In vitro evaluation of Protandim® Nrf2 Synergizer® and its effect on oxidative stress activity after exposure of cells to 5 environmental toxins.

Objective: Evaluate the effects of Protandim® Nrf2 Synergizer® on viability and ROS activity after exposing various cell lines to environmental toxins.
Sponsor: LifeVantage Corp.
Reference: LifeVantage Corp., Lehi, Utah 84043

Introduction

Protandim Nrf2 Synergizer has been proven to decrease general oxidative stress in humans by 40% in 30 days (Nelson et al. 2006) and the mechanism of action has been well established.* †

The blend of 5 standardized botanicals works through activating the Nrf2-pathway, allowing the sequestered Nrf2-protein to be released and translocated into the nucleus and bind to the Antioxidant Response Element (ARE) promoter region on the DNA. This leads to bioprotective downstream activation of genes coding for enzymes involved in mediating oxidative stress damage.

Oxidative stress occurs when the production of reactive oxygen species (ROS) exceeds the capacity of cellular antioxidant defenses, disrupting redox regulation and potentially damaging lipids, proteins, and DNA. ROS are continuously generated during normal cellular metabolism and can also be generated from environmental influences as well as the foods we eat. The body normally maintains a balance between ROS generation and its antioxidant defense, but if there is an imbalance in favor of ROS generation then this imbalance can lead to a condition known as oxidative stress. Oxidative stress can disrupt cellular energy production, signaling, structural integrity, and normal tissue repair processes. It also can damage critical cellular components, including lipids, proteins, and DNA.

Over time, this oxidative damage may contribute to cellular dysfunction, cell stress and die-off (senescence), reduced tissue resilience, and biological processes associated with aging. Age-related issues include reduced cellular energy, slower recovery and repair, declining cardiovascular and metabolic function, changes in cognition, loss of muscle function, skin aging, and reduced cellular resilience.

In today's world, we are exposed to many types of everyday stress that challenge our mind, body, and cells. We are not exposed to just one challenge at a time but rather experience multiple mental, physical, lifestyle/diet, and environmental challenges simultaneously that can cause oxidative stress. Continuously being exposed to these challenges can lead to poor sleep, feeling fatigued, brain fog, mood changes, digestive changes, mental stress, and accelerated cellular aging

Today, a growing body of evidence is showing that certain lifestyle and environmental toxins such as microplastics, pollution, phthalates, glyphosates, and ultra-processed foods have a detrimental effect on our bodies. Table 1 shows the potential effects in the body and its associated mechanism of action.

Table 1. Environmental toxins with potential effects in the body after exposure

Toxins	Mechanism of Action	Potential Effects in Body	Prevalence and Health-Impact Statistics
Microplastics	Oxidative stress, particle-associated celluar stress	Vascular, immune, metabolic, and tissue effects	Micro- and nanoplastics were detected in **100% of non-tumor brain samples** and **99.4% of brain-tumor samples** analyzed in a 2026 study. In patients undergoing surgery for carotid artery disease, **polyethylene was detected in 58.4% of artery plaques.** Preclinical studies show evidence of interference of micro- or nanoplastics with circulation in the brain causing **neurological and behavioral abnormalities.** Other research shows **learning and memory impacts** in animal models. Patients with heart issues in general, whose plaques contained micro- or nanoplastics had a **4.5-times higher rate** of heart attack, stroke, or death during approximately 34 months of follow-up. (Huang et al 2025, Li et al. 2026, Marfella et al. 2024, Zuri et al. 2024)
Air Pollutions (particulate matter)	Oxidative stress, endothelial stress, mitochondrial stress	Respiratory and cardiovascular effects	**99% of the global population** breathes air exceeding WHO air-quality guidelines. Indoor and outdoor air pollution are associated with approximately **7 million premature deaths annually**. (WHO, Gangwar et al. 2020, Daiber et al. 2020)

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Table 1 cont.

