The neuroinflammatory response refers to the activation of the brain and spinal cord's immune system in response to injury, infection, neurodegenerative processes, or other disturbances in the central nervous system (CNS). It’s a double-edged sword: it can be protective in acute settings, but damaging if it becomes chronic or dysregulated.

Here’s a breakdown of key elements:

 Key Components of the Neuroinflammatory Response: Microglia:

The resident immune cells of the CNS.

When activated, they release cytokines, chemokines, and reactive oxygen species (ROS).

Can assume either neuroprotective (M2-like) or neurotoxic (M1-like) phenotypes.

Astrocytes:

Support neurons and maintain the blood-brain barrier (BBB).

When reactive (astrogliosis), they can amplify inflammation and produce pro-inflammatory mediators.

Cytokines & Chemokines:

Includes IL-1β, TNF-α, IL-6, and MCP-1.

These signaling molecules coordinate immune responses but can damage neurons if uncontrolled.

Blood-Brain Barrier (BBB):

Normally restricts peripheral immune cells from entering the CNS.

Neuroinflammation can disrupt the BBB, allowing peripheral immune cells to infiltrate and worsen the damage.

⚠ Causes of Neuroinflammation: Trauma (e.g., TBI, spinal cord injury)

Infections (e.g., meningitis, encephalitis)

Neurodegenerative diseases (e.g., Alzheimer’s, Parkinson’s, MS)

Autoimmune diseases (e.g., neuromyelitis optica)

Chronic stress or systemic inflammation

 Clinical Relevance: Acute neuroinflammation helps contain injury or infection.

Chronic neuroinflammation is implicated in neurodegeneration, cognitive decline, and psychiatric disorders.

Modulating neuroinflammation is a therapeutic target in conditions like multiple sclerosis, epilepsy, and glioblastoma.