



Lyme Disease

Immune system: friend or foe

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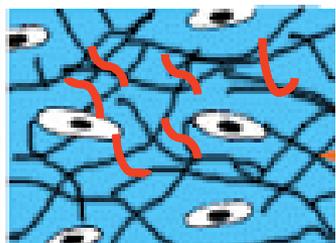
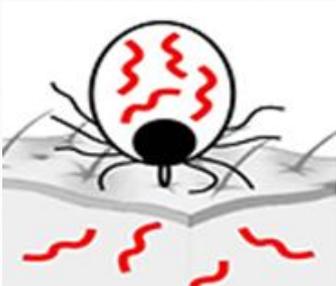
What is Lyme Disease

- ◆ Bacterial infection with worldwide prevalence
- ◆ Systemic zoonosis – disease transmitted by animal(s)
- ◆ Disease transmitted by arthropod vector, which are ticks of the genus *Ixodes*
- ◆ Ticks harbor a bacterium of genus *Borrelia*



Transmission of *Borrelia sp.*

A uninfected with *Borrelia sp.* tick (or its life form) feeds on an infected vertebrate



Migration -> into tick

Replication -> midgut

Migration -> salivary glands



Biting and releasing -> blood

Colonization -> host's skin

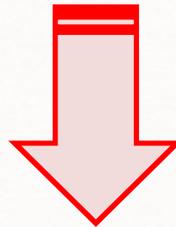
Dissimination -> other tissues/organs



Colonization -> other organs, ECM

Infection of Host by *Borrelia sp.*

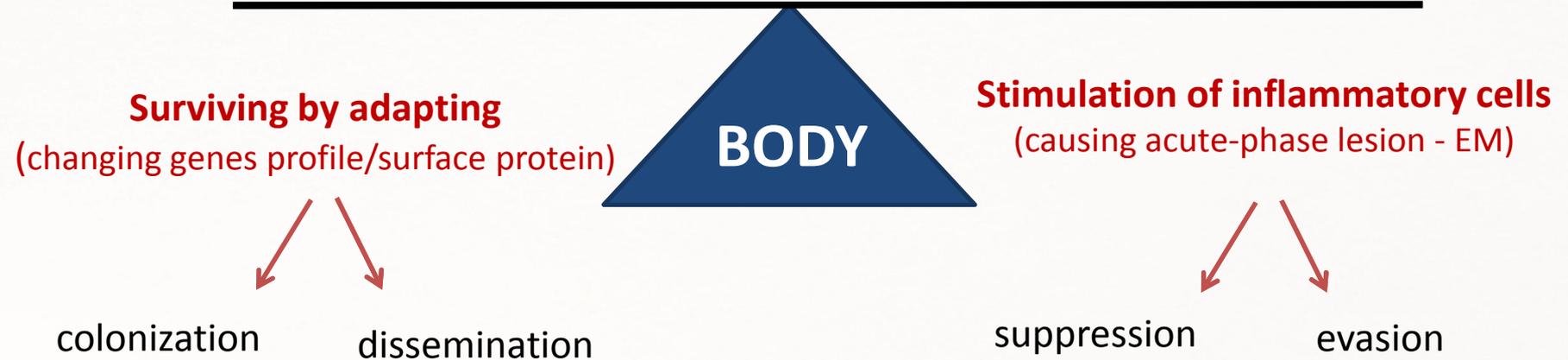
Tick's bite



- ◆ activating local inflammation
- ◆ evading host defense
- ◆ facilitating dissemination
- ◆ becoming invasive spreading quickly

Borrelia sp.