Toxins	Mechanism of Action	Potential Effects in Body	Prevalence and Health-Impact Statistics
Horomone Disruptors (phthalates)	Oxidative stress, altered cellular signaling, endocrine disruption	Endocrine, reproductive, and metabolic effects	BPA was detected in **93% of Americans tested** in a nationally representative CDC survey. A large U.S. analysis estimated that prenatal phthalate exposure was associated with approximately **56,600 preterm births in one year**. (Calafat et al. 2008, Transande et al. 2024, Gore et al. 2015)
Pesticides (glyphosates)	Oxidative stress	Cellular challenges	**81% of Americans age 6+** had glyphosate detected in their urine—evidence of recent exposure. Children in the highest prenatal organophosphate-exposure group averaged **7 fewer IQ points** than those in the lowest group. (Ospina et al. 2022, Chang et al. 2023, Bouchard et al. 2011)
Ultra-processed foods (methylglyoxal)	Excess energy intake, metabolic stress, glycemic/lipid effects, oxidative stress	Obesity, cardiovascular effects	Ultra-processed foods provide **53% of adult calories and nearly 62% of youth calories** in the United States. In a controlled NIH trial, participants ate approximately **500 extra calories per day** and gained an average of **2 pounds in two weeks** on an ultra-processed diet. (Williams et al. 2025, Hall et al. 2019, Lane et al. 2024)

A cell study was developed to further investigate and evaluate the beneficial effects of Protandim Nrf2 Synergizer on the environmental influences of microplastics, pollution, hormone disruptors, pesticides, and ultra-processed food/diet-related stressors (methylglyoxal). Three different cell types were chosen for a multi-tissue exposure platform:

1. HepG2 cells model aspects of liver metabolism, including the processing of absorbed compounds and foreign substances.
2. Dermal fibroblasts model how skin and connective tissue cells respond to environmental stressors.
3. Caco-2 cells model aspects of the intestinal lining, a key point of contact for substances consumed in food and drink.

MATERIALS AND METHODS

The study was conducted in two parts:

1. A dose/viability finding study for liver (HepG2), fibroblasts (CCD-986Sk) cells, and gut (Caco-2).

To assess potential cytotoxicity and identify suitable concentrations for subsequent experiments, cell viability was evaluated following exposure to each test stressor alone or Protandim Nrf2 Synergizer using the CellTiter-Glo® Luminescent Cell Viability Assay. This assay quantifies ATP as a marker of metabolically active cells.

Cells were seeded at a density of 500 cells / well in 384-well white-walled Cultur-Plate-384 cell culture plates (Perkin-Elmer, Waltham MA, Catalog #6007680) and exposed to test agents the following day. Cells were allowed to grow in the presence of test agent for 24 hours for dose-course studies, at which point CellTiter-Glo (Promega Corp., Madison WI, Catalog #G7571) reagent was added at a volume equal to the cell culture medium in the plate, according to manufacturer's instructions. Luminescence was read on an Envision 2104 multilabel reader (Perkin-Elmer, Waltham WI). (Table 2)

Table 2. Viability/dosing matrix.

Treatment	Dosage	Duration (hrs)
Protandim Nrf2 Synergizer	400, 200, 100, 75, 50, 25, 12.5, 0 ug/ml	24
Microplastics	0.727, 0.545, 0.364, 0.182, 0.091, 0.045, 0.023, 0 % solids	24
Pollution	200, 100, 50, 25, 12.5, 6.25, 3.12, 0 ug/ml	24
Hormone disruptors	200, 100, 50, 25, 12.5, 6.25, 3.12, 0 ug/ml	24
Pesticide	10, 5, 2.5, 1.25, 0.625, 0.3125, 0.15625, 0 mM	24
Ultra-processed food (methylglyoxal)	1, 0.3, 0.1, 0.03, 0.01, 0.003, 0.001, 0 mM	24

2. Modern toxin evaluation after Protandim Nrf2 Synergizer treatment in all three cell lines, using imagery and ROS assay kits.
The following modern toxins were used:

a. Microplastics—100 nm FluoSpheres™ carboxylate-modified fluorescent polystyrene nano-particles from InVitrogen (Thermo Fisher Scientific Cat. # F8803)

b. Pollution—NIST SRM 2786 Fine Atmospheric Particulate Matter <4µm from Sigma-Aldrich (Cat. NIST2786)

c. Hormone disruptors—Phthalates DEHP (CAS: 117-81-7) from Sigma-Aldrich (Cat. 36735)

d. Pesticide—Pure Glyphosate (CAS: 1071-83-6) from Sigma-Aldrich (Cat. 45521)

e. Ultra-processed food—Methylglyoxal, a representative for ultra-processed food (CAS: 78-98-8) from Sigma-Aldrich (Cat. M0252)

The following fluorescent ROS assay was used: DCFDA/H2DCFDA–Cellular ROS Assay Kit by Abcam. Fluorescence was measured using a fluorescence microscopy where stressed cells are stained green and the nucleus is stained blue.

