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Comprehensive summary of PubMed studies involving GW0742

Introduction

GW0742 is a synthetic agonist of the peroxisome-proliferator-activated receptor beta/delta (PPARβ/δ). Research in metabolic, cardiovascular, neurological, inflammatory and oncological fields has used GW0742 to probe PPARβ/δ biology, improve disease models, or identify potential adverse effects. The summaries below review every PubMed-indexed study that investigated or referenced GW0742 up to May 2025. Each entry lists the study's goal, model, key findings and conclusions.

Metabolic and endocrine studies

Antidiabetic and metabolic regulation

Study	Summary
Development of PPAR-δ agonist GW0742 as an antidiabetic drug (2015) [1]	Diabetic rats treated with GW0742 displayed improved glucose homeostasis and insulin sensitivity. GW0742 increased GLUT4 expression in muscle, reduced PEPCK levels in liver and improved hyperglycemia in streptozotocin-induced type 1 diabetic rats. The authors suggested that PPAR-δ activation by GW0742 may be useful for treating diabetes [1] .
Restoring lipid-induced endothelial dysfunction via CPT-1 up-regulation (2015) [2]	In palmitate- and high-fat-diet-induced endothelial dysfunction, GW0742 restored eNOS phosphorylation and reduced reactive oxygen species (ROS). The protective effect required up-regulation of carnitine palmitoyltransferase-1 (CPT-1); pharmacological or genetic inhibition of CPT-1 abolished GW0742's benefits [2] . Thus PPARβ/δ activation counters lipid-induced endothelial dysfunction by enhancing fatty-acid oxidation.
GW0742 restores endothelial function in type 1 diabetes (2017) [3]	Aortas from diabetic rats showed impaired acetylcholine-induced relaxation and decreased eNOS phosphorylation. GW0742 treatment increased eNOS phosphorylation and decreased NADPH oxidase-driven superoxide production and pre-pro-endothelin-1 expression. These effects were reversed by the PPARβ/δ antagonist GSK0660, indicating that GW0742 improves endothelial function in diabetes through PPARβ/δ activation [3] .

Study	Summary
Protection from lipopolysaccharide (LPS)-induced endothelial dysfunction requires UCP2 (2016) [4]	In a murine LPS-induced sepsis model, GW0742 prevented endothelial dysfunction by reducing ROS and NADPH oxidase expression. It restored nitric oxide (NO) production by up-regulating uncoupling protein-2 (UCP2) and lowering endoplasmic-reticulum stress. PPARβ/δ antagonism or UCP2 inhibition abolished these effects [4].
GW0742 attenuates lupus patient plasma–induced endothelial dysfunction (2017) [5]	Plasma from systemic lupus erythematosus patients induced ROS production, endoplasmic-reticulum stress and impaired NO production in endothelial cells. GW0742 restored NO bioavailability and reduced ROS and ER-stress markers. The rescue was prevented by PPARβ/δ antagonism or silencing, suggesting a therapeutic role for PPARβ/δ agonists in lupus-associated endothelial dysfunction [5].
Non-genomic redox modulation (2014) [6]	In cardiac myocytes, GW0742 rapidly attenuated ERK1/2 and Akt phosphorylation triggered by growth agonists. The effect was not reversed by GSK0660 but was inhibited by the phosphatase inhibitor vanadate. GW0742 prevented oxidation and inactivation of PTEN and abolished ROS generation, revealing a non-genomic mechanism whereby PPARβ/δ ligands reduce hypertrophic signaling [6].
Combined AMPK and PPARβ/δ activation improves endurance (2016) [7]	Mice trained for four weeks received GW0742 and the AMPK activator AICAR. The combination markedly increased running distance and endurance compared with either treatment alone. Metabolism shifted toward fatty-acid utilization, with increased plasma non-esterified fatty acids, glycogen sparing and induction of genes such as Pgc-1α, Cd36 and Lpl, demonstrating synergy between PPARβ/δ and AMPK activation [7].
GW0742 stimulates neuronal maturation (2016) [8]	In primary cortical neurons, GW0742 accelerated differentiation and neurite outgrowth. The PPARβ/δ antagonist GSK0660 abolished these effects, and PPARβ/δ activation triggered BDNF-dependent signaling. These findings show that PPARβ/δ agonism promotes neuronal differentiation [8].
Cardiac hypertrophy due to hyperglycemia (2018) [9]	Hyperglycemic H9c2 cardiomyocytes exhibited elevated intracellular Ca^{2+}, brain-type natriuretic peptide and β-myosin heavy-chain expression. GW0742 reduced these hypertrophic markers, decreased ROS and improved mitochondrial potential. The benefits were blocked by GSK0660, implicating PPARδ activation in the attenuation of hyperglycemia-induced cardiomyocyte hypertrophy [9].
Telmisartan and cardiac fibrosis (2016) [10]	Diabetic rats developed cardiac fibrosis with up-regulation of STAT3 and extracellular matrix proteins. Telmisartan improved fibrosis through the PPARδ/STAT3 pathway; the protective effect was diminished by the PPARδ antagonist GSK0660. Cardiomyocytes treated with GW0742 reproduced telmisartan's beneficial effects, supporting a role for PPARδ activation in telmisartan-mediated cardioprotection [10].

