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Oxidized phospholipids in ferroptosis, immunity and inflammation

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A B S T R A C T

Oxidized phospholipids (oxPL) are generated by enzymatic or non-enzymatic reactions and play diverse roles in immunity and inflammation. OxPL are elevated in tissues from many human diseases and are now recognized as endogenous damage-associated molecular patterns (DAMPs) that alert the immune system to challenge. Early studies focused on the role(s) of non-enzymatically-formed oxPCs in cardiovascular disease, while more recently, the controlled generation of enzymatically-oxidized PL (eoxPL) in blood cells and their participation in physiological hemostasis has been delineated. In the last decade, there has been an explosion of research into their formation and roles in ferroptosis, a form of cell death driven by iron and lipid oxidation. This mini review aims to bring the reader up to date with recent work in this area, focused on discoveries over the last few years that firmly extend our knowledge of the roles of oxPL as mediators of ferroptosis, innate and adaptive immunity.

1. Generation and structures of oxPL

OxPL can be generated in vivo either by enzymatic or non-enzymatic peroxidation of esterified PUFAs. The formation of oxPL in human inflammatory disease has been known about for decades. Seminal studies undertaken in the 1980–1990's first established that lipid oxidation, including of PL, is a characteristic feature of atherosclerotic lesions, where large numbers of diverse species are associated with foam cells. These observations led to a quest to understand the formation, and biological roles of oxPL, particularly of phosphatidylcholine (PC) that were identified in lesions, and subsequently found to be bioactive through mechanisms consistent with a central role in promoting disease (summarized in Ref. [1]). Early studies on oxPL in atherosclerosis focused mainly on species that contained truncated FA chains, such as 1-hexadecanoyl-2-(5(6)-epoxy-9-oxo-11-hydroxy-7E,14Z-prostadienoyl)-*sn*-glycero-phosphocholine (POVPC) and 1-hexadecanoyl-2-glutaroyl-*sn*-glycero-3-phosphocholine (PGPC) (Fig. 1) [2]. As these were the forms identified in atheroma lesions, it was generally considered that oxPL formation was an uncontrolled free radical dependent event in vivo. However, the action of vascular LOXs and COXs can also lead to formation of truncated oxPL, if initial hydroperoxide species are not rapidly reduced and therefore undergo beta-scission of PUFA chains. Through this, propagation of peroxidation would form oxPL indistinguishable from those generated through Fenton-type reactions. Although mainly considered to be non-enzymatic in origin, since they were truncated structures generated through secondary oxidation, the initiation of their formation is still unknown and the relative impact of enzymatic vs non-enzymatic mechanisms in formation of oxPL in atherosclerotic vessels is an open question. In that regard, chiral analysis of fatty acyl (FA) chains released from atheroma-derived oxPL has shown that several can originate from either enzymatic or non-enzymatic reactions in that tissue [3–5].

Many studies describing physiological and pathological effects of oxPL used oxidized palmitoyl-arachidonoyl-phosphatidylcholine (OxPAPC) that was generated by exposure of precursor PAPC to air. OxPAPC comprises dozens of oxidized species with either full-length or truncated residues [6]. The most abundant molecular species which are present in oxPAPC preparations are shown in Fig. 1, however there are no standardized protocols for generating this, and defined mixtures of oxPAPC do not exist. Essentially all biological effects of OxPAPC have been reproduced using one or several of synthetic molecular oxPL species containing either truncated residues such as POVPC and PGPC, truncated residues with α,β-carbonyls [7], full-length cyclic residues such as 1-hexadecanoyl-2-(5(6)-epoxy-9-oxo-11-hydroxy-7E,14Z-prostadienoyl)-*sn*-glycero-phosphocholine (PEIPC), or full-length linear hydroperoxides (Fig. 1) [8]. Because only a few synthetic species are commercially available, the structure-activity relationships are poorly investigated and will not be discussed in this minireview.