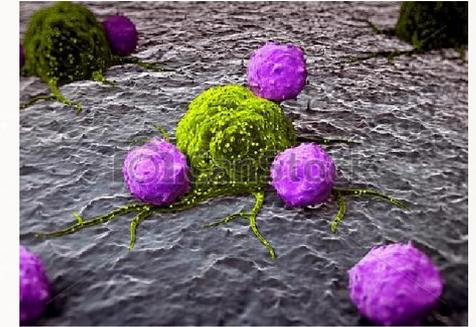
Immune system



Innate System

Response to pathogens in a generic

**Surface
barriers**

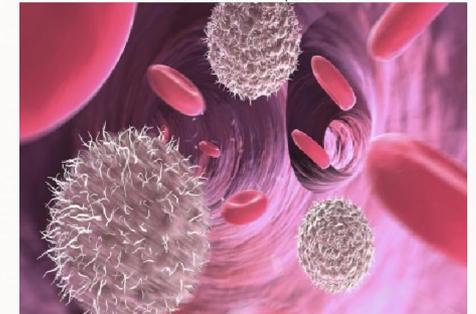


© Can Stock Photo - csp13533938

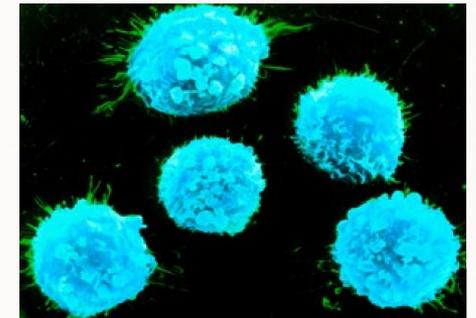
Inflammation



Complement



**Cellular
barriers**

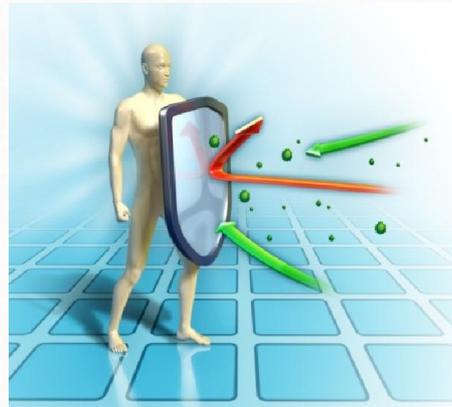
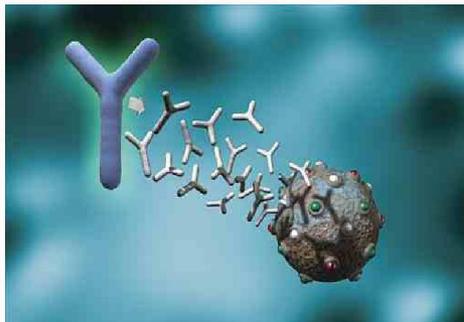


Adaptive System

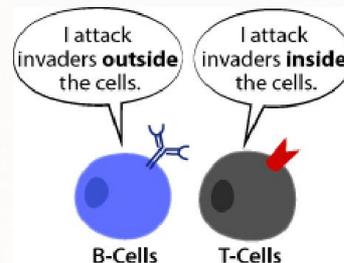
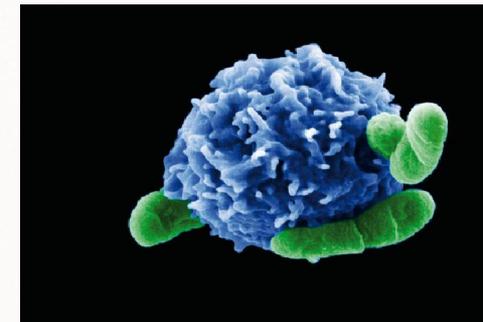
Response to pathogens not in a generic way

Types of leukocytes - lymphocytes: B and T cells derived from hematopoietic stem cells in the bone marrow

