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Research Summary of ZLN005 (PGC-1α Activator) Studies

Overview of ZLN005

ZLN005 is a small-molecule activator of peroxisome-proliferator-activated receptor gamma co-activator-1 alpha (PGC-1α). PGC-1α is a transcriptional co-activator that promotes mitochondrial biogenesis, oxidative metabolism and anti-oxidant defenses. High-throughput screening identified ZLN005 as a compound that increases PGC-1α transcription in skeletal muscle cells [1]. Unlike many earlier ligands, ZLN005 crosses membranes and is orally bioavailable. The compound has since been used in diverse models to test whether boosting PGC-1α improves metabolic disorders, mitochondrial dysfunction or inflammatory injury. The following sections summarize all known PubMed studies referencing ZLN005.

Metabolic and Diabetic Models

Discovery and metabolic effects in skeletal muscle and db/db mice (2013)

- **Models/intervention –** High-throughput screening of L6 myotubes identified ZLN005 as a non-toxic compound that increased PGC-1α promoter activity. In L6 myotubes ZLN005 dose-dependently increased PGC-1α mRNA, GLUT4 and mitochondrial genes, and stimulated glucose uptake and palmitic-acid oxidation [1]. The stimulation required a MEF2 binding site and coincided with AMPK activation; pharmacologic or genetic inhibition of AMPK blunted the effect [2].
- **Metabolic outcomes –** Chronic oral dosing (30 mg kg^{-1}) in diabetic db/db mice for four weeks improved random and fasting glucose, glucose tolerance, insulin sensitivity, and decreased plasma triglycerides and non-esterified fatty acids while not changing body weight [3]. Respiratory exchange ratio decreased, indicating increased fat oxidation. Skeletal muscle PGC-1α expression and downstream genes increased whereas hepatic PGC-1α decreased, leading to down-regulation of gluconeogenic genes [4].
- **Conclusion –** ZLN005 enhances skeletal-muscle glucose uptake and fatty-acid oxidation via AMPK-dependent PGC-1α induction and improves glycemic control in diabetic mice [4].

High-glucose cardiomyocyte injury (2016)

- **Models/intervention –** Neonatal mouse cardiomyocytes exposed to high glucose (33 mM). ZLN005 was co-incubated for 24 h and SIRT1 inhibitor EX527 was used to test mechanism.
- **Key findings –** High glucose induced oxidative injury and suppressed autophagy; ZLN005 restored cell viability, reduced apoptosis and increased autophagy proteins (ATG5, beclin-1, LC3 II/I) [5]. ZLN005 also elevated SIRT1 expression, and SIRT1 inhibition blunted these protective effects [5].
- **Conclusion –** ZLN005 protects cardiomyocytes from high-glucose cytotoxicity by upregulating SIRT1 and autophagy [5].

Sulforaphane-Urolithin-A comparison in muscle cells (2024)

- **Models/intervention –** C2C12 myotubes treated with sulforaphane, urolithin A or ZLN005 (10–20 μM) for 4–48 h to examine antioxidant capacity, mitophagy and mitochondrial biogenesis.
- **Key findings –** ZLN005 did not increase nuclear PGC-1α at 24 or 48 h but transiently activated AMPK by 4 h [6]. It increased TFAM promoter activity (~40 % increase in luciferase reporter) and modestly enhanced mitochondrial respiration at 24 h but these effects dissipated by 48 h [7].
- **Conclusion –** While ZLN005 modestly increased AMPK activity and TFAM promoter activity, this study suggested it is a weak activator of mitochondrial biogenesis in myotubes [7].

Cardiovascular and Cardiac Injury

Cardiomyocyte maturation (2020)

- **Models/intervention –** Human embryonic stem cell–derived cardiomyocytes (hESC-CMs) were treated with 10 μM ZLN005 for 48 h during days 10–12 of differentiation.
- **Key findings –** ZLN005 increased PGC-1α mRNA and protein (~1.7-fold) [8] and upregulated mitochondrial biogenesis and oxidative phosphorylation genes. It increased mitochondrial number, DNA copy number and oxygen consumption [8]. Treated cardiomyocytes exhibited longer sarcomeres, improved connexin-43 expression and more mature action potentials and calcium transients [9].
- **Conclusion –** ZLN005 promotes metabolic and structural maturation of hESC-derived cardiomyocytes by enhancing PGC-1α–driven mitochondrial biogenesis [8].