Extensive research has identified diverse oxPL formed in many diseases including infection, auto-immunity and cancer. A subset of oxPL comprising mainly phosphatidylethanolamine (PE) species termed enzymatically-oxidized PL (eoxPL), generated through regulated

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processes during innate immune response was discovered and characterized. Enzymatic mechanisms involve either direct oxidation of PL-esterified PUFA by 15-lipoxygenases (LOX) including 15-LOX1 (*ALOX15*) and 15-LOX B (*ALOX15B*) [9–12], or esterification of LOX or cyclooxygenase (COX) products with lysophospholipids via the Lands' cycle [13–15]. The most abundant isomers are 12-HETE-PEs generated by 12-LOX in thrombin-activated platelets, with levels in the ng range and around a third of the 12-HETE generated ending up rapidly esterified into PL pools [13]. Similar to this, neutrophils generate a series of 5-HETE-containing PL in response to agonist activation [14], while IL-4 treated human monocytes form 15-HETE-PEs via the action of 15-LOX1 [11]. The murine 12/15-LOX in peritoneal macrophages similarly generates both 12- and 15-HETE-PLs [16], while platelet COX-1 is a significant source of both 11- and 15-HETE-PLs [17]. Overall, levels of these lipids are considered to be relatively abundant, being in the ng/mg tissue or 10^6 cells range. The identification of eoxPL was originally achieved using precursor scanning and later using untargeted methods [18]. Methods for quantitation of the most abundant, including synthesis of standards, were published previously, with more recent studies applying novel computational approaches to aid oxPL identification [19,20].

An extensive review in 2009 summarized the state of the art at that time [1]. Other reviews that summarize our knowledge of oxPL focus on their potential role as biomarkers, drug targets and drug leads [21], and others on their chemical [22] or enzymatic [23,24] generation. As an update to these, this minireview will focus on two areas where recent research has significantly extended our knowledge of oxPL during the last 5 years, ferroptosis, and innate and adaptive immunity.

2. Ferroptosis: involvement in cancer and neurological diseases

The concept that Fenton-like chemistry involving lipid peroxidation occurs in human disease and is detrimental to cell and tissue function has been known for decades, with the free radical hypothesis of aging being extensively researched during the 1980s-1990's [25]. However, since antioxidants were not found to be capable of preventing disease or delaying aging back then, research into this idea waned. However, recent years have seen a resurgence in interest, leading to the coining of the term ferroptosis in 2012 [26]. This was demonstrated as a non-necrotic and non-apoptotic form of cell death that is triggered by uncontrolled phospholipid peroxidation, itself promoted by loss of glutathione peroxidase 4 (GPX4) (Fig. 2) [27]. It was postulated that ferroptosis had potential to be harnessed for promoting cancer cell death or targeted for prevention of neurodegeneration [26].

In the last 13 years, huge strides have been made in delineating many of the fundamental underpinning processes involved, and a new impetus

