While some of these inflammatory episodes are linked to a problem of chemical nature (accidental injection of chemical contaminants or incorrect doses of certain compounds), the majority of cases are directly associated with the presence of contaminants of microbial origin that are present in the solutions used to prepare the dialysis solutions.

Lipopolysaccharides (LPSs) and peptidoglycans (PGNs) are the main contaminants of microbial origin which present a high risk of triggering an inflammation when they are present in trace amounts.

The standard tests theoretically make it possible to discard batches which are loaded with contaminants of this type and which therefore present a health risk. However, these tests are not satisfactory, since aseptic inflammatory episodes are still reported, even though the solutions had been declared healthy.

Thus, despite the constant attention of those participating the field, in terms of reducing the risk of contaminations, in particular by improving detection thereof, there still remains a need to improve the performance levels of the detection of contaminants which can induce an inflammation.

Summary

In one aspect, there is provided a method for detecting pro-inflammatory contaminants of glucose polymers, said contaminants being capable of triggering, separately or in combination, an inflammatory reaction, characterized in that it comprises at least one *in vitro* inflammatory response test using a cell line which makes it possible to detect at least one inflammatory response factor, the cell line being either a macrophage or a macrophage-differentiated cell line, or a cell expressing one or more Toll-Like Receptors (TLRs) or NOD-like receptors such as TLR2, TLR4 or NOD2 and making it possible to detect the responses of the receptor(s), or a combination thereof.

Detailed description of the invention

It is to the Applicant company's credit to have taken into account the presence of molecules capable of exacerbating the inflammatory response induced by other contaminants, in particular LPS or PGNs, especially via a mechanism of cooperation between TLRs (Toll-Like Receptors) and NOD (Nucleotide-binding Oligomerization Domain-containing protein)-like receptors. Indeed, to consider only the effect of the isolated contaminants on inflammation is reductive.

Contrary to LPS, which is a ligand recognized by receptors of TLR4 (Toll-Like Receptor) type, PGN (but also numerous glycolipids and lipopeptides) is a ligand recognized by receptors of TLR2 type which induces a weak inflammatory response in *in vitro* and *in vivo* models, thereby implying that these molecules must be present at higher concentrations in order to be detected.

Thus, in the models using mononuclear cells (PBMCs, primary monocytes/macrophages or monocyte lines), LPS induces a significant response for concentrations of about 1 ng/ml, whereas PGN concentrations at least 100 times higher are required to obtain a similar response (w/w ratio).

In addition, while soluble PGNs (MW $\approx$ 125 kDa) induce an inflammatory response via the activation of TLR2, the depolymerization products thereof, the minimum structure of which still bioactive is muramyl dipeptide (MDP), interact with NOD-like intracellular receptors.