RESULTS

The study was conducted in two parts:

1. Dosage and Viability

Final dosing was established by comparing concentrations to relative viability percentages. At least a 90% viability was desired and the following treatment concentrations were established for all cells. (Table 3)

Table 3. Dosing established from viability assays

Treatment	Established Dosage
Pollution	40 ug/ml
Pesticides	4 mM
Hormone disruptors	20 ug/ml
Microplastics	0.045%
Ultra-processed food	200 uM

Table 4 shows the % cell death of HepG2, fibroblasts, and Caco-2 cells treated with microplastics, pollution, hormone disruptors, pesticides, and ultra-processed food. The resulting data makes clear how toxic these treatments are to the survivability of each cell line, especially pollution and microplastics.

Table 4. Percent death after treating cells with stressors at the recommended dosages.

	% Cell Death		
	HepG2	Fibroblasts	Caco-2
Pollution	-31%	-34%	-29%
Pesticides	-3%	-7%	-8%
Hormone disruptors	-8%	-7%	-6%
Microplastics	-21%	-22%	-26%
Ultra-processed food	-8%	-7%	-5%

2. ROS and Fluorescence Microscopy

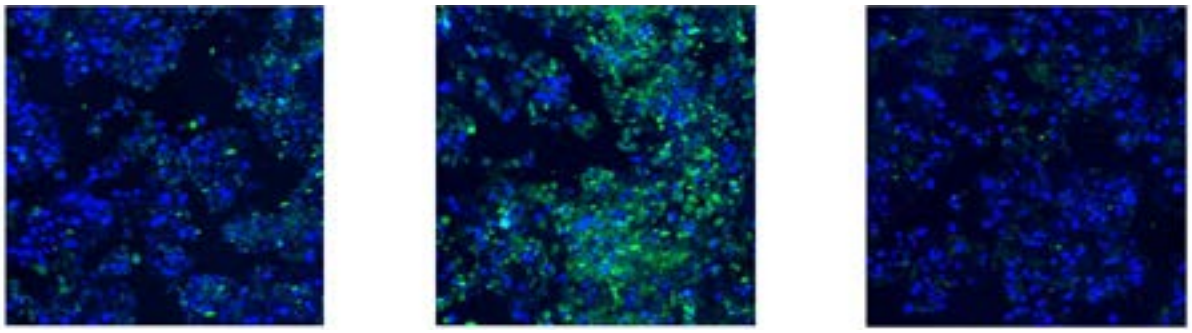
After determining the proper dosage and timepoint for evaluation, fibroblasts, HepG2, and Caco-2 cells were treated with and without toxins at those dosages. Each treatment was run in triplicate, and reduction in ROS was calculated. Images under a fluorescent microscope were also created.

Figures 1–12 show the fluorescent images of HepG2 cells, fibroblast cells, and Caco-2 cells under the influence of pollution, pesticides, hormone disruptors, microplastics, and ultra-processed food. Panel A depicts the control cells at rest and without any treatment. Panel B represents the cells treated with one of the toxins listed in the Materials & Method section. The third panel, C., represents the cells under stress and treated with Protandim Nrf2 Synergizer. The green color depicts the ROS created during stress. A reduction in green fluorescence is desirable and represents less ROS generated.

a. Oxidative Stress results in HepG2 cells

HepG2: Pollution

A. **B.** **C.**

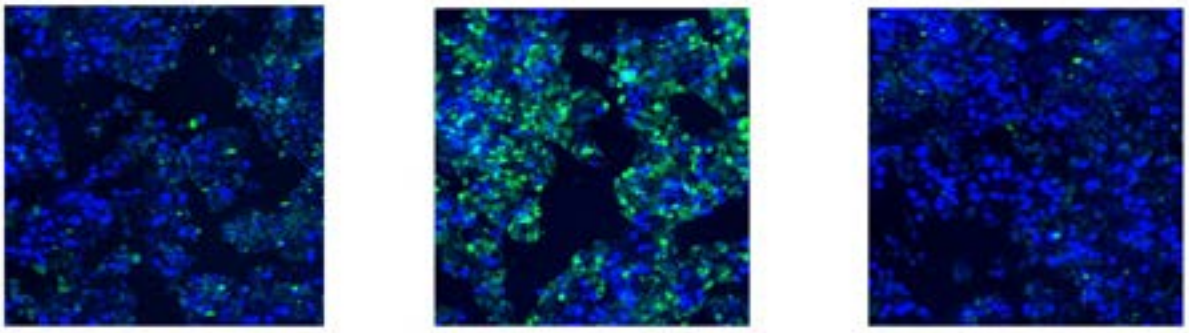


Cells without stressor **Cells stressed only** **Cells stressed + Nrf2 (US)**

Figure 1A-C. This depicts HepG2 cells under a fluorescent microscope at baseline (A.), under stress treatment with pollution (particulate matter) (B.), and stressed cells after treatment with Protandim Nrf2 Synergizer (C.). During stress treatment, cells turn more fluorescent green to indicate oxidative stress (ROS) is generated. Panel C shows a reduction in oxidative stress (less green fluorescence) with the addition of Protandim Nrf2 Synergizer.*

