ERR AGONIST · EXERCISE MIMETIC · ENDURANCE · MITOCHONDRIA

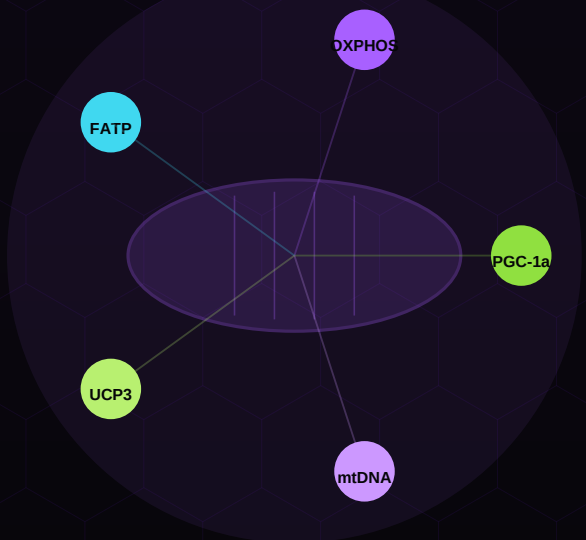
SLU-PP-332

Endurance · Mitochondria · Fat Oxidation

The world's most potent ERR alpha/gamma agonist — producing the metabolic signature of endurance training without exercise.

ERRa/g	70%	~5x	Oral
Dual Agonist	More mtDNA*	Endurance*	Bioavailable

DOWNSTREAM TARGETS



What is SLU-PP-332?

A synthetic small-molecule dual agonist of ERRa and ERRg — the 'exercise-in-a-pill' receptor

SLU-PP-332 is a potent synthetic agonist of the estrogen-related receptors ERRalpha and ERRgamma — orphan nuclear receptors that serve as master transcriptional regulators of mitochondrial biogenesis and oxidative metabolism.

Developed at St. Louis University and described in 2023 Nature Communications research, SLU-PP-332 activates the same transcriptional programmes triggered by endurance exercise — dramatically increasing mitochondrial density, oxidative phosphorylation capacity, and fatty acid oxidation without physical activity.

In preclinical studies, treated mice ran nearly 50% further on a treadmill, showed 70% more mitochondrial DNA in skeletal muscle, and demonstrated significant improvements in body composition and metabolic health markers.

ERRa and ERRg are constitutively active orphan receptors with no known endogenous ligand, making SLU-PP-332 the first pharmacological tool to directly and selectively activate both isoforms simultaneously.

MOLECULAR IDENTITY

SLU-PP-332 | GSK4716 Analogue

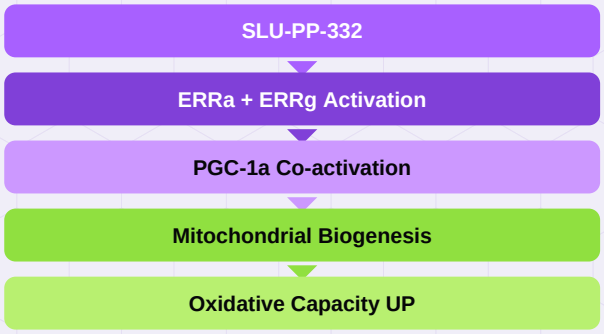
ERRa/g dual agonist | Oral small molecule | MW ~400 Da

The ERR receptor family sits upstream of PGC-1alpha — the master regulator of mitochondrial biogenesis.

By directly activating ERRa and ERRg, SLU-PP-332 bypasses the need for AMPK, SIRT1, or calcium signalling that normally transduce exercise signals. This makes it one of the most direct exercise mimetics ever identified.

Beyond muscle, ERRg is highly expressed in heart, brain, and brown adipose tissue — meaning SLU-PP-332 simultaneously enhances cardiac metabolism, neuroprotection, and thermogenic fat burning.

SLU-PP-332 is distinct from cardarine (GW501516, PPAR-delta agonist) — it acts on a separate, upstream transcription factor axis with a fundamentally different downstream gene expression profile.



Key Benefits

Preclinical data redefining what metabolic science can achieve

Endurance Enhancement

Preclinical treadmill studies show SLU-PP-332-treated mice run nearly 50% further than controls, with dramatic improvements in time-to-exhaustion — a magnitude of effect unprecedented for a single oral compound.