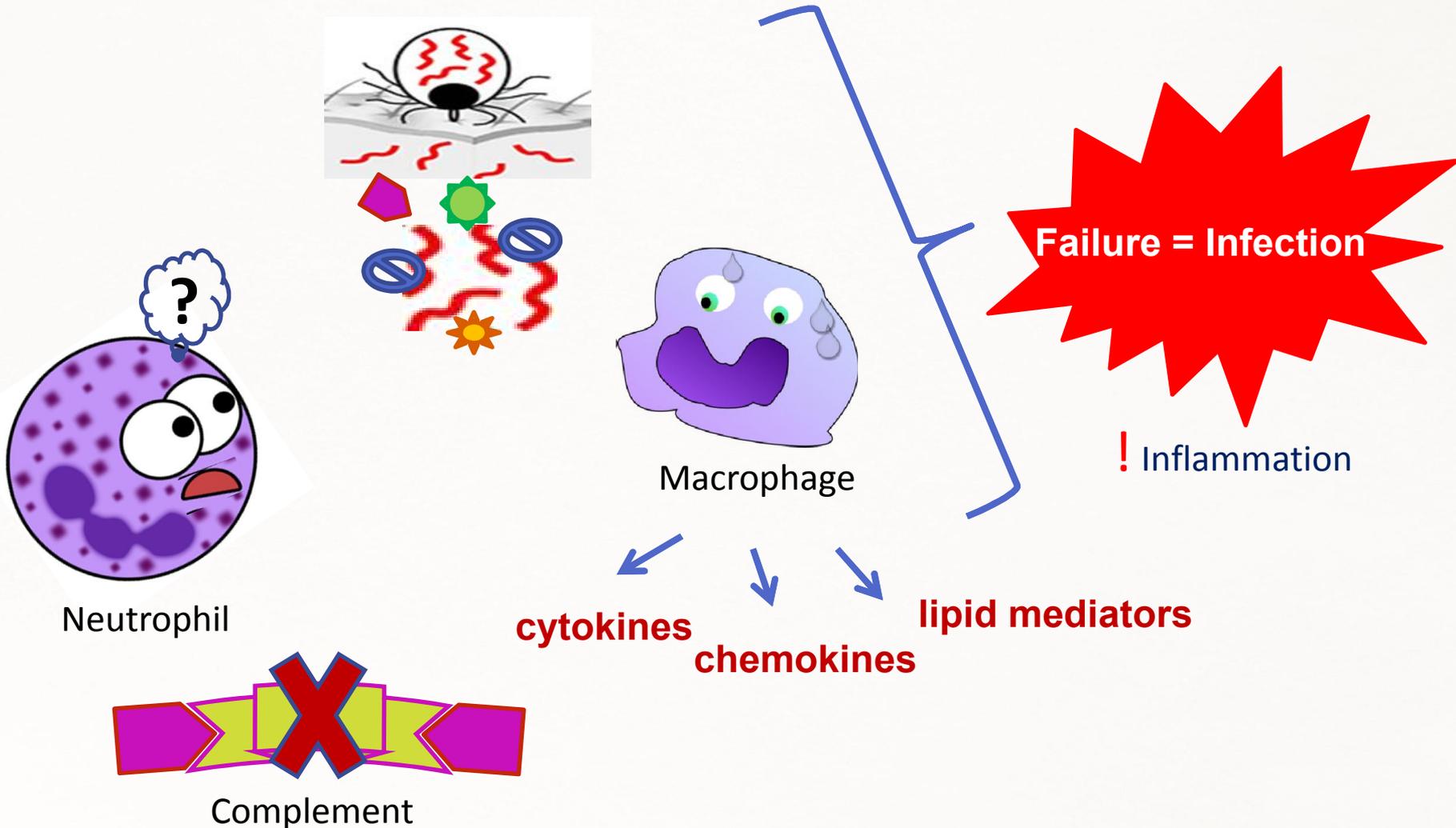
B cells are involved in the humoral immune response



T cells are involved in cell-mediated immune response



Strategy of Infection by *Borrelia* sp.



Adaptation of *Borrelia sp.*

Some genes of *Borrelia sp.* are expressed only in the mammals and others only when the bacteria are in the tick

OspA (*bbB19*)

Helps in colonization of tick's midgut, expressed in unfed ticks, down-regulated during feeding by unknown signals

OspC (*bbB19*)

Essential for initiation of infection in mammals and for colonization of certain tissues, adherence to vascular endothelium, binds to cells, host substrate(s) unknown

VisE (*bbF32*)

Required for persistent infection in mammals, adherence to vascular endothelium, binds to cells, host substrate(s) unknown



Tilly *et al.*, 2008

Survival Strategies of *Borrelia sp.*

Borrelia sp. in a host

Immune system suppression

Immune system evasion/escape

Innate:

- ◆ complement inhibition
- ◆ induction of anti-inflammatory cytokines
- ◆ tolerating of monocytes/macrophages

Adaptive:

- ◆ induction of anti-inflammatory cytokines
- ◆ tolerating of lymphocytes
- ◆ complement inhibition; plasminogen binding
- ◆ sequestration of antibodies in immune complexes

Phase and antigenic variations:

- ◆ gene conversion
- ◆ mutation and recombination
- ◆ viable expression of antigens/lipoproteins

Physical isolation (seclusion):

- ◆ intracellular: fibroblasts, ECs, neuronal cells, synovial cells, phagocytes, etc.
- ◆ extracellular: latent forms, immunologically privileged sites, motility

Embers et al., 2004

Products Required for Host Infection

Innate immune system recognize spirochetes and control their numbers but are inadequate to completely clear an infection causing persistency

