INNATE IMMUNITY

INFECTION → EFFECTOR CELLS RECRUITMENT → RECOGNITION ACTIVATION OF EFFECTOR CELLS → ELIMINATION OF INFECTIOUS AGENT

ADAPTATIVE IMMUNITY

INFECTION → CONVEYING OF ANTIGNE TOWARDS LYMPHOIDS ORGANS → RECOGNITION BY NAIVE B & T LYMPHOCYTES → CLONAL EXPANSION & DIFFERENTIATION IN EFFECTOR CELLS → ELIMINATION OF INFECTIOUS AGENT

PROTECTIVE IMMUNITY & IMMUNOLOGICAL MEMORY

RE-INFECTION → RECOGNITION BY PRE-FORMED ANTIBODIES AND EFFECTOR T LYMPHOCYTES → ELIMINATION OF INFECTIOUS AGENT

RE-INFECTION → RECOGNITION BY MEMMORY T & B LYMPHOCYTES → FAST EXPANSION & DIFFERENTIATION → ELIMINATION OF INFECTIOUS AGENT

~~NON SPECIFIC IMMUNITY~~

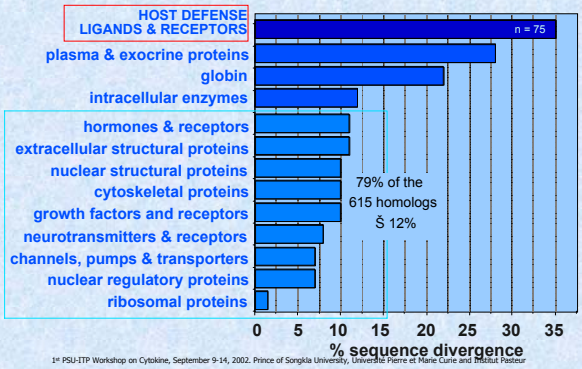
INNATE IMMUNITY

RECEPTORS	Fixed in the genome
RECOGNITION	Conserved molecular patterns (LPS, LTA, mannans, glycans)
SELF & NON-SELF DISCRIMINATION	Perfect : selected over evolutionary time
ACTION TIME	Immediate activation of effectors
RESPONSE	Costimulatory molecules : cytokines (IL-1, IL-6), chemokines (IL-8)

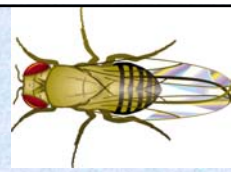
From C.A. Janeway J. Immunol, 1998, 161, 544

ANALYSIS OF HUMAN - RODENT (mouse and rat) HOMOLOGS

Murphy Cell 1993,72, 823



INNATE IMMUNITY OF DROSOPHILA

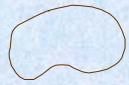


Physical and chemical barriers

Melanization & coagulation

Phagocytosis et encapsulation

Production of antimicrobial peptides



- Drosomycin
- Drosocin
- Defensin
- Cecropin
- Attacin
- Diptericin
- Metchnikowin

INNATE IMMUNITY

Recognition mechanisms

PAMPs — Pathogen Associated Molecular Pattern

PRRs — Pattern Recognition receptors

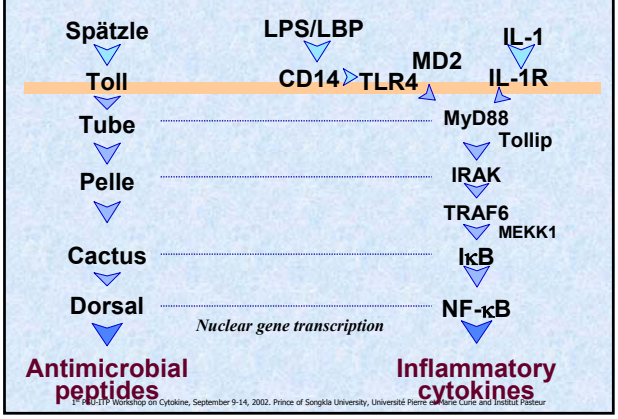
PAMPs are compounds present in infectious agents, but absent in host cells.