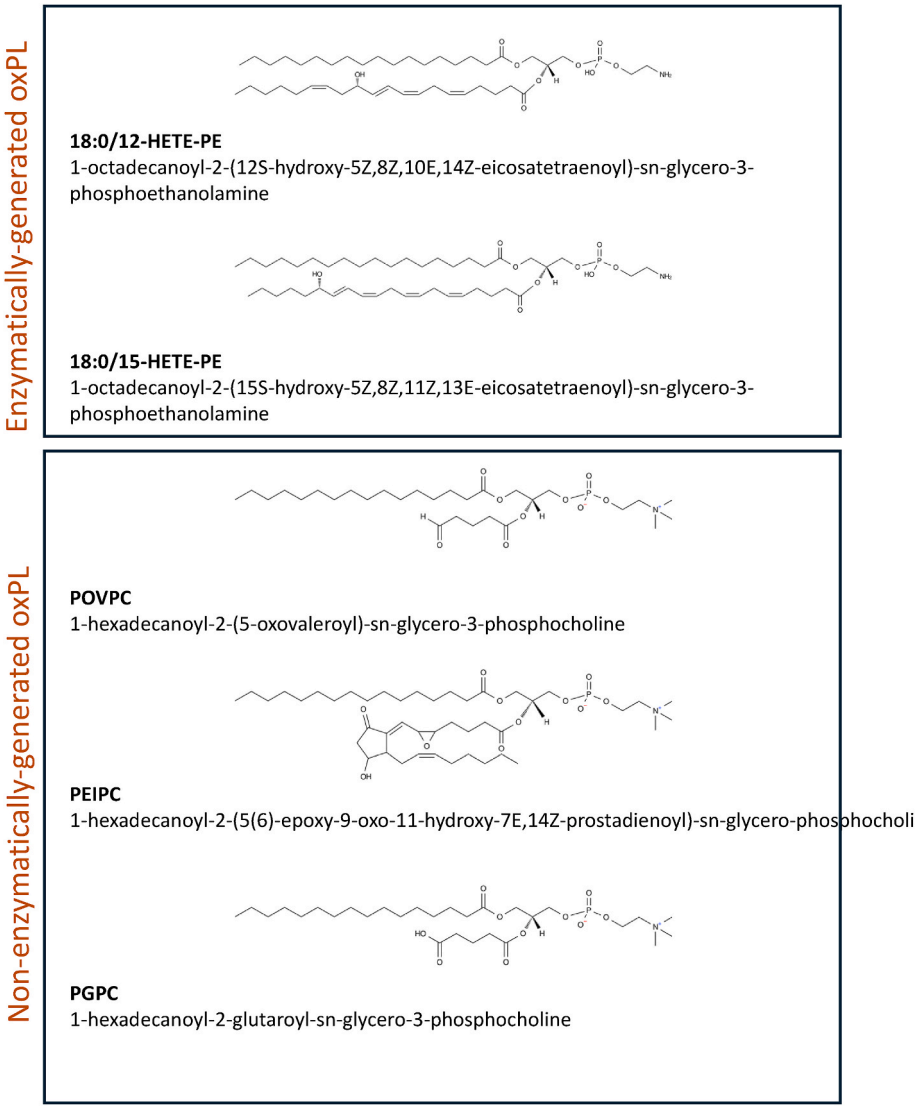

Fig. 1. Examples of the most well-known abundant oxPL structures showing both enzymatically and non-enzymatically generated species.

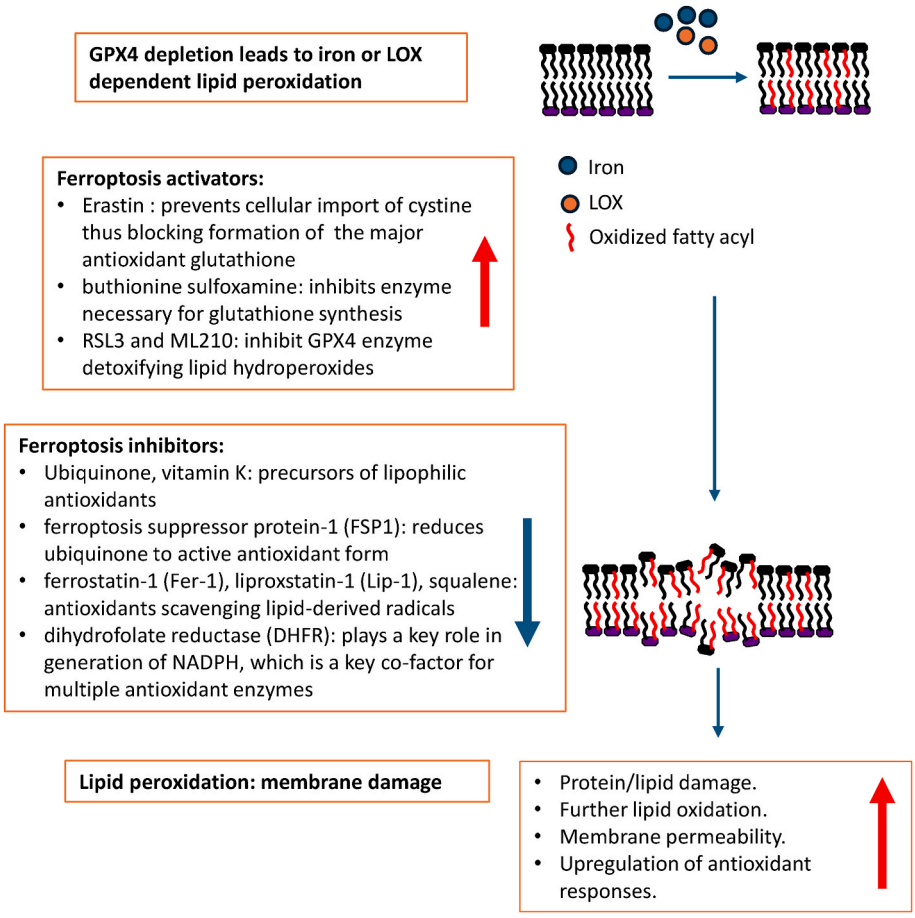

Fig. 2. Ferroptosis results from iron-dependent lipid peroxidation following inactivation of GPX4. This leads to membrane breakdown and cell death.