Study	Summary
Telmisartan reduces TNF-α–induced VEGF-C production via PPARδ (2014) [11]	Human proximal renal tubular epithelial cells produced VEGF-C in response to TNF-α via p38MAPK/HSP27 signaling. Telmisartan acted as a PPAR-δ activator and reduced TNF-α–induced VEGF-C production by decreasing p38MAPK phosphorylation. The results suggest that telmisartan lowers inflammation-induced lymphangiogenesis in a PPAR-δ-dependent manner [11].
Telmisartan activates endogenous PPAR-δ in mesangial cells (2014) [12]	Telmisartan increased PPAR-response-element luciferase activity and up-regulated fatty-acid-oxidation genes in human mesangial cells. These effects were blocked by the PPAR-δ antagonist GSK0660 but not by a PPAR-γ antagonist. Telmisartan also suppressed TGF-β1–induced PAI-1 and collagen IV expression and ERK phosphorylation, indicating that telmisartan prevents fibrotic changes via PPAR-δ activation [12].

Obesity and lipid metabolism

Study	Summary
High-fat diet–induced metabolic dysfunction (2018) [13]	In mice fed a high-fat diet, GW0742 improved glucose tolerance, decreased hepatic inflammation and shifted metabolism toward β-oxidation. It markedly reduced endoplasmic-reticulum stress markers and hepatic apoptosis, suggesting that PPARβ/δ activation may treat non-alcoholic fatty-liver disease [13].
GW0742 improves fatty-acid oxidation in Alzheimer's disease models (2019) [14]	Astrocytes derived from Alzheimer's disease patients and APP/PS1 mice showed impaired fatty-acid oxidation. GW0742 treatment increased carnitine palmitoyltransferase-1a, normalized fatty-acid oxidation and reversed memory deficits in APP/PS1 mice. It enhanced neurogenesis and neuronal differentiation without reducing amyloid-β load; thus PPARβ/δ agonism improved cognition by restoring metabolic function [14].
Lysophosphatidic acid/ PPARδ synergy in lipoprotein metabolism (2013)	In vascular smooth muscle cells, GW0742 up-regulated pyruvate dehydrogenase kinase-4 and down-regulated glucose transporter 1, thereby reducing glycolytic flux and VSMC proliferation. Ligand-coated stents releasing GW0742 decreased neointimal hyperplasia and stent thrombosis by inhibiting inflammatory gene expression and promoting endothelial regeneration [15].

Cardiovascular and inflammatory conditions

Vascular protection and restenosis

Study	Summary
GW0742 suppresses neointimal hyperplasia after arterial injury (2013) [16]	In mice subjected to wire-mediated arterial injury, GW0742 reduced intimal thickening by decreasing IL-6 production, suppressing proliferating cell nuclear antigen and enhancing endothelial marker CD31. It attenuated vascular smooth-muscle-cell proliferation while promoting endothelial regeneration [16].
PPARδ ligand-coated stents (2016) [15]	Rabbit arteries implanted with GW0742-coated stents displayed reduced neointima formation and luminal narrowing compared with control stents. GW0742 inhibited inflammatory gene expression, vascular smooth-muscle-cell proliferation and thrombocyte activation while promoting endothelial cell proliferation. The effects required PPAR-δ and involved metabolic reprogramming of smooth-muscle cells [15].
Right-heart hypertrophy and pulmonary hypertension (2010) [17]	In mice subjected to pulmonary artery banding, GW0742 treatment protected against right-ventricular hypertrophy and fibrosis. It normalized expression of genes such as angiopoietin-like 4, improved cardiac function and suggested that PPARβ/δ agonists could be beneficial in pulmonary hypertension [17].

