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).

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 Nav1.9

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\(_{v}\)1.8 and Na\(_{v}\)1.9 are resistant to micromolar concentrations of TTX (TTX-R), while Na\(_{v}\)1.1, Na\(_{v}\)1.6, and Na\(_{v}\)1.7 are inhibited by nanomolar concentrations of the neurotoxin tetrodotoxin (TTX-Sensitive, TTX-S). The three VGSC Na\(_{v}\)1.7, Na\(_{v}\)1.8 and Na\(_{v}\)1.9 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).