to find pharmacological agents to regulate ferroptosis has become a major focus for drug development. Many new proteins and small molecule inhibitors or activators were uncovered, both endogenous and pharmacological. These include small molecules such as erastin [26] (blocks the cystine-glutamate antiporter system x_c^-), buthionine sulfoxamine (glutathione synthesis inhibitor) [28], RSL3 and ML210 (both GPX4) [29] all of which can trigger ferroptosis and have been extensively used in model systems. These firmly establish the protective roles of thiol reductive pathways in preventing ferroptosis in healthy tissues. Separate to this, the radical trapping activities of ubiquinone and vitamin K, promoted by the NAD(P)H oxidizing enzyme, ferroptosis suppressor protein-1 (FSP1) provide a central mechanism for preventing mitochondrial ROS triggered lipid peroxidation [30–32]. This can be mimicked using pharmacological radical trapping agents, such as the synthetic agents ferrostatin-1 (Fer-1) and liproxstatin-1 (Lip-1), both identified through high-throughput screening approaches [26,33], as well as squalene, generated through the cholesterol synthesis pathway at higher levels when cholesterol levels are raised [34]. Another important antioxidant pathway that dampens ferroptosis involves redox cycling of biopterin by dihydrofolate reductase (DHFR) [35,36].

According to the current model, ferroptosis is due to unrestrained PL peroxidation which eventually leads to membrane rupture [37,38]. Exactly what form of peroxidation is involved has not been extensively clarified and many studies use non-specific outputs such as malondialdehyde or 4-hydroxynonenal as indicators. The application of liquid chromatography-tandem mass spectrometry (LC/MS/MS) to the characterization of the complex mixture of resulting lipid oxidation products has identified the most abundant PE and phosphatidylcholine (PC) to be targets [30,39,40], however specific positional isomers or enantiomers were not defined. A role for 15- or 12/15-lipoxygenase (15-LOX, *Alox15*) in triggering ferroptosis was originally proposed [27,40], however studies on mice lacking *Alox15* ruled out a role for enzymatic oxidation, for example in driving embryonic lethality of GPX4 deficiency [41]. Furthermore, LC/MS/MS showed complex mixtures of isomers consistent with non-enzymatic oxidation [30]. The pattern of products may vary depending on the cell and tissue, related to the underlying PL composition with higher levels of longer chain PUFA being present in brain, and plasmalogens being enriched in both brain tissue and immune cells. Whether a ferroptosis signature exists for particular tissues is not yet known. Nowadays, ferroptosis is mainly considered to be a non-enzymatic cell death process that could in some situations be sensitized by the actions of LOXs [42]. However, how initiation happens isn't fully clear since a seeding hydroperoxide as well as free metals would always be required. Considering that identifying ferroptosis and clearly distinguishing it from other forms of cell damage can be technically difficult, a recent recommendation was published providing guidance for researchers in the field [43].

Many recent studies consider that ferroptosis could be therapeutically harnessed, with most focusing on cancer [8,44–50], neurodegeneration [51], and renal failure [52,53]. Relating to cancer, the premise is that selective activation of ferroptosis, could trigger cell death in the tumor without impacting the host, particularly in the case of persister and de-differentiated cancers. Early work conducted in sarcoma cells identified selective cell death inducers that acted through glutathione depletion [54] and was later recognized as ferroptosis. Several cancers are highly sensitive to ferroptosis inducers, including triple-negative breast cancers and tumors that express FSP1 which include non-small cell lung cancer and pancreatic ductal adenocarcinoma, making them highly relevant targets (reviewed in Ref. [55]). Another therapeutic possibility could be to induce the immune system to