HepG2: Pesticides

A. **B.** **C.**



Cells without stressor **Cells stressed only** **Cells stressed + Nrf2 (US)**

Figure 2A-C. This depicts HepG2 cells under a fluorescent microscope at baseline (A.), under stress treatment with pesticides (glyphosates) (B.), and stressed cells after treatment with Protandim Nrf2 Synergizer (C.). During stress treatment, cells turn more fluorescent green to indicate oxidative stress (ROS) is generated. Panel C shows a reduction in oxidative stress (less green fluorescence) with the addition of Protandim Nrf2 Synergizer.*

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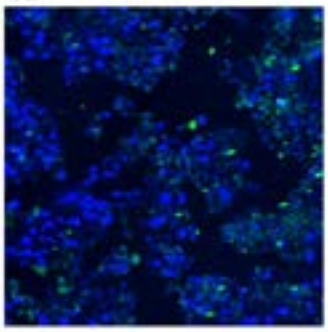
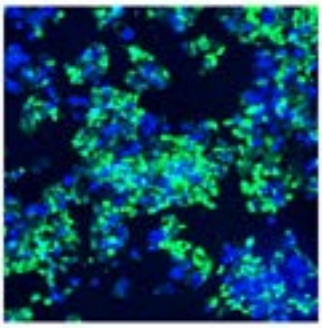
HepG2: Hormone Disruptors

A.



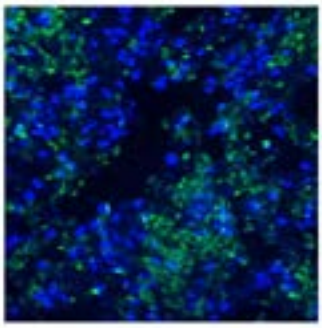
Cells without stressor

B.



Cells stressed only

C.

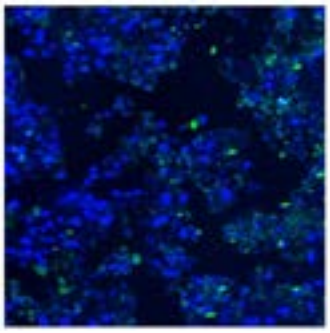


Cells stressed + Nrf2 (US)

Figure 3A-C. This depicts HepG2 cells under a fluorescent microscope at baseline (A.), under stress treatment with hormone disruptors (phthalates) (B.), and stressed cells after treatment with Protandim Nrf2 Synergizer (C.). During stress treatment, cells turn more fluorescent green to indicate oxidative stress (ROS) is generated. Panel C shows a reduction in oxidative stress (less green fluorescence) with the addition of Protandim Nrf2 Synergizer.*

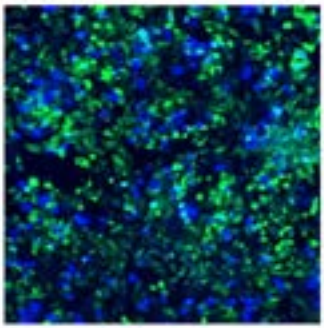
HepG2: Microplastics

A.



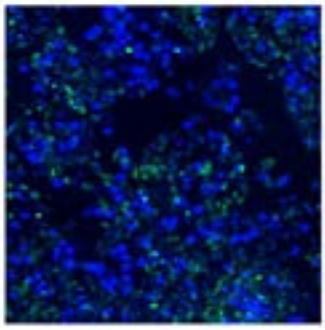
Cells without stressor

B.



Cells stressed only

C.



Cells stressed + Nrf2 (US)

Figure 4A-C. This depicts HepG2 cells under a fluorescent microscope at baseline (A.), under stress treatment with microplastics (B.), and stressed cells after treatment with Protandim Nrf2 Synergizer (C.). During stress treatment, cells turn more fluorescent green to indicate oxidative stress (ROS) is generated. Panel C shows a reduction in oxidative stress (less green fluorescence) with the addition of Protandim Nrf2 Synergizer.*

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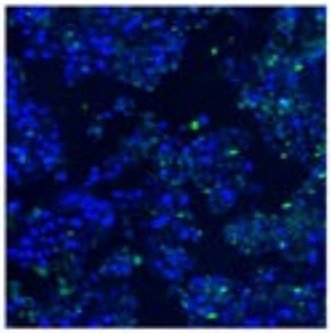
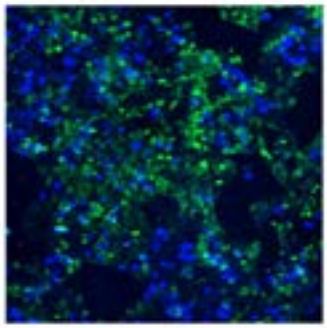
HepG2: Methylglyoxal

A.