- ◆ Antigen presenting cells (macrophages and dendritic cells) in the peripheral tissues (e.g., at the site of the tick bite), may subsequently migrate to lymph nodes and stimulate T cell and B cell responses
- ◆ Killing of *Borrelia sp.* by the phagocytes resident in the periphery and perhaps neutrophils attracted to the feeding lesion, NK cells
- ◆ Complement helps control *Borrelia sp.* numbers by opsonizing the bacteria (facilitating phagocytosis) or by direct killing via the alternative pathway

Bacteria survive in the face of an antibody either due to “hiding” in sites protected from antibodies or evasion antibody reactivity by varying antigens or masking reactive proteins



Crucial Aspects of Effective Therapy

What should be taken into consideration during LD treatment

1. Eradication of pathogens
2. Boosting immunity and controlling of inflammation
3. Metabolic support for affected organs
4. Dietary support



Tested Formula 1

Composition of formula1 selected for *in vivo* studies:

Vitamin D3

Vitamin B-complex

Vitamin C

Baicalein (*Scutellaria baicalensis*)

10-HAD (Royal jelly)

Iodine/Kelp

Monolaurin (Coconut oil)

Luteolin (*Sophora japonica*)

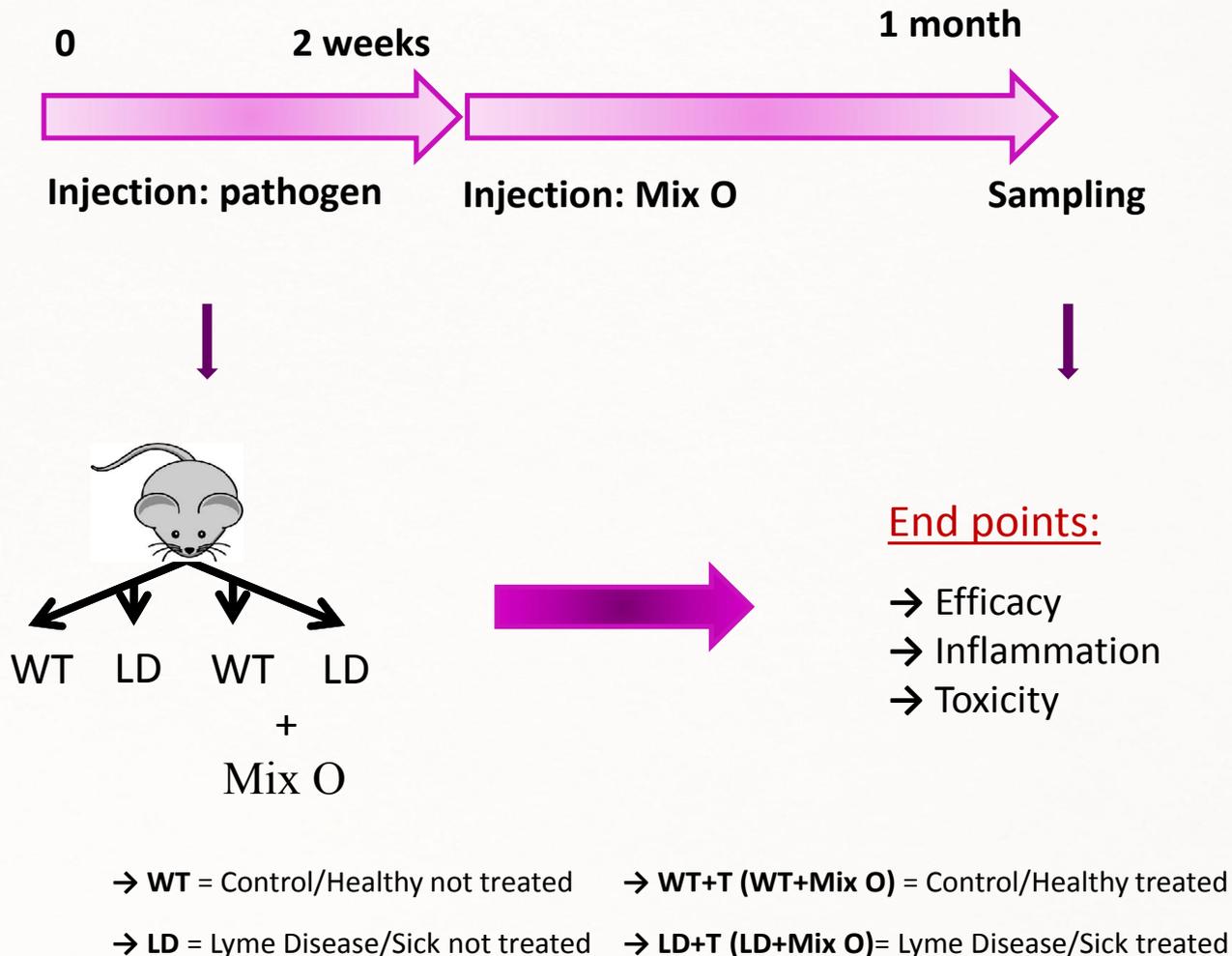
Rosmarinic acid (*Rosmarinus officinalis*)