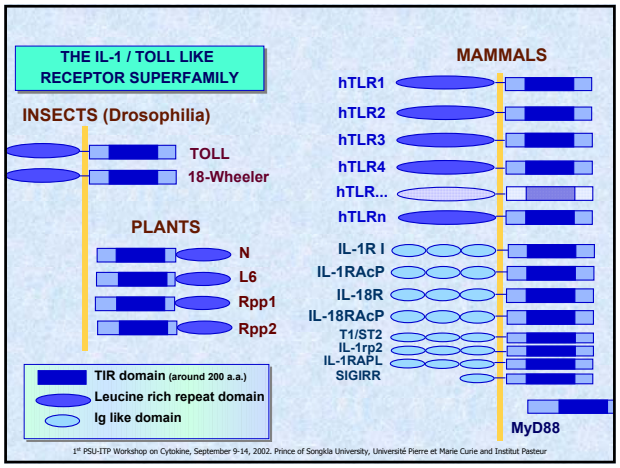
Rather conserved structures required for the survival of pathogens

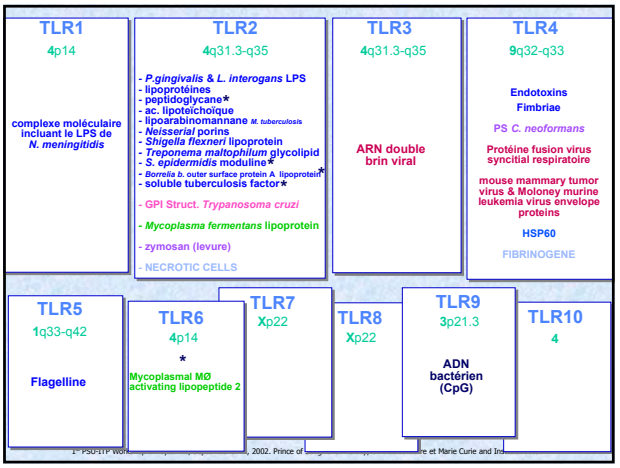
PRRs are receptors of the host selected through evolution to recognize some PAMPs.

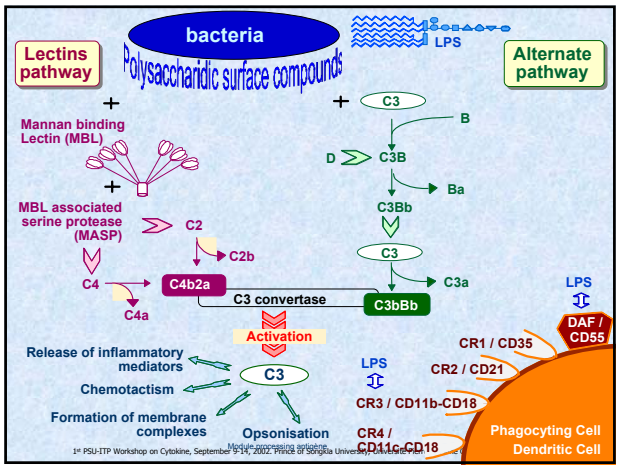
PRRs allow recognition between self and non-self

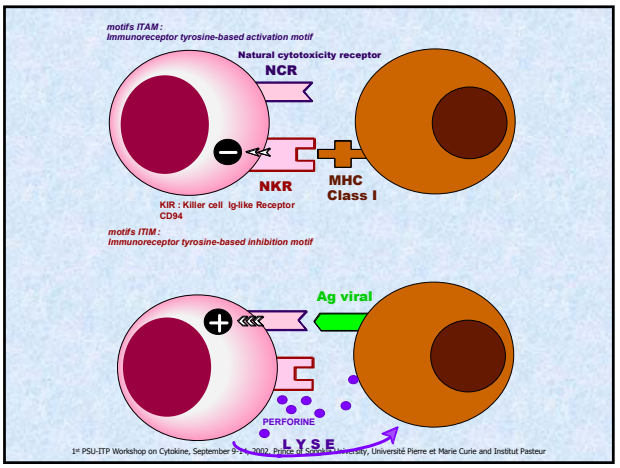
IL-1R / TLR SIGNALING











Polly Matzinger offered a new definition of immunology by postulating that immune system is not there to discriminate between self and non-self but to recognize and respond to danger signals (Annu. Rev. Immun. 1994).

She postulated that any insult to heathy tissues generate alarm signals that initiate the activation of the immune response. She conceived the concept of danger as an internal signal and pathogens are recognized by the tissue damage they generate.

"the 4 Ds of the danger model : distress, damage, destruction, and death"