Foreign body response

Foreign body response (FBR) is a complex biological process that occurs when a foreign object is implanted into a living organism. The response is characterized by a series of cellular and molecular events that attempt to remove the foreign material and restore tissue homeostasis.

When a foreign body is implanted, the immune system recognizes it as a threat and triggers a series of responses aimed at removing it. Immune cells, such as macrophages and neutrophils, migrate to the site of the implant and attempt to engulf and degrade the foreign material. However, some foreign materials, such as synthetic polymers and metals, are resistant to degradation and cannot be removed by the immune system.

In response to these persistent foreign bodies, macrophages can fuse together to form multinucleated giant cells (MGCs), which surround and attempt to wall off the foreign material. The MGCs can also release enzymes and reactive oxygen species that attempt to degrade the foreign material.

As the FBR progresses, a fibrous capsule may form around the foreign body, consisting of collagen, fibronectin, and other extracellular matrix proteins. This capsule can isolate the foreign body from the surrounding tissue, but it can also limit the diffusion of oxygen and nutrients to the implant and impede its integration with the host tissue.

The severity and duration of the FBR depend on several factors, including the size, shape, and composition of the foreign body, as well as the location and immune status of the host tissue. Chronic or unresolved FBRs can lead to tissue damage, implant failure, and chronic inflammation. Therefore, minimizing the FBR is a major goal in the design of biomedical implants and materials

Lipids can also regulate immune activity and their presence may also contribute to biomaterialinduced foreign body responses (FBR) and fibrosis. Here we demonstrate that the surface presentation of lipids on implant affects FBR by influencing reactions of immune cells to materials as well as their resultant inflammatory/suppressive polarization. We employed time-of-flight secondary ion mass spectroscopy (ToF-SIMS) to characterize lipid deposition on implants that were surfacemodified chemically with immunomodulatory small molecules. Multiple immunosuppressive phospholipids (phosphatidylcholine, phosphatidylinositol, phosphatidylethanolamine, and sphingomyelin) were all found to deposit preferentially on implants with anti-FBR surface modifications in mice. Significantly, a set of 11 fatty acids was enriched on unmodified implanted devices that failed in both mice and humans, highlighting relevance across species. Phospholipid deposition was also found to upregulate the transcription of anti-inflammatory genes in murine macrophages, while fatty acid deposition stimulated the expression of pro-inflammatory genes. These results provide further insights into how to improve the design of biomaterials and medical devices to mitigate biomaterial material-induced FBR and fibrosis ¹⁾.

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