Ischemia, sepsis and shock

Study	Summary
Gut ischemia/ reperfusion injury (2010) [18]	In mice subjected to superior mesenteric artery occlusion and reperfusion, GW0742 reduced mortality and histological gut damage. It lowered myeloperoxidase activity, pro-inflammatory cytokines, adhesion molecules, nitrotyrosine formation and NF-κB expression, and attenuated apoptosis. The results suggest that PPARβ/δ agonism helps resolve inflammation after ischemia/reperfusion [18].
Acute lung injury induced by carrageenan (2010) [19]	GW0742 administered before and after carrageenan challenge in mice reduced lung inflammation, histological damage, neutrophil infiltration, myeloperoxidase activity, lipid peroxidation and cytokine production. The study proposed that PPARβ/δ agonists like GW0742 may be useful for treating acute lung injury [19].
Bleomycin-induced lung injury (2010) [20]	GW0742 markedly decreased bleomycin-induced lung inflammation and apoptosis. It reduced cytokine production, leukocyte infiltration, NF-κB activation, oxidative stress and apoptosis markers, suggesting that GW0742 protects against pulmonary damage [20].

Study	Summary
Elastase-induced emphysema (2016) [21]	Pulmonary administration of GW0742 in an elastase-induced emphysema model increased surfactant proteins, shortened alveolar wall distance and improved respiratory function. GW0742 improved tissue elastance and forced expiratory volume/forced vital capacity ratio, indicating potential therapy for emphysema [21].
Ischemic stroke and intracerebral hemorrhage	In rodent models of middle cerebral artery occlusion, GW0742 pretreatment reduced infarct volume, edema, inflammatory cytokines (IL-1β, TNF-α), NF-κB, Bax and iNOS while increasing Bcl-2 expression, leading to improved neurological scores [22]. In collagenase-induced intracerebral hemorrhage, GW0742 lowered brain edema, blood-brain barrier leakage and neuronal apoptosis and improved neurological outcomes [23]. In neonatal germinal-matrix hemorrhage, GW0742 reduced mast-cell degranulation via a PPARβ/δ/CD300a/SHP1 pathway and improved neurobehavior [24].
Spinal cord injury (2010) [25]	In a mouse model of spinal cord trauma, GW0742 treatment reduced neutrophil infiltration, cytokine production, nitrosative stress and apoptosis, leading to improved motor recovery. Co-administration of the PPARβ/δ antagonist GSK0660 abolished the protective effects, demonstrating that PPARβ/δ activation mediates neuroprotection [25].
Acute pancreatitis (2012) [26]	Mice subjected to cerulein- or taurocholate-induced acute pancreatitis exhibited severe pancreatic edema, neutrophil infiltration and apoptosis. GW0742 treatment reduced serum amylase and lipase, decreased cytokines and adhesion molecules and lowered markers of apoptosis and NF-κB activation. The study concluded that GW0742 attenuates pancreatic damage in multiple models of pancreatitis [26].
Resuscitated porcine septic shock (2013) [27]	In pigs with pre-existing atherosclerosis subjected to long-term fecal peritonitis and resuscitation, GW0742 failed to improve hemodynamics, gas exchange, metabolism or organ function. The authors attributed the lack of benefit to low renal PPARβ/δ expression in this strain, suggesting that receptor down-regulation in co-morbidities limits GW0742 efficacy [27].
Delayed activation improves long-term survival in mouse sepsis (2018) [28]	Mice underwent cecal ligation and puncture to induce sepsis and received delayed GW0742 injections. GW0742 increased 28-day survival by 50 %, elevated interferon-γ and lowered monocyte chemoattractant protein-1, and reduced plasminogen activator inhibitor-1 while increasing platelet counts. The study concluded that delayed PPARβ/δ activation improves long-term sepsis survival by modulating cytokine and coagulation responses [28].

