

FOR LICENSED PRACTITIONERS ONLY

# Redox Signalling & Cellular Communication: A Practitioner's Evidence Guide

The biology, the evidence hierarchy, and the language — for clinicians exploring between-visit cellular wellness support.

- 3 pages of evidence-graded content
- 9 peer-reviewed references
- 5 step patient conversation guide

## KEY PRINCIPLE

"The goal is not to eliminate reactive species — it is to support the cellular machinery that maintains appropriate redox balance."

## PRACTICE FREEDOM

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## FROM OXIDATION TO COMMUNICATION

# What Redox Signalling Actually Is

The term **redox** refers to reduction–oxidation reactions. For decades, clinical conversation focused on damage: free radicals, oxidative stress, and antioxidants. That framing was incomplete.

Contemporary cell biology shows that reactive oxygen and nitrogen species — at physiologically appropriate concentrations — function as **signalling molecules**: chemical messengers that regulate gene expression, modulate enzyme activity, and govern cellular repair.

"Reactive oxygen species are not merely toxic byproducts of aerobic metabolism but are also important regulators of cell signalling pathways."

— Sies et al., PMC 2022 [1]

**The Balance Principle:** The goal is not to eliminate reactive species — it is to **support the cellular machinery that maintains appropriate redox balance.**



## Key Molecular Players

MOLECULE	SIGNALLING ROLE
<b>H2O2 (Hydrogen Peroxide)</b>	Second messenger; activates downstream repair targets
<b>Nitric oxide (NO)</b>	Regulates vascular tone & endothelial function
<b>NRF2 pathway</b>	Master transcription factor; upregulates antioxidant enzymes [2]
<b>SOD / Catalase</b>	First enzymatic antioxidant defences; mitochondrial health

# What the Research Shows — and Where the Gaps Are

An honest evidence appraisal. Practitioner credibility depends on intellectual honesty about evidence quality.

EVIDENCE TIER	WHAT IT SHOWS	CONFIDENCE
<b>Established mainstream biology</b>	ROS as signalling molecules; NRF2 pathway function; redox regulation of gene expression [1][2]	High
<b>Disease-mechanism research</b>	Redox dysregulation implicated in vascular dysfunction, neurodegeneration, metabolic disease [3][4]	Mod-High
<b>Oxidative stress biomarkers</b>	8-OHdG, oxidised LDL, F2-isoprostanes as validated measurable markers [5]	Moderate
<b>Cellular / in vitro product studies</b>	Company-presented data on antioxidant enzyme efficiency, NRF2 nuclear translocation in cultured cells	Low-Mod
<b>Human safety data (ASEA)</b>	12-week RCT (n=106); no significant adverse lab findings reported	Mod (safety)
<b>Performance RCT (Utah State)</b>	No significant difference in VO <sub>2</sub> max or ventilatory threshold vs placebo [6]	Null result

### WHAT THE LITERATURE SUPPORTS

A 2022 review in Science Advances described redox signalling as operating at "the crossroads of human health and disease," noting its role in immune regulation and cellular ageing.

### WHAT REMAINS PRELIMINARY

Translation from cell biology to proven clinical intervention is not automatic. The null VO<sub>2</sub>max result [6] is a reminder that mechanistic plausibility does not guarantee clinical efficacy.

## The Biomarker Opportunity

Validated oxidative stress biomarkers — **8-OHdG** (urinary marker of oxidative DNA damage) and **oxidised LDL** — have established reference ranges and can track changes in oxidative load over time. [5] This creates a framework for objective, patient-centred monitoring that does not require disease claims.

### NRF2 Activation Pathway

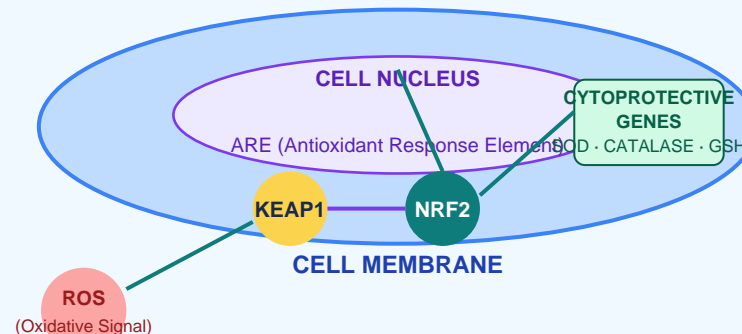


Fig 2. NRF2 Activation Pathway — oxidative signal triggers cytoprotective gene expression.

# How to Talk About This With Patients and Colleagues

*In no other area of integrative practice does word choice carry more regulatory and reputational risk.*

## THE THREE-TIER CLAIMS LADDER

### Compliant Language Framework

#### TIER A — ALWAYS APPROPRIATE

Structure / Function Language

'supports cellular communication'

'promotes oxidative balance'

#### TIER B — USE WITH CITATIONS

Mechanistic Research Language

NRF2 pathway in cell studies

oxidative stress markers (e.g., MDA)

#### TIER C — AVOID

Disease Claims

'treats inflammation'

'cures fatigue'

Professional Medical Compliance Guidance  
Claims Based on Current Regulatory Standards

## FIVE-STEP PATIENT CONVERSATION

### Introducing Redox Signalling

#### 1 Start with biology they already understand

*"You know how your body has built-in repair systems — like how a cut heals? Those processes depend on chemical signals inside your cells. Researchers call this redox chemistry."*

#### 2 Normalise reactive species

*"At the right levels, these reactive molecules are actually messengers — telling your cells when to activate repair, ramp up antioxidant defences, and regulate inflammation."*

#### 3 Name the problem without a disease claim

*"In chronic conditions — persistent fatigue, ongoing pain, slow recovery — there's often evidence that this signalling system is under stress. The balance is off."*

#### 4 Introduce the support concept

*"I'm exploring whether we can support that cellular communication system between visits — not to treat a condition but to give your body's own repair signals better conditions to work."*

#### 5 Set expectations honestly

## WITH COLLEAGUES

### Lead With Mechanisms

When introducing this topic with peers, use mechanistic language and acknowledge uncertainty:

*"I've been exploring the redox signalling literature and looking at whether there are practical ways to support oxidative balance as part of a between-visit protocol. Here's what the evidence shows, and here's what I'm being careful about."*

#### Regulatory Guardrails

The FDA distinguishes clearly between **structure/function claims** (permitted) and **disease claims** (not permitted for supplements). The FTC requires substantiation for all health-related product claims. [7][8]

## REFERENCES

[1] Sies et al. (2022). Defining roles of ROS in cell biology. PMC8971743.

[2] Tonelli et al. (2018). Transcriptional Regulation by Nrf2. PMC5737637.

[3] Liguori et al. (2018). Oxidative stress, aging, and diseases. PMC5927356.