activate immunogenic cell death, sensitized by the presence of ferroptotic cancer cells [56], although ferroptosis-derived damage-associated molecular patterns (DAMP) (e.g. oxPL) don't appear to induce an anti-tumor immune response [57]. Identifying mechanisms to selectively sensitize cancer cells may be needed, for example through altering extracellular lipid supply, to restrict their accumulation in susceptible PL pools, as recently shown in vitro [58]. As summarized in a recent extensive review [55], inducing ferroptosis presents an exciting possibility for cancer therapeutics, however the challenge remains selectively targeting the tumor itself, for example via GPX4 inhibition. Drugs that sense cancer specific markers, for example, PROTACs or nanoparticles could be used, but additional studies are needed, and clinical studies or trials have not yet been initiated in this area. See also this review for studies on the role of ferroptosis in particular cancers, e.g. lung, liver and hematological malignancies [59].

Several neurodegenerative diseases have been associated with iron elevation in the brain (summarized here [59]) suggesting common mechanisms that involve ferroptosis, although research into this is still evolving. In support of a role for iron in the pathology of Alzheimer's Disease, ferritin is elevated in cerebrospinal fluid of APOE4 carriers, and predicts outcome [60], while knockout of presenilin 1/2, or expression of mutant forms, reduce GPX4 expression in vitro [61]. In Parkinson's disease, a similar increase in ferritin along with reduced GSH was observed decades ago [62], while recent mouse studies found that overexpression of mutated α-synuclein leads to a Parkinsonian phenotype that can be reduced using ferroptosis inhibitors [63]. A mechanism was proposed where α-synuclein expression is associated with altered levels of ferroptosis sensitive ether PL, although the biochemical process by which this occurs isn't yet clear and further studies are needed to substantiate this hypothesis [64]. So far, therapeutic strategies targeting elevated iron have not proven to be effective [59]. Another challenge is that studies measuring oxPL in brain tissue post-mortem may not accurately reflect the situation in vivo, including where long-term storage may lead to elevations in oxPL levels.

In summary, research into key mechanisms of iron/oxPL-driven cell death in human disease has undergone a huge resurgence in interest in recent years. Discoveries of the many new biochemical pathways and processes involved are leading to novel targets being identified that move beyond simple radical trapping antioxidants, providing new opportunities to selectively and specifically target this pathway. Combined with new generation therapeutic strategies such as CRISPR/Cas9, PROTAC and nanoparticle drug delivery, there is reason to be optimistic that activation or inhibition of ferroptosis may have wide applicability to preventing human diseases of aging in the future.

3. Innate and adaptive immunity: an update on formation, signaling and therapeutic opportunities for oxPL

Interest in delineating the role(s) of PL oxidation in immunity surged in the 1990's following the identification of OxPAPC, POVPC, PGPC and PEIPC as pro-atherogenic components of minimally oxidized LDL, capable of triggering inflammation in the vascular wall [2]. Since then, a large body of evidence demonstrated that exogenous oxPL induce endothelial expression of leukocyte adhesion molecules, stimulate adhesion of monocytes, and induce pro-inflammatory cytokines and chemokines in various cell types [65]. Novel insights into the mechanisms of generation and signaling actions of oxPL in immunity and inflammation continue to be revealed, and the field is now looking