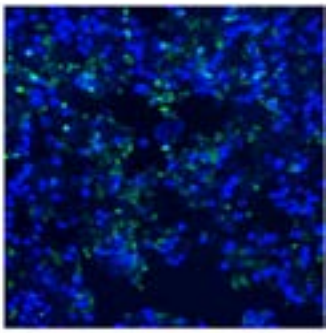
Cells without stressor

B.



Cells stressed only

C.



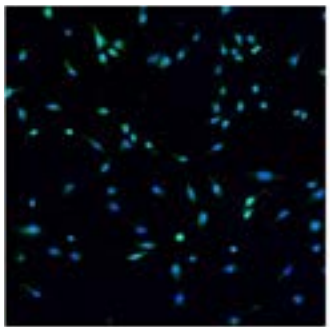
Cells stressed + Nrf2 (US)

Figure 5A-C. This depicts HepG2 cells under a fluorescent microscope at baseline (A.), under stress treatment with methylglyoxal simulating ultra-processed foods (B.), and stressed cells after treatment with Protandim Nrf2 Synergizer (C.). During stress treatment, cells turn more fluorescent green to indicate oxidative stress (ROS) is generated. Panel C shows a reduction in oxidative stress (less green fluorescence) with the addition of Protandim Nrf2 Synergizer.*

b. Oxidative stress results in fibroblasts

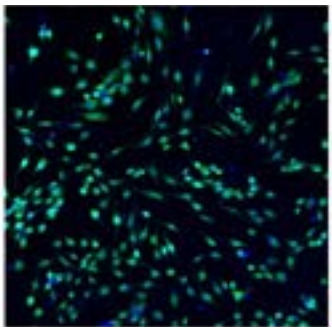
Fibroblasts: Pollution

A.



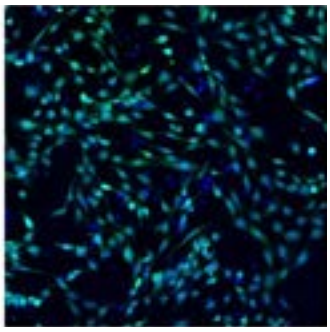
Cells without stressor

B.



Cells stressed only

C.



Cells stressed + Nrf2 (US)

Figure 6A-C. This depicts fibroblast cells under a fluorescent microscope at baseline (A.), under stress treatment with pollution (particulate matter) (B.), and stressed cells after treatment with Protandim Nrf2 Synergizer (C.). During stress treatment, cells turn more fluorescent green to indicate oxidative stress (ROS) is generated. Panel C shows a reduction in oxidative stress (less green fluorescence) with the addition of Protandim Nrf2 Synergizer.*

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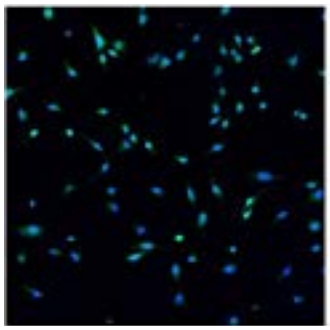
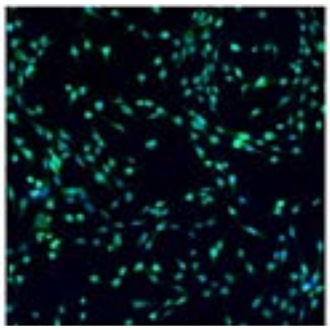
Fibroblasts: Pesticides

A.



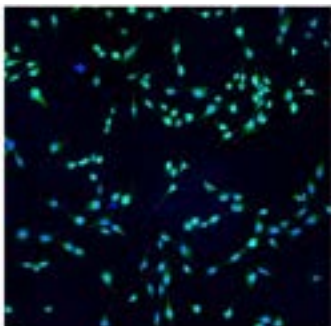
Cells without stressor

B.



Cells stressed only

C.