Goc et al., JAM 2015



Aim:

Testing of Formula 1 in Lyme Disease Animal Model



Examinations

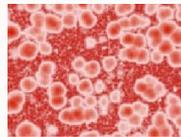
- ◆ Adverse events
- ◆ Food intake, water consumption
- ◆ Weight
- ◆ Blood and tissue sampling for determining the laboratory parameters



Clinical Parameters



Weight = No change



Morphology



Food/Water = No change

WBC
Neutrophils

RBC
Monocytes

HGB
Eosinophils

HCT
Basophils

MCV
Lymphocytes

MCH/MVHC
Platelets

→ **WT, WT+T, LD+T** = No change

→ **LD** = Elevated level of monocytes

Pathogen Detection

Skin

Bladder

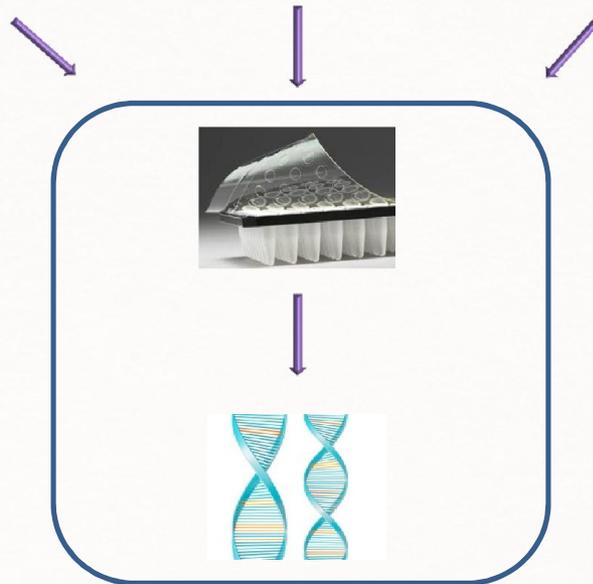
Kidney

Liver

Spleen

Joint

Heart

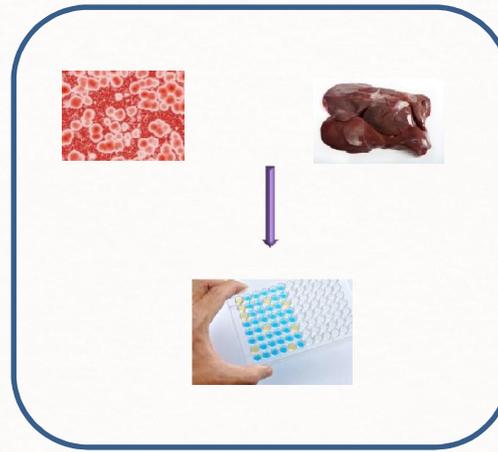


→ **WT, WT+T** = No presence

→ **LD** = Presence

→ **LD+T** = Presence reduced to 90%

Inflammation and Toxicity



Cytokines Panel CRP SAA Clusterin KIM-1 RPA-1 Haptoglobin Fibrinogen GGT Creatine Kinase SDH Creatinine

Inflammation

→ WT, WT+T, LD+T = No
→ LD = Yes

Toxicity

→ No change

Summary

- ◆ Selected formula 1 significantly reduced pathogen's presence *in vivo*
- ◆ Selected formula 1 effectively reduced levels of inflammatory markers *in vivo*
- ◆ Selected formula 1 did not displayed toxic effects *in vivo*



“Scientific Guide in Natural Approach to LD for HP”



Thank you

Lyme Research Laboratory



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