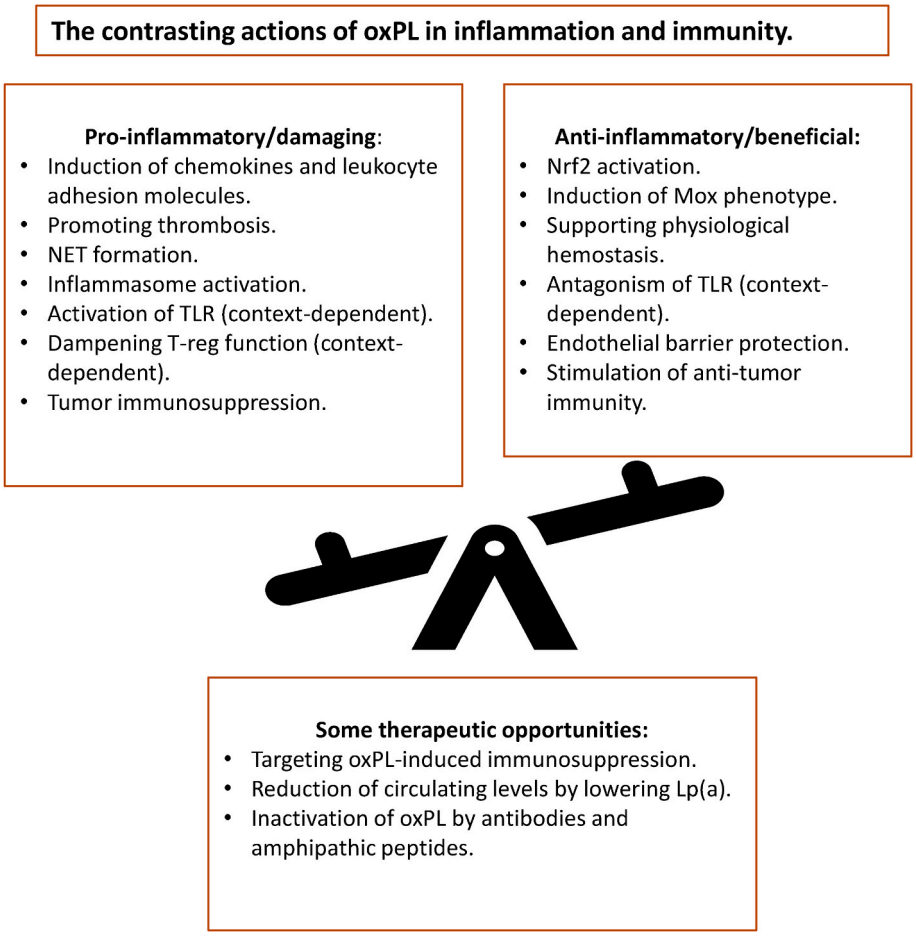

Fig. 3. A summary of contrasting actions of oxPL in inflammation and immunity, highlighting recent therapeutic avenues.

towards clinical translation, as outlined below (Fig. 3). We summarize the background in the next paragraphs, and follow this with expanded text on current state-of-the-art of the role of oxPL in immunity.

Enzymatically-oxidized PL (eoxPL) are generated in circulating blood cells via cyclooxygenase (COX) and lipoxygenase (LOX)-dependent generation of oxylipins, followed by their esterification by Lands cycle enzymes into PL [15]. In vitro and in vivo studies show that they can promote phosphatidylserine-dependent coagulation in blood cell membranes (summarized here [24]). This was recently found to be altered in atherosclerotic cardiovascular disease (ASCVD) and rheumatoid arthritis (RA), directly contributing to the elevated thrombotic risk observed in these conditions [66,67]. In ASCVD, a significant increase in the most abundant platelet 12-LOX-derived eoxPL was found along with reduced generation of related isomers made by COX-1, due to aspirin supplementation [66]. This highlights a need to consider the impact of common drug therapies on oxPL generation and their bioactivity. In antigen induced arthritis (AIA) in mice, the elevation of eoxPL was driven by interleukin-6 signaling, and contributed to an immune response evidenced by higher levels of serum anti-eoxPL IgG immunoreactivity [67]. In platelets, acylation of 12-HETE to form diacyl-PE eoxPL is catalyzed by lysophosphatidylcholine acyltransferase 3 (LPCAT3), but this enzyme is not involved in formation of other eoxPL including those generated by COX-1 [66]. This indicates that the formation of eoxPL is not only regulated by oxygenases but also specific Lands cycle enzymes. The isoforms involved in forming other eoxPL in platelets or in white cells are so far not characterized.

A major step forward was recent demonstration of pathogenic roles for endogenously generated oxPLs. In mouse models, blocking antibodies directed against oxPC reduce chronic and acute inflammatory diseases including atherosclerosis [68], liver disease [69,70] and ischemia-reperfusion injury [71]. In humans, recent observations of a clinical association of Lp(a) with cardiovascular disease supports a pathogenic role for oxPL [72]. In human plasma, oxPL are primarily bound to Lp(a) rather than other blood lipoprotein classes making this their major circulating carrier [73,74]. Due to this, it has been proposed that the well-known pro-inflammatory and pro-thrombotic vascular actions of Lp(a) are mediated by oxPL thus providing a mechanistic link to cardiovascular disease risk [75].