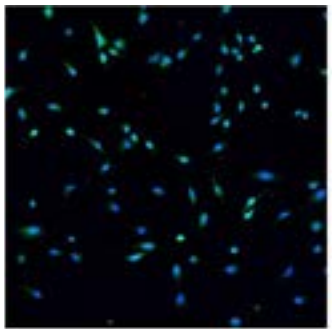


Cells stressed + Nrf2 (US)

Figure 7A-C. This depicts fibroblast cells under a fluorescent microscope at baseline (A.), under stress treatment with pesticides (glyphosates) (B.), and stressed cells after treatment with Protandim Nrf2 Synergizer (C.). During stress treatment, cells turn more fluorescent green to indicate oxidative stress (ROS) is generated. Panel C shows a reduction in oxidative stress (less green fluorescence) with the addition of Protandim Nrf2 Synergizer.*

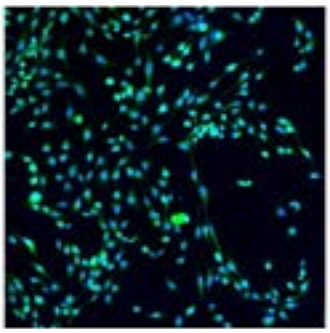
Fibroblasts: Hormone Disruptors

A.



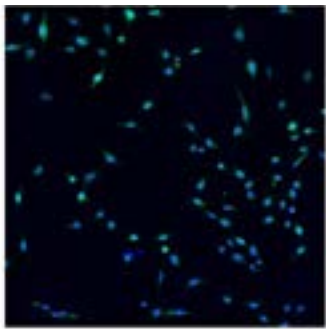
Cells without stressor

B.



Cells stressed only

C.



Cells stressed + Nrf2 (US)

Figure 8A-C. This depicts fibroblast cells under a fluorescent microscope at baseline (A.), under stress treatment with hormone disruptors (phthalates) (B.), and stressed cells after treatment with Protandim Nrf2 Synergizer (C.). During stress treatment, cells turn more fluorescent green to indicate oxidative stress (ROS) is generated. Panel C shows a reduction in oxidative stress (less green fluorescence) with the addition of Protandim Nrf2 Synergizer.*

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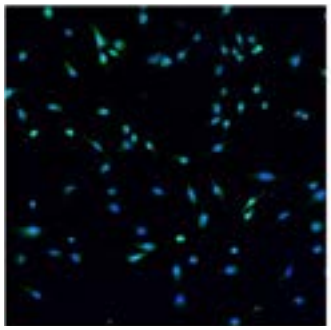
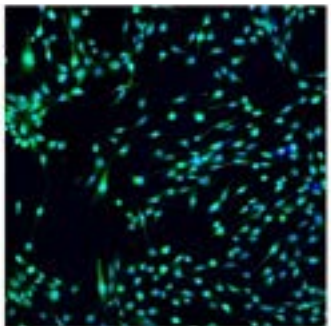
Fibroblasts: Microplastics

A.



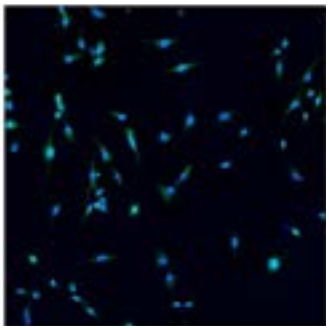
Cells without stressor

B.



Cells stressed only

C.



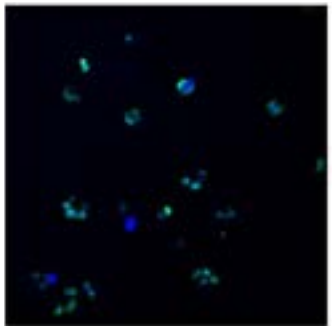
Cells stressed + Nrf2 (US)

Figure 9A-C. This depicts fibroblast cells under a fluorescent microscope at baseline (A.), under stress treatment with microplastics (B.), and stressed cells after treatment with Protandim Nrf2 Synergizer (C.). During stress treatment, cells turn more fluorescent green to indicate oxidative stress (ROS) is generated. Panel C shows a reduction in oxidative stress (less green fluorescence) with the addition of Protandim Nrf2 Synergizer.*

c. Oxidative stress results in Caco-2 cells

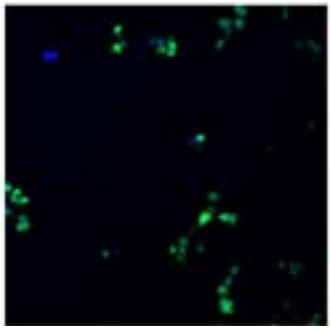
Caco-2: Pesticides

A.