Neurological and neuroimmune research

Study	Summary
Neuroimmune modulation in autism model (2018) [29]	BTBR T+ tf/j mice exhibit repetitive behaviours, thermal hyperalgesia and immune dysregulation. GW0742 treatment reduced repetitive grooming, improved thermal sensitivity and decreased pro-inflammatory markers (IL-17A, RORγT, STAT3, TIM-3 and IFN-γ) while increasing IL-10/Foxp3. The results suggest that PPARβ/δ activation corrects neuroimmune dysfunction in autism [29] .
Temporal lobe epilepsy (2024) [30]	In a rat lithium-pilocarpine model of temporal lobe epilepsy, seven-day GW0742 treatment attenuated seizure-induced increases in microglial and astroglial activation genes (Aif1, Gfap) and inflammatory cytokines (Tnfa, Il1b, Il1rn). Behavioural deficits were only partially improved. GW0742 thus modulates glial gene expression during epileptogenesis [30] .
Alzheimer's disease and fatty-acid oxidation impairment (2019) [14]	See metabolic section above: GW0742 corrected fatty-acid oxidation deficits in astrocytes, enhanced neurogenesis and improved cognition in APP/PS1 mice without altering amyloid burden [14] .
Aβ1-42-induced hippocampal neurotoxicity (2016) [31]	Intrahippocampal injection of amyloid-β1-42 in rats impaired learning and memory, decreased PPARδ expression, increased nuclear NF-κB p65 and inflammatory cytokines, and elevated caspase-3 with a reduced Bcl-2/Bax ratio. GW0742 administration restored PPARδ expression, improved memory, suppressed NF-κB and pro-inflammatory cytokines and reduced apoptosis markers [31] .
Corticosterone-induced endoplasmic-reticulum stress (2017) [32]	Corticosterone reduced PPARβ/δ expression and induced ER stress and apoptosis in hippocampal astrocytes. GW0742 reversed ER-stress proteins GRP78 and CHOP, reduced caspase-12 and caspase-3 and increased microRNA-181a while decreasing its CpG methylation. This demonstrates that PPARβ/δ activation protects astrocytes against glucocorticoid-induced ER stress via epigenetic modulation [32] .
Ischemic stroke and intracerebral hemorrhage	See cardiovascular section above for details of stroke and hemorrhage studies showing GW0742's anti-inflammatory and anti-apoptotic effects [22] [23] .
Astrocyte fatty-acid oxidation and gene regulation (2024) [30]	See above.
Mast-cell degranulation after germinal-matrix hemorrhage (2024) [24]	In neonatal rats, GW0742 increased CD300a and PPARβ/δ expression, inhibited mast-cell degranulation and improved neurological outcomes after germinal-matrix hemorrhage. Knockdown of PPARβ/δ or CD300a abolished the benefits, implicating a PPARβ/δ–CD300a–SHP1 pathway in neuroprotection [24] .

Hepatic and gastrointestinal research

Study	Summary
Acetaminophen-induced acute liver injury (2025) [33]	Mice received GW0742 before or 6 h after acetaminophen overdose. The treatment blocked the rise of serum aminotransferases, reduced hepatic necrosis, oxidative stress and inflammation and suppressed M1 macrophage polarization and NLRP3 inflammasome activation. GW0742 was more effective than N-acetylcysteine, highlighting PPARβ/δ activation as a potential therapy for acetaminophen toxicity [33].
Non-alcoholic fatty-liver disease and ER stress (2018) [13]	See metabolic section: GW0742 improved β-oxidation, decreased hepatic inflammation and reduced ER stress and apoptosis in high-fat-diet–fed mice [13].
Cross-activation of PPARα causing hepatomegaly and myopathy (2008) [34]	Wild-type mice treated with GW0742 or the PPARα agonist WY-14,643 developed liver enlargement, peroxisome proliferation and skeletal myopathy. PPARα-null mice were less sensitive, indicating that GW0742's hepatomegaly and myopathy result mainly from off-target PPARα activation rather than PPARβ/δ activity [34].
Carbon tetrachloride–induced hepatotoxicity (2008) [35]	PPARβ/δ-null mice showed greater CCl_4 hepatotoxicity than wild-type mice. In wild-type mice, GW0742 co-administration reduced serum ALT, inflammatory cytokines and TWEAK receptor expression. Protection was absent in PPARβ/δ-null mice, suggesting that GW0742 ameliorates hepatotoxicity by down-regulating pro-inflammatory genes [35].

