ERRATUM

The following passages must be amended:

1. page *23*, addition of a citation in Figure 7: Production and structure of oxidized phospholipids and its components (modified after Ashraf & Srivastava, 2010; Bochkov et al. 2010).
2. page *24*, addition of a citation in Figure 8: Anti-inflammatory effects and mechanisms of OxPL (Bochkov et al. 2010).

OxPL mediate a diverse spectrum of bioactivities. For instance, OxPL are involved in the inhibition of lipopolysaccharide-induced activation of toll-like receptors (Bochkov et al., 2010).

Furthermore, OxPL are suggested to assume a role in acute inflammation (sepsis or acute lung injury) (Imai et al., 2008), neurodegenerative diseases like Alzheimer´s or Parkinson´s and chronic inflammation (Furnkranz et al., 2005; Usui et al., 2009). The peroxidation of phospholipids can lead to an enrichment of lysoforms resulting from both enzymatic and non-enzymatic hydrolysis.

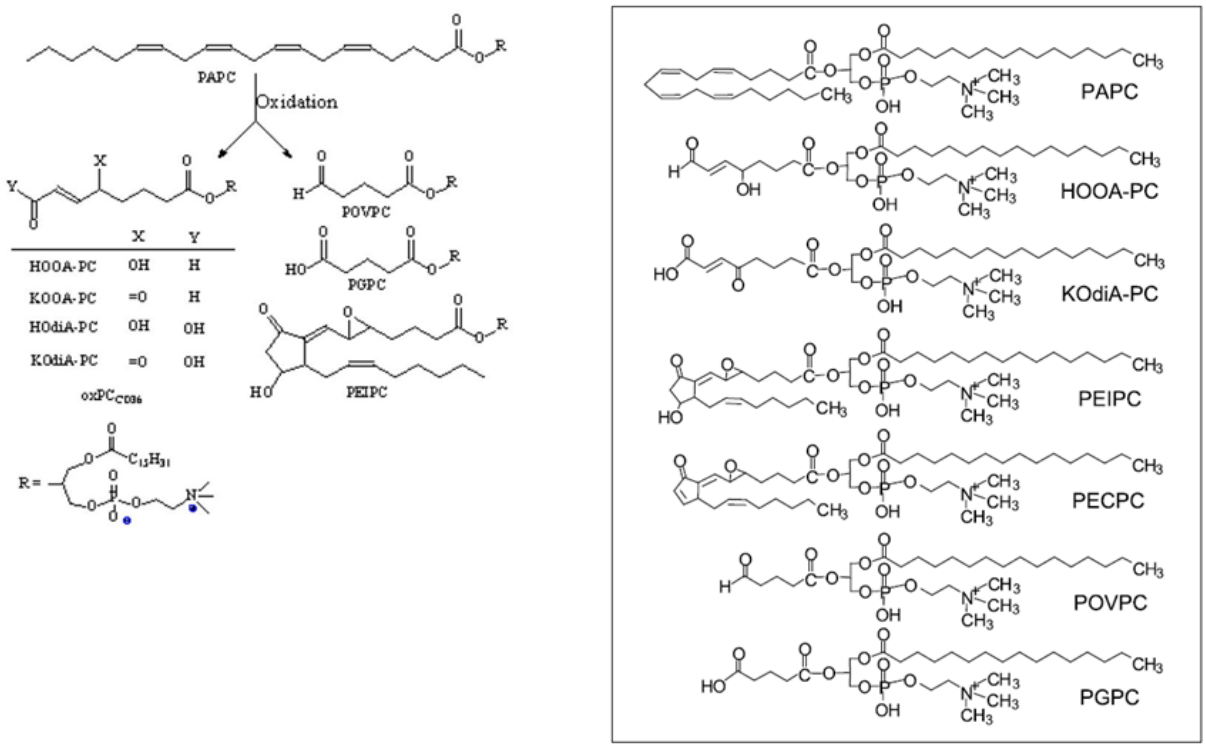

Figure 1: Production and structure of oxidized phospholipids and its components (modified after Ashraf & Srivastava, 2010; Bochkov et al. 2010). Oxidized phospholipids are generated by oxidation of PAPC with reactive oxygen species. Oxidation of phospholipids and subsequent reactions form a broad variety of oxidized phospholipids with most diverse bioactivities.

Lysophospholipids can both bind and activate G protein-coupled receptors as well as Lysophosphatidic acid *receptors* 1 and 4 (LPA1 to LPA4) (Anliker and Chun, 2004; Tomura et al., 2005).