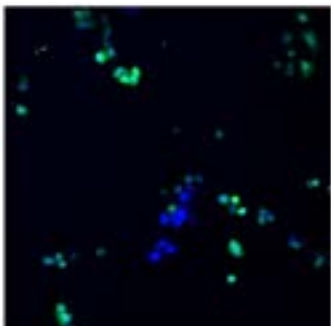
Cells without stressor

B.



Cells stressed only

C.



Cells stressed + Nrf2 (US)

Figure 10A-C. This depicts Caco-2 cells under a fluorescent microscope at baseline (A.), under stress treatment with pesticides (glyphosates) (B.), and stressed cells after treatment with Protandim Nrf2 Synergizer (C.). During stress treatment, cells turn more fluorescent green to indicate oxidative stress (ROS) is generated. Panel C shows a reduction in oxidative stress (less green fluorescence) with the addition of Protandim Nrf2 Synergizer.*

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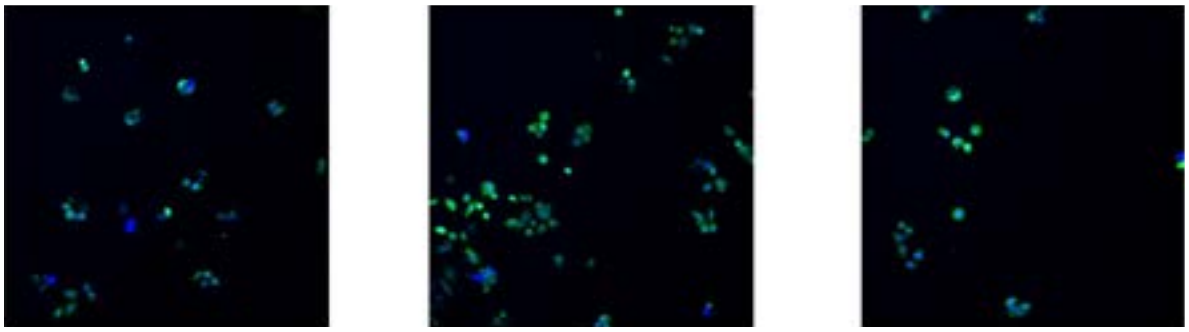
Caco-2: Hormone Disruptors

A. **B.** **C.**

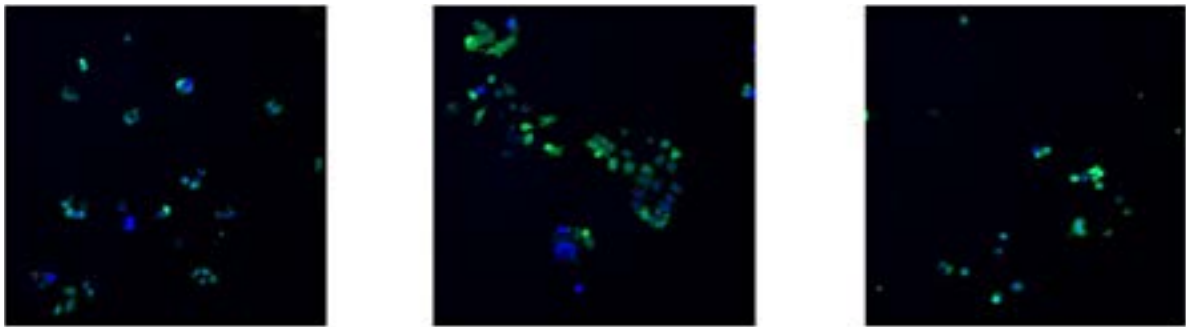


Cells without stressor **Cells stressed only** **Cells stressed + Nrf2 (US)**

Figure 11A-C. This depicts Caco-2 cells under a fluorescent microscope at baseline (A.), under stress treatment with hormone disruptors (phthalates) (B.), and stressed cells after treatment with Protandim Nrf2 Synergizer (C.). During stress treatment, cells turn more fluorescent green to indicate oxidative stress (ROS) is generated. Panel C shows a reduction in oxidative stress (less green fluorescence) with the addition of Protandim Nrf2 Synergizer.*

Caco-2: Methylglyoxal

A. **B.** **C.**



Cells without stressor **Cells stressed only** **Cells stressed + Nrf2 (US)**

Figure 12A-C. This depicts Caco-2 cells under a fluorescent microscope at baseline (A.), under stress treatment with methylglyoxal stimulating ultra-processed foods (B.), and stressed cells after treatment with Protandim Nrf2 Synergizer (C.). During stress treatment, cells turn more fluorescent green to indicate oxidative stress (ROS) is generated. Panel C shows a reduction in oxidative stress (less green fluorescence) with the addition of Protandim Nrf2 Synergizer.*

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After evaluating the fluorescent microscopy images, we decided to run further studies to quantify the ROS intensity in fibroblasts and HepG2 cells. Table 5 summarizes the decrease in ROS intensity seen in the images for HepG2 and fibroblast cells after the addition of Protandim Nrf2 Synergizer as compared to stressed cells.