Mitochondrial Biogenesis

Skeletal muscle analysis confirms 70% increase in mitochondrial DNA copy number and significant upregulation of OXPHOS complex subunits — the same adaptation that takes months of endurance training to achieve.

Fat Oxidation & Body Composition

ERRa/g activation dramatically upregulates fatty acid transport proteins and beta-oxidation enzymes, shifting substrate utilisation toward fat — producing improvements in body composition independent of caloric restriction.

Cardiac Protection

ERRg is the dominant nuclear receptor in cardiac metabolism. SLU-PP-332 enhances cardiac energy efficiency, improves contractile reserve, and shows protection in models of heart failure and ischaemia-reperfusion injury.

Metabolic Disease Therapy

In diet-induced obese and diabetic models, SLU-PP-332 normalises blood glucose, reduces hepatic lipid accumulation, and improves insulin sensitivity — addressing metabolic syndrome from its mitochondrial root cause.

Neuroprotection

Brain-expressed ERRg supports neuronal energy metabolism. Emerging data suggests SLU-PP-332 may protect against neurodegeneration by maintaining mitochondrial health in neurons — an area of active investigation.

SLU-PP-332 vs. GW501516 (Cardarine) vs. AICAR vs. Actual Exercise

Research & Dosing

Emerging science from the frontier of exercise pharmacology

2012

ERR Biology Established

Extensive preclinical work established ERRalpha and ERRgamma as the dominant transcriptional regulators of cardiac and skeletal muscle oxidative metabolism — and confirmed their activation recapitulates exercise-induced gene expression.

2019

First ERR Dual Agonists

St. Louis University team synthesised early ERRa/g dual agonists derived from the GSK4716 scaffold. Dose-dependent upregulation of PGC-1a, OXPHOS genes, and mitochondrial biogenesis markers confirmed in cell culture.

2022

SLU-PP-332 Described

Refined lead compound SLU-PP-332 showed dramatically improved potency and oral bioavailability. In vitro ERRa/g binding confirmed sub-nanomolar activity, with clean off-target selectivity profiling.

2023

Nature Communications Publication

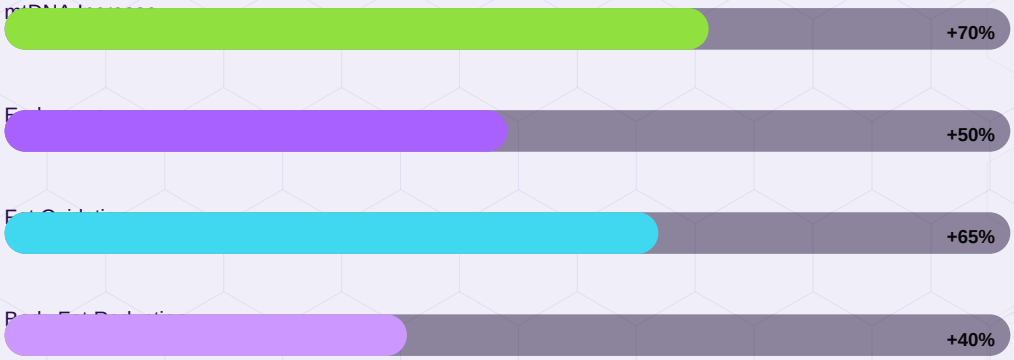
Landmark study published in Nature Communications demonstrated: 70% increase in skeletal muscle mtDNA, 45-50% improvement in treadmill endurance, significant fat oxidation upregulation, and cardiac protection — all in sedentary mice.

2024

Ongoing Investigation

Multiple academic groups are now investigating SLU-PP-332 in models of heart failure, metabolic syndrome, neurodegeneration, and ageing. Phase I human safety studies are in active preparation at multiple institutions.