Ischemia-induced neuronal injury (2017; tMCAO and OGD)

- **Models/intervention –** Rats underwent transient middle cerebral artery occlusion (tMCAO); ZLN005 (2.5 mg kg^{-1}) was administered intravenously 2 h, 4 h or 6 h after ischemia. Well-differentiated PC12 cells were subjected to oxygen–glucose deprivation (OGD) and treated with 1–5 μM ZLN005.
- **Key findings –** ZLN005 significantly reduced brain infarct volume and improved neurological deficit scores when given up to 6 h post-stroke [10]. It increased cortical PGC-1α and its targets cytochrome c, COX5b and AOX [11] and upregulated antioxidant genes (SOD1, HO-1) and improved SOD activity [12]. In PC12 cells, ZLN005 pretreatment protected against OGD-induced death, increased PGC-1α and cytochrome c mRNA and protein, and upregulated SOD1 and HO-1 [13].
- **Conclusion –** ZLN005 has neuroprotective effects after ischemic injury by activating PGC-1α and antioxidant pathways [14].

Myocardial infarction – deleterious effect (2025)

- **Models/intervention –** Mice underwent permanent coronary artery occlusion (myocardial infarction). ZLN005 was administered for 14 days to increase PGC-1α. Primary cardiomyocytes under oxygen–glucose deprivation were also treated.
- **Key findings –** Following MI, PGC-1α levels declined; chronic ZLN005 treatment increased PGC-1α but significantly increased the Bax/Bcl-2 ratio (indicator of apoptosis) and decreased cardiac function on ECG [15]. In vitro, ZLN005 decreased mitochondrial function and ATP production in OGD-treated cardiomyocytes [15].

- **Conclusion –** Sustained upregulation of PGC-1α by ZLN005 under ischemic stress exacerbated myocardial injury and impaired heart function [15], highlighting that context and timing of PGC-1α activation are critical.

Kidney and Liver Injury

Unilateral ureteral obstruction (renal fibrosis) (2022)

- **Models/intervention –** Mice with unilateral ureteral obstruction (UUO) were treated orally with ZLN005 (10 mg kg^{-1} per day) for seven days. In vitro, renal tubular epithelial cells (TECs) were exposed to TGF-β1.
- **Key findings –** ZLN005 reduced renal fibrosis markers (fibronectin, α-SMA, COL1A1) and improved histopathology to a degree comparable to fenofibrate [16]. It decreased inflammatory cell infiltration and down-regulated Il-1β, Il-6, Tnf-α and iNOS mRNAs [17]. ZLN005 increased PGC-1α and TFAM expression and enhanced fatty-acid oxidation gene expression while reducing lipogenic genes [18]. In vitro, ZLN005 reversed TGF-β1-induced fibrotic phenotype, restored mitochondrial membrane potential and ATP levels, and increased mtDNA copy number [19].
- **Conclusion –** ZLN005 ameliorates renal fibrosis by restoring PGC-1α-mediated mitochondrial homeostasis and reducing inflammation [16].

Renal ischemia–reperfusion injury (2021)

- **Models/intervention –** Rats underwent renal ischemia (45 min) followed by reperfusion; ZLN005 was given intraperitoneally at 25 mg kg^{-1}. In vitro, HK2 renal cells were subjected to hypoxia/reoxygenation. CPT-1 inhibition (etomoxir) or knockdown was used.
- **Key findings –** Ischemia reduced cortical PGC-1α and CPT-1α; ZLN005 increased both and reduced tubular injury, oxidative stress (H_2O_2, MDA) and apoptosis [20]. It increased anti-apoptotic Bcl-2/Bax ratio and reduced cleaved caspase-3; these benefits were abolished by CPT-1 inhibition [21]. In HK2 cells ZLN005 improved cell viability, reduced ROS and lipid droplets, and increased PGC-1α/CPT-1α in a dose-dependent manner; these effects were negated when CPT-1α was inhibited [22].
- **Conclusion –** ZLN005 protects kidneys from ischemia–reperfusion injury by restoring PGC-1α-mediated mitochondrial fatty-acid oxidation [20].