Table 5. Decrease in ROS intensity after the addition of Protandim Nrf2 Synergizer to stressed cell culture.

Stressor	HepG2	Fibroblasts
Pollution	-158%	-44%
Pesticides	-181%	-120%
Hormone disruptors	-92%	-87%
Microplastics	-80%	-59%
Ultra-processed food	-70%	-44%

Discussion & Conclusion

Oxidative stress is one of several interconnected biological processes that can contribute to the accumulation of cellular damage, declining cellular resilience, and age-related changes in tissue function, such as cardiovascular, eye, brain, and skin tissue. Excessive oxidative stress can contribute to lipid oxidation, endothelial dysfunction, mitochondrial energy decline, and altered immune-cell signaling.

Cells experience ROS stress every day through normal biological processes, even without external factors. However, environmental and lifestyle stressors increasingly add to the load. Exposure to many of these toxins is so ingrained into our lives, they would be next to impossible to eliminate. Regulations, manufacturing practices, and consumer choices are improving with greater awareness, but the exposures are unlikely to disappear entirely.

Over a lifetime, chronic exposure to these stressors can contribute to accumulating oxidative damage that may negatively impact biological processes associated with long-term health and aging.

Protandim Nrf2 Synergizer was developed to activate the NRF2 pathway to significantly increase the production of antioxidant enzymes to combat elevated ROS and reduce oxidative stress caused by internal and external triggers. A 2006 study by Nelson et al showed a 40% decline in oxidative stress in 30 days of supplementing with Protandim Nrf2 Synergizer. *†

In the current study, we further investigated the beneficial effects of Protandim Nrf2 Synergizer on oxidative stress associated with modern toxins our bodies are exposed to every day: microplastics, pollution (fine particulate matter), hormone disruptors (phthalates), pesticides (glyphosates), and ultra-processed food/diet (methylglyoxal).

Liver, skin, and gut cells were chosen as in vitro cell models because they are our first line of defense from toxin exposures. The viability data showed how damaging all five stressors are on the three cell types. At certain exposure levels, they cause cell death. Lower exposures cause considerable damage that, over time, would negatively impact overall health.

The strongest results were observed in stressed HepG2 cells and fibroblasts after treatment with Protandim Nrf2 Synergizer.

The largest reductions in oxidative stress were seen in stressed HepG2 cells for all toxins. (Table 5) This is consistent with the liver's central role as the body's major metabolic and detoxifying organ. HepG2 liver cells have extensive capacity for dealing with oxidative stress-causing chemicals and byproducts.

After adding Protandim Nrf2 Synergizer to stressed HepG2 cells, we saw decreases in ROS intensity by -158% (pollution), -181% (pesticides), -92% (hormone disruptors), -80% (microplastics), and -70% (methylglyoxal)

Fibroblasts respond to oxidative stress through stress adaptation, damage control, and cellular repair. After adding Protandim Nrf2 Synergizer to stressed fibroblast cells, we saw decreases in ROS intensity by -44% (pollution), -120% (pesticides), -87% (hormone disruptors), -59% (microplastics), and -44% (methylglyoxal).

The greater magnitude of ROS reductions in HepG2 cells suggests that liver cells may be more responsive to Protandim Nrf2 Synergizer than fibroblasts. The results for pollution and pesticides also indicate that Protandim Nrf2 Synergizer not only eliminated the impact of toxin exposure, it also helped oxidative stress levels fall below the unstressed baseline. *

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Collectively, these findings demonstrate reductions in ROS across multiple cell types and environmental and dietary stressors. This may help explain, in part, the dramatic reduction in age-related oxidative stress observed in the earlier Nelson et al. 2006 study. *

You may not feel oxidative stress changes happening inside your cells. But over time, supporting your body's antioxidant defenses can help protect them from oxidative stress caused by daily exposure to multiple environmental toxins. These study results show how Protandim Nrf2 Synergizer can be an important way to maintain long-term cellular health and support your energy, exercise recovery, resilience to challenges, healthier looking skin, and overall well-being. *

References

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† Results from a human clinical study on Protandim NRF2 Synergizer showed reduction of oxidative stress by 40% in 30 days.

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