Preclinical Effect Magnitude vs. Sedentary Control



Dosing Guide

Form	Oral capsule / solution
Preclinical dose	~10-30 mg/kg (mouse)
Human equiv. est.	Not yet established
Route	Oral (good bioavailability)
Half-life	~4-8 hours (estimated)
Timing	Morning with food
Cycle	Research protocols only
Storage	Cool, dry, dark

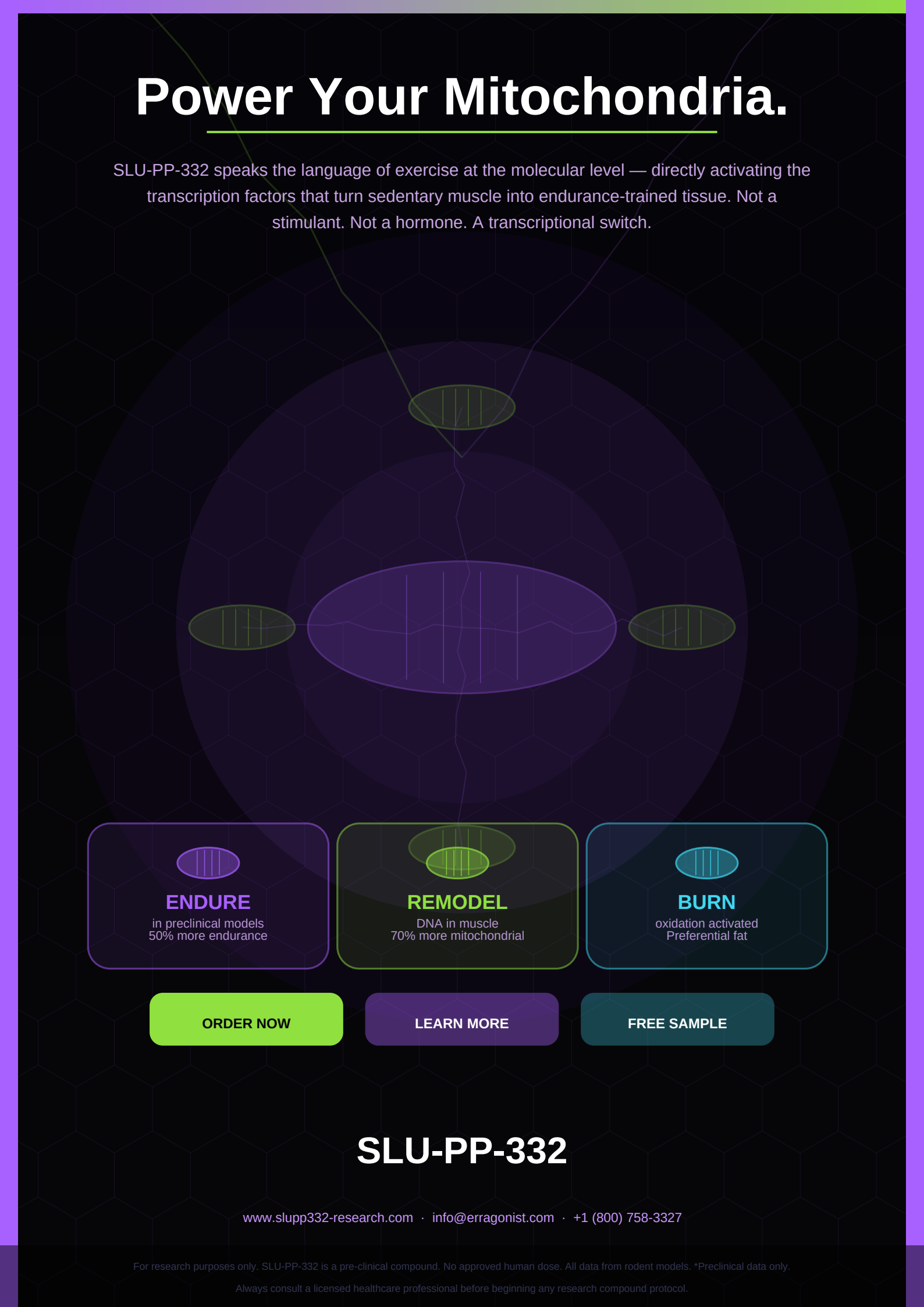
Synergistic Compounds

NAD+ · NMN · 5-Amino 1MQ · Berberine · AIC

Pre-clinical compound. No approved human dosing. All data from rodent studies. Research use only. Physician supervision required.

Power Your Mitochondria.

SLU-PP-332 speaks the language of exercise at the molecular level — directly activating the transcription factors that turn sedentary muscle into endurance-trained tissue. Not a stimulant. Not a hormone. A transcriptional switch.



ENDURE
in preclinical models
50% more endurance

REMODEL
DNA in muscle
70% more mitochondrial

BURN
oxidation activated
Preferential fat

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