Ischemia-reperfusion liver injury and metastasis (2024)

- **Models/intervention –** Mice received intraperitoneal ZLN005 (10 mg kg^{-1}) for three days before partial hepatic ischemia (90 min) followed by reperfusion and injection of MC38 colon cancer cells.
- **Key findings –** ZLN005 pretreatment reduced serum ALT, AST and LDH, decreased hepatic necrosis, ROS and apoptosis [23]. It decreased neutrophil and macrophage infiltration and NET formation, and reduced inflammatory cytokine production. ZLN005 increased mitochondrial mass (COXIV, TOMM20) and mtDNA and upregulated PGC-1α/NRF1/TFAM gene expression [24]. It decreased hepatic tumor burden and increased intratumoral cytotoxic T cells [24].
- **Conclusion –** Pretreatment with ZLN005 protects against liver ischemia–reperfusion injury, preserves mitochondrial biogenesis and mitigates metastasis [23].

Lung and Respiratory Diseases

Alveolar epithelial cell aging (2023)

- **Models/intervention –** In bleomycin-treated mice and idiopathic pulmonary fibrosis (IPF) patients, alveolar epithelial cells (AECs) showed reduced PGC-1α and increased senescence marker p21^WAF1. A549 AECs were exposed to H_2O_2 to induce senescence; ZLN005 pretreatment was added.
- **Key findings –** ZLN005 increased PGC-1α and NRF-1 expression, decreased p21^WAF1 and improved mitochondrial morphology and function (increased ATP, membrane potential and oxygen consumption, decreased ROS) [25] . Knockdown of PGC-1α abolished these protective effects [26] .
- **Conclusion –** ZLN005 protects alveolar epithelial cells from oxidative stress–induced aging by enhancing PGC-1α and mitochondrial function [25] .

Asthma model (2024)

- **Models/intervention –** Ovalbumin-induced allergic asthma mice received intraperitoneal ZLN005 (5 mg kg^{-1}).
- **Key findings –** ZLN005 reduced inflammatory cell infiltration in bronchoalveolar lavage fluid and lung tissue, decreased serum IgE and OVA-specific IgE and lowered Th2 cytokines IL-4, IL-5 and IL-13 [27] . It suppressed NF-κB-p65 activation and NLRP3 inflammasome activity, reducing IL-1β and IL-18 [28] .
- **Conclusion –** ZLN005 exhibits anti-inflammatory and anti-asthmatic effects by inhibiting NF-κB/NLRP3 signaling [27] .

Migrasome secretion and pulmonary fibrosis (2023)

- **Models/intervention –** LPS-challenged human lung fibroblasts. ZLN005 was used to elevate PGC-1α.
- **Key findings –** LPS induced PGC-1α reduction, increased α-SMA and promoted migrasome formation with mitochondrial DNA release. ZLN005 increased PGC-1α, reduced α-SMA and migrasome formation, and increased mitochondrial mass and mtDNA copy number [29] .
- **Conclusion –** PGC-1α activation by ZLN005 suppresses migrasome-mediated pulmonary fibrosis mechanisms [29] .

Rhinovirus infection in bronchial epithelial cells (2024)

- **Models/intervention –** Human bronchial epithelial cells were infected with human rhinovirus C15 (HRV-C15). ZLN005 or oligomycin A (positive control) was added.
- **Key findings –** HRV-C15 caused barrier loss and replication in epithelial cells. ZLN005 improved barrier function at 12 h post-infection and decreased viral replication; at 24 h, barrier protection was weaker but still present [30] .
- **Conclusion –** PGC-1α activation by ZLN005 partially protects airway epithelial barrier integrity and suppresses viral replication during rhinovirus infection [30] .

Allergic rhinitis – α-asarone study (2023)

- **Models/intervention –** Allergic rhinitis model investigating α-asarone's SIRT1/PGC-1α pathway. ZLN005 was co-administered to confirm pathway involvement.

- **Key findings –** α-asarone reduced mitochondrial ROS, restored mitochondrial function and barrier integrity and alleviated nasal inflammation. ZLN005 produced similar PGC-1α activation, and inhibition of SIRT1/PGC-1α abolished the benefits [31].
- **Conclusion –** ZLN005 served as a positive control demonstrating that PGC-1α activation reduces mitochondrial ROS and inflammation in allergic rhinitis [31].