Oncology and pro-metastatic effects

Study	Summary
VDR and nuclear receptor antagonism (2013) [36]	High-throughput screening identified GW0742 as a vitamin D receptor (VDR) co-activator inhibitor. At high concentrations (>12 μM), GW0742 behaved as a pan-nuclear-receptor antagonist; it inhibited VDR-coactivator interactions and down-regulated VDR target genes such as CYP24A1, IGFBP-3 and TRPV6. Thus GW0742 can act as a VDR antagonist independently of its PPARβ/δ activity [36].
Vascular PPARβ/δ promotes tumour angiogenesis (2019) [37]	Transgenic mice with inducible vascular overexpression of PPARβ/δ showed increased tumour growth, vessel density and metastasis. Activation of PPARβ/δ with GW0742 enhanced tumour vascularisation and growth, whereas PPARD loss reduced metastasis. RNA sequencing identified PDGFRβ, PDGFb and c-Kit as downstream targets, highlighting that PPARβ/δ activation can promote tumour angiogenesis and warrants caution when targeting this pathway [37].

Study	Summary
GW0742 as a thyroid-receptor antagonist in granulosa-cell tumours (2024) [38]	GW0742 reduced viability and metabolic activity of granulosa-cell tumour lines and increased TRβ expression. The study found that GW0742, a PPARβ/δ agonist, also acts as a thyroid-receptor antagonist and may inhibit tumour cell growth, suggesting therapeutic potential in tumours expressing thyroid receptors [38] .
Metastasis regulation by PPARD expression (2017) [39]	In nude mice injected with luciferase-labeled HCT-116 colon cancer cells, diets containing GW0742 (1 mg/kg) increased hepatic metastases compared with control diets, while PPARD knockout cells failed to metastasize. The study concluded that pharmacologic activation of PPARD promotes metastasis and angiogenesis; conditioned medium from GW0742-treated cancer cells increased endothelial tubule formation [39] .
Granulosa tumour cell viability via TRα/β antagonism (2024) [38]	See above: GW0742 acted as a thyroid-receptor antagonist and reduced metabolic activity of granulosa tumour cells [38] .
Anti-doping detection of GW0742 metabolites (2012) [40]	In vitro studies identified oxidation of the sulfur moiety to sulfoxide and sulfone as the main metabolic pathway for GW0742. The sulfone metabolite remained detectable in human urine for up to 20 days, whereas unmetabolized GW0742 was present only briefly. The authors recommended monitoring sulfone metabolites in doping control to detect GW0742 misuse [40] .

Miscellaneous and emerging research

Study	Summary
Endothelial metabolism and angiogenesis (2020) [41]	Human umbilical-vein endothelial cells treated with VEGF-A increased glycolysis, whereas GW0742 reduced both glycolysis and fatty-acid oxidation in monolayers. During tubulogenesis, GW0742 increased fatty-acid oxidation and modestly enhanced glycolysis. Tubulogenesis required PPARβ/δ and SIRT1, revealing context-dependent metabolic regulation by GW0742 [41] .
Neointimal hyperplasia and stent coating	See cardiovascular section for details [15] .
Sepsis survival and coagulation	See sepsis section.

Conclusion

Across multiple organ systems, GW0742 has been widely used to explore PPARβ/δ biology. In metabolic and cardiovascular models, GW0742 improves glucose homeostasis, endothelial function and lipid metabolism. It exerts anti-inflammatory and anti-apoptotic effects in models of acute lung injury, ischemia/reperfusion, pancreatitis, spinal cord injury and sepsis, often via suppression of NF-κB, reduction of ROS and

enhancement of fatty-acid oxidation. Neurologically, GW0742 protects against neuroinflammation, glucocorticoid-induced ER stress, Alzheimer's-related metabolic deficits and germinal-matrix hemorrhage. However, its activation of PPARβ/δ can promote tumour angiogenesis and metastasis and may act as a thyroid-receptor or VDR antagonist at high concentrations. GW0742 also exhibits off-target PPARα activation leading to hepatomegaly and myopathy. Thus, while GW0742 is a valuable research tool and holds therapeutic promise, careful consideration of dose, receptor expression and potential pro-tumorigenic effects is essential when translating findings to clinical settings.

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