Pro-inflammatory responses to oxPL are complex and context-dependent, being triggered by multiple receptors from different families, including endocytotic, pattern-recognition, G-protein-coupled receptors and ligand-activated ion channels [65]. Recently, oxPAPC and its components KOdiA-PC, POVPC, PGPC, and PEIPC were shown to activate both canonical and noncanonical inflammasomes producing IL-1β [76–78]. In contrast to inflammatory cytokines, oxPAPC generally signals independently from the major pro-inflammatory transcription factor NFκB, instead using alternative signaling pathways, such as unfolded protein response and electrophilic stress response [65,79,80]. Unlike inflammatory cytokines, pathogen-associated molecular patterns (PAMPs) and interleukins, which induce conventional M1 and M2 phenotypes in macrophages, oxPAPC induces Mox differentiation characterized by high Nrf2-dependent gene expression. Here, the Mox phenotype is characterized by reduced phagocytosis and chemotaxis, as well as a specific gene expression pattern [81,82]. Mox macrophages adopt a particular metabolic state characterized by reduced respiration and an enhanced pentose phosphate pathway, which is required for cellular antioxidant defence [83]. Metabolic changes are also seen during pro-inflammatory activation of arterial endothelial cells by Lp(*a*)-bound oxPL which activates glycolysis, while inhibition of glycolysis by a small molecule PFK158 reduces inflammatory gene expression [75]. Here, activation of glycolysis in endothelial cells by oxPAPC may in part be mediated via Nrf2-dependent signaling [80]. Along with this, oxPL are increasingly recognized as pathologically relevant pro-inflammatory DAMP, molecules generated or released during stress and disease to alert the immune system [76]. The first use of the term in relation to oxPL was in 2011, when Miller et al. proposed that oxidation specific epitopes including oxPL should be considered DAMPs since they mediate a variety of relevant immune actions [84].

Our knowledge of the impact of oxPL on innate immunity has significantly increased in recent years, in particular, several studies have identified how these lipids can act cooperatively with other inflammatory agents to regulate leukocyte function. For example, oxPL can modulate metabolic profiles and inflammatory responses of macrophages co-activated by PAMPs and cytokines. Here, co-treatment of lipopolysaccharide (LPS)-primed macrophages with oxPAPC or PEIPC stimulates glycolysis and boosts oxidative phosphorylation and ATP-coupled respiration, in contrast to the effect of LPS alone. In addition, the transition to a hypermetabolic state was accompanied by strongly increased interleukin-1β (IL-1β) synthesis and secretion induced by PEIPC, POVPC and PGPC [85]. Similarly, enhanced chemokine production was observed after co-treatment of endothelial or monocytic cells with oxPAPC, when combined with TNFα or IL-1β [86]. OxPL activate neutrophils to release neutrophil extracellular traps (NET), an important effector reaction for these cells. Recently POVPC, PGPC, and PAzPC-induced NET formation was found to be inhibited by HDL [87] and plant flavonoids [88]. Thus, these lipids directly activate innate immune cells through a variety of receptors, signaling and effector mechanisms, in some cases, acting in tandem with other inflammatory agonists.

Several recent studies extend our knowledge of the regulation of adaptive immunity by oxPL. For example, these lipids induce "hyperactivated" dendritic cells (DCs) that secrete IL-1β but don't undergo pyroptosis. Because of their greater survival time, more IL-1β is produced thus leading to stronger stimulation of T cells [76]. As a result, conventional DC1 cells co-treated with LPS and OxPAPC or PGPC were proposed to stimulate long-lasting anti-tumor immunity [89].