Traumatic brain injury (TBI) – inflammation-induced mitochondrial dysfunction (2024)

- **Models/intervention –** Human TBI brain samples were analyzed for transcriptomic changes. Mouse hippocampal HT-22 cells were treated with TNF-α with or without ZLN005; a mouse TBI model received ZLN005.
- **Key findings –** TBI patients exhibited down-regulation of mitochondrial oxidative-phosphorylation genes and up-regulation of inflammatory pathways. TNF-α impaired mitochondrial respiration and induced oxidative stress and apoptosis in HT-22 cells. ZLN005 restored OXPHOS gene expression, improved mitochondrial function and reduced apoptosis [32]. In the TBI mouse model, ZLN005 alleviated neuronal death, preserved mitochondrial integrity and improved cognitive function [32].
- **Conclusion –** Activation of PGC-1α by ZLN005 counteracts inflammation-induced mitochondrial dysfunction and neuronal damage after traumatic brain injury [32].

Neurological and Pain-Related Disorders

Perioperative neurocognitive disorders (PND) (2024)

- **Models/intervention –** A mouse model of perioperative neurocognitive disorder was created using anesthetic surgery. Animals received intraperitoneal ZLN005 (5 or 7.5 mg kg^{-1}) three days before and after surgery.
- **Key findings –** Surgery decreased hippocampal PGC-1α and NRF-1 expression and increased inflammatory cytokines IL-6 and IL-1β. ZLN005 restored PGC-1α/NRF-1 expression, increased mitochondrial proteins (Atp5d, Atp5k, Cox5a), improved ATP production and decreased ROS [33]. It reduced IL-1β and IL-6 and improved cognitive performance [34].
- **Conclusion –** ZLN005 alleviates perioperative neurocognitive disorders by enhancing mitochondrial biogenesis and reducing neuroinflammation [33].

Neuropathic pain (spared nerve injury and chronic constriction injury) (2022–2024)

- **Models/intervention –** Rats with spared-nerve injury (SNI) or chronic constriction injury (CCI) received intrathecal injections of ZLN005 (50–100 μg).
- **Key findings –** ZLN005 increased paw withdrawal threshold, reversed mechanical allodynia and delayed the onset of pain when administered early [35]. Its analgesic effect was negated by the PGC-1α inhibitor SR-18292. Mitochondrial biogenesis markers (mtDNA, PGC-1α, NRF-1, TFAM) were increased and neuroinflammation suppressed (reduced IL-1β and IL-18) [36]. A review summarized that intrathecal ZLN005 reduced ROS and alleviated pain in CCI mice and increased paw withdrawal threshold while reducing ROS in SNI rats.
- **Conclusion –** PGC-1α activation by ZLN005 attenuates neuropathic pain via mitochondrial biogenesis and reduced oxidative stress and neuroinflammation [36].

Infectious Diseases and Immunity

Sepsis and polymicrobial infection (2024)

- **Models/intervention –** Mice underwent cecal ligation and puncture (CLP) to induce polymicrobial sepsis. ZLN005 was administered intraperitoneally (10 mg kg^{-1}) at 2 h post-surgery.
- **Key findings –** ZLN005 significantly improved survival, reduced circulating TNF-α, IL-1β and IL-6, and decreased lung and spleen damage [37]. It increased PGC-1α and TFAM expression in peritoneal macrophages and monocytes and improved bacterial clearance and mitochondrial reserve capacity [38] [39].
- **Conclusion –** ZLN005 augments macrophage mitochondrial biogenesis, suppresses cytokine storm and improves survival in sepsis [37].

Mycobacterial infection (MAC) (2023)

- **Models/intervention –** RAW 264.7 and THP-1 macrophages infected with Mycobacterium avium complex (MAC) were treated with ZLN005 or metformin.
- **Key findings –** MAC infection decreased PGC-1α and TFAM expression; ZLN005 restored both mRNA and protein levels [40]. ZLN005 enhanced phagocytic uptake and intracellular killing of MAC and improved mitochondrial membrane potential [41].
- **Conclusion –** ZLN005 enhances macrophage immune function against mycobacterial infection by boosting mitochondrial biogenesis and function [40].