It has already been shown that OxPAPC and the component PEIPC facilitate the prostaglandin E2 *receptor* 2 and prostaglandin D2 *receptor* 1, leading to inflammation by mediation enhancement of cAMP levels (Li et al., 2006). Furthermore, OxPL and in particular its components PGPC and POVPC stimulate peroxisome proliferator-activated receptors (PPAR) and toll-like receptor 4 (Lee et al., 2000; Davies et al., 2001; Walton et al., 2003). In addition, OxPL have a role in interacting with scavenger receptors CD36 and SR-BI; both are important for the detection of apoptotic cells and the production of foam cells (Podrez et al., 2002; Bochkov et al., 2010).

Mohammad Z. Ashraf and Swati Srivastava (2012). Oxidized Phospholipids: Introduction and Biological Significance, Lipoproteins - Role in Health and Diseases, Prof. Gerhard Kostner (Ed.), InTech, DOI: 10.5772/50461. Available from: https://www.intechopen.com/books/lipoproteins-role-in-health-and-diseases/oxidized-phospholipids-introduction-and-biological-significance

In summary, OxPL have a broad pharmacological spectrum and have a role in several receptor-mediated mechanisms with an activation of quite a few signaling pathways (Bochkov et al., 2010).

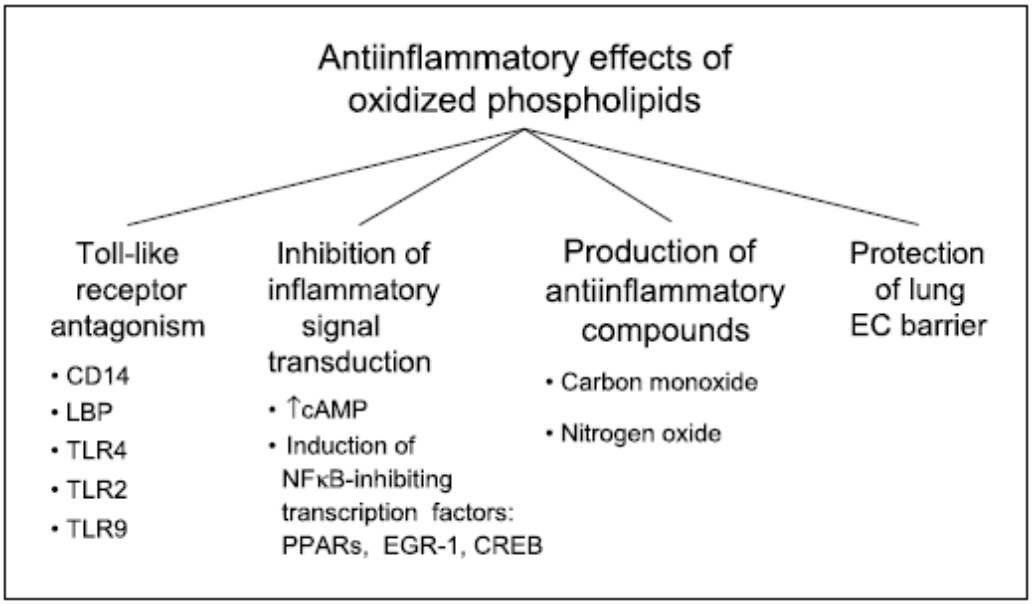

Figure 2: Anti-inflammatory effects and mechanisms of OxPL (Bochkov et al. 2010).

11.1 The voltage-gated sodium channel $Na_V1.9$

The transmission and transduction of nociceptive signals after inflammation or injury is depended on an enhanced excitability of sensory neurons. In this, voltage-gated sodium channels have a critical role. They may influence increased responsiveness to endogenous pronociceptive irritants such as OxPL. Voltage-gated sodium channels have a conserved structure of 24 transmembrane segments arranged into four homologous domains (DI-DIV). Each domain consists out of six transmembrane segments (S1-S6) (Catterall et al., 2005). There are nine genes encoding for the pore-forming alpha-subunit of the channels (SCN1A-SCN5A; SCN8A-SCN11A). The voltage-sensing domain can be found in S4 domains, while the segments S5 and S6 contribute to the pore domain. The channel pore is selective for sodium ions.

$Na_v1.8$ and $Na_v1.9$ are resistant to micromolar concentrations of TTX (TTX-R), while $Na_v1.1$, $Na_v1.6$, and $Na_v1.7$ are inhibited by nanomolar concentrations of the neurotoxin tetrodotoxin (TTX-Sensitive, TTX-S). The three VGSC $Na_V1.7$, $Na_V1.8$ and $Na_V1.9$ are in particular expressed on peripheral neurons and all three channels have been shown to have an important role in human pain disorders (Faber et al., 2012; Dib-Hajj et al., 2013; Huang et al., 2013; Leipold et al., 2013; Leipold et al., 2015).