In contrast to their pro-inflammatory activities, oxPL were recently reported to be immunosuppressive. As one example, intra-tumor accumulation of oxidized phosphatidylcholines impaired the functions of $CD8^+$ tumor-infiltrating lymphocytes [90]. Here, functionality of cells was rescued by knockdown of CD36, a scavenger receptor known to bind oxPL on the surface of endocytosed oxLDL suggesting that accumulation of the lipids in adaptive immune cells in lipid-rich tumor microenvironments is immunosuppressive. As further evidence of immunosuppression, accumulation of oxidized lipids during ferroptosis inhibits T cell function and promotes tumor growth; an effect that could be reproduced by hydroperoxides of phosphatidylcholine and phosphatidylethanolamine [8]. In addition, OxPAPC, POVPC or PGPC induced by antitumor treatment can recruit myeloid derived suppressor cells, inhibiting immune responses and supporting tumor growth [91]. OxPL also impair Treg phenotype and function. Here, treatment with exogenous oxPAPC induced a Th1 shift in Treg and reduced their functional capacity to counteract atherosclerosis after adoptive transfer [92].

Accumulating evidence for the pathological pro-inflammatory activity of oxPLs make these lipids a promising drug target. Several approaches are currently being trialed as outlined below. The most advanced is based on lowering blood levels of Lp(a), which removes circulating oxPLs bound to this lipoprotein [73]. Drugs reducing Lp(a) have been recently discussed here [93]. At the time of writing, several siRNA based drugs that target Lp(a) formation, including zerlasiran (NCT05537571) and muvalaplin (NCT05778864) have successfully finished phase II, while pelacarsen (NCT04023552) and olpasiran (NCT05581303) are in clinical phase III studies on cardiovascular events as listed on ClinicalTrials.gov. Results from these trials should be available from 2026. A second approach is based on antibodies that bind and neutralize oxPL. This has been found to be effective in mouse studies but has not yet been tested in humans [68–71]. In addition to passive immunization or transgenic overexpression of antibodies, vaccination with *Streptococcus pneumonia*, which raises antibodies that cross-react with oxPC, was shown many years ago to inhibit the proatherogenic effects of oxPL, reducing atherosclerosis in mice [94]. Taking this further, studies on the effect of a pneumococcal vaccine are currently

being undertaken in humans [95]. A third strategy to reduce or neutralize oxPL is based on administration of amphipathic peptides, for example, ApoA-I mimetic peptides which are known to inhibit their effects. In this regard, peptide 6F produced in transgenic tomatoes was recently shown to reduce accumulation of oxPL in mouse jejunum, following feeding with western diet [96]. Last, pro-inflammatory signaling mechanisms induced by oxPL could be targeted. This has been recently tested using an amphipathic helical peptide L37pA that targets scavenger receptors B and CD36 and was shown to inhibit inflammatory signaling and normalize endothelial permeability induced by truncated oxPL [97]. In addition, the induction of pro-inflammatory chemokines by oxPL can be inhibited by drugs supporting proteostasis, such as inducers of heat shock proteins and chemical chaperones [98].

4. Concluding statements

As summarized in this review, research into the biology and pathophysiology of oxPL continues at pace, with recent studies revealing new mechanisms of their formation, biological mechanisms of action and novel potential therapeutic approaches for vascular disease, cancer and neurodegeneration. As some oxPL are generated by regulated enzymatic pathways and required for homeostatic functions including hemostasis and innate immunity, a challenge will be to ensure that preventing their bioactivities doesn't lead to unintended consequences such as bleeding or infection risk. Beyond this, there are several areas needing further research. First, the origin of many oxPL in various disease states is still unclear, including in ferroptosis and atherosclerosis. Understanding this is key to identifying small molecules or other approaches that prevent their formation. Exactly how oxPL are removed from membranes has not yet been determined, but is likely to involve Lands cycle enzymes, and represents another area that could be therapeutically relevant. Last, how formation of eoxPL regulates oxylipin bioactivity is not well understood, but could involve either removal of oxylipins through esterification to form oxPL, or release of oxylipins from oxPL using phospholipases triggering signaling. Answering these questions in the coming years will generate a far deeper understanding of the biology and biochemistry of these intriguing lipids and hopefully lead to novel therapeutic strategies for common human diseases, including inflammation and cancer.

CRediT authorship contribution statement

Valerie B. O'Donnell: Writing – review & editing, Writing – original draft, Conceptualization. **Valery Bochkov:** Writing – review & editing, Writing – original draft.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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