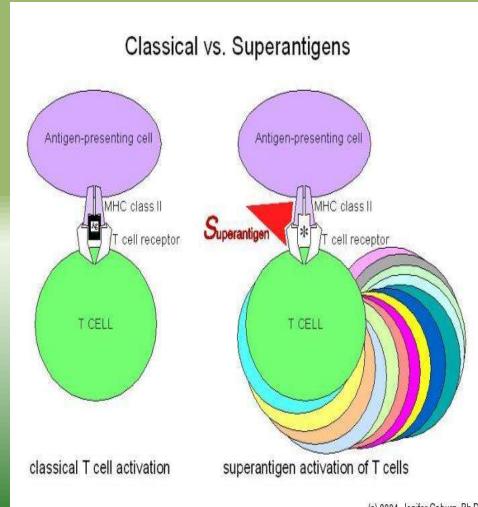


Fig 5.18 © 2001 Garland Science

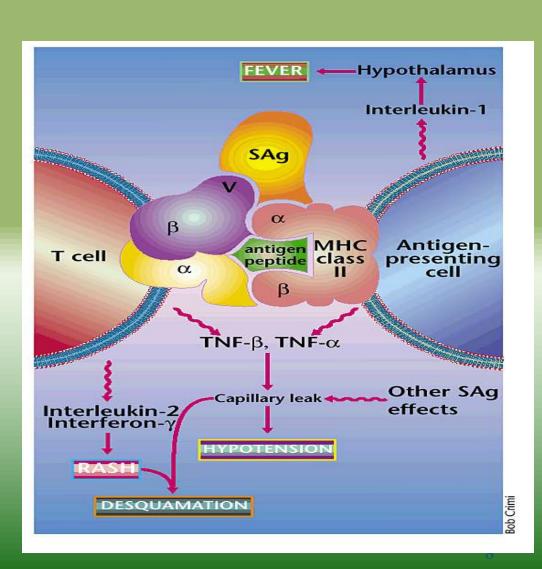
## MECHANISM OF ACTION

- Binding to MHC class ll
- Binding to T cell receptor
- T cell signalling



# SIGNIFICANCE

- Direct Effects
  - -Inflammatory activity
  - -Anergy and Deletion
  - -Cytotoxicity
  - -Isotype switch
- Indirect Effects
  - -Emesis
  - -Mitogenic activity
  - -Monocytic cell activation



### TOXIC SHOCK SYNDROME

- Staphylococcal toxin (TSST-1)
- Streptococcal toxin (STSS/ TSLS)
- Symptoms
  - \* Sudden high fever usually over 39°C
  - \* Vomiting
  - \* Diarrhoea
  - \* Rash that looks like sunburn
  - \* Dizziness
  - \* Muscle aches
  - \* Sore throat

  - \* Fainting or near fainting when standing up \* Skin peeling may occur in later stages of the illness.

### **SUMMARY**

 SAGs remain the most powerful cell mitogen discovered which can lead to massive T cell activation and excessive cytokine release.

#### REFERENCES

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