Ocular Diseases

Retinal pigment epithelial (RPE) protection (2018)

- **Models/intervention –** ARPE-19 cells were treated with ZLN005. Oxidative stress was induced by H_2O_2, oxidized LDL or $NaIO_3$.
- **Key findings –** ZLN005 upregulated PGC-1α and downstream transcription factors, increased mitochondrial genes and improved basal and maximal respiration [42]. It protected cells from oxidative damage; this protection was lost when PGC-1α was silenced [42].
- **Conclusion –** ZLN005 promotes mitochondrial metabolism and protects RPE cells from oxidative stress through PGC-1α [42].

Proliferative diabetic retinopathy (PDR) study (2025)

- **Models/intervention –** Human retinal Müller glial cells were treated with high glucose or the hypoxia mimetic $CoCl_2$ and with ZLN005 (20 μM). Vitreous samples and epiretinal membranes from PDR patients were analyzed.
- **Key findings –** In Müller cells, ZLN005 increased PGC-1α protein and significantly upregulated VEGF secretion but not angiopoietin 2, MCP-1 or MMP-9 [43]. ZLN005 pretreatment attenuated ROS generation induced by high glucose or H_2O_2 [44]. Vitreous samples from PDR patients showed elevated PGC-1α and ERR-α levels.
- **Conclusion –** ZLN005 demonstrates that PGC-1α/ERR-α activation promotes angiogenic factors in retinal Müller cells and decreases ROS, suggesting a role in PDR pathogenesis [45].

Retinitis pigmentosa (RP) (2025 abstract)

- **Models/intervention –** In rd1 mice (a model of RP), ZLN005 was administered.
- **Key findings –** ZLN005 improved visual function, alleviated thinning of the outer nuclear layer and enhanced mitochondrial biogenesis via PGC-1α/NRF-1/TFAM pathway [46].
- **Conclusion –** PGC-1α activation with ZLN005 delays photoreceptor degeneration in RP [46].

Bone and Dental Cells

Cementoblast inflammatory response (OCCM-30 cells) (2022)

- **Models/intervention –** OCCM-30 cementoblasts exposed to TNF-α were treated with pathway inhibitors and ZLN005 to modulate PGC-1α.
- **Key findings –** TNF-α suppressed PGC-1α; blocking p38 MAPK restored its expression. ZLN005 upregulated PGC-1α and inhibited TNF-α-induced pro-inflammatory cytokines; PGC-1α silencing reversed the anti-inflammatory effect [47].
- **Conclusion –** ZLN005 attenuates TNF-α-induced inflammatory responses in cementoblasts by upregulating PGC-1α [47].

Vision and Eye Protection – Additional Preclinical Studies

Ischemia-induced neuronal/optic injury (2018 citation)

The PDR article's references noted a Cell Mol Neurobiol study in which ZLN005 ameliorated ischemia-induced neuronal injury in vitro and in vivo [48]. This study overlaps with the tMCAO/OGD model described under cardiovascular/neurological disorders.

Summary

Studies across multiple organ systems show that ZLN005 is a versatile small-molecule activator of PGC-1α. In metabolic and diabetic contexts, ZLN005 enhances skeletal-muscle glucose uptake and fatty-acid oxidation and improves glycemic control in db/db mice [4]. In kidney and liver injury, ZLN005 restores mitochondrial biogenesis, reduces inflammation and fibrosis and improves survival [16] [24]. Neuroprotective effects have been demonstrated in ischemic stroke, perioperative neurocognitive disorder, traumatic brain injury and neuropathic pain, where ZLN005 upregulates PGC-1α/NRF-1, enhances antioxidant defenses and mitigates neuroinflammation [14] [33] [36]. ZLN005 also improves immune function in sepsis and mycobacterial infection, attenuates asthma and fibrotic lung disease, and modulates angiogenic pathways in ocular disease. However, sustained PGC-1α activation during myocardial infarction worsened cardiac function, indicating that therapeutic context and dosing are critical [15]. Overall, the collective literature suggests that ZLN005 is a promising tool to probe PGC-1α biology and may hold therapeutic potential for diseases characterized by mitochondrial dysfunction and inflammation.

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