A Proposed UV Energy Definition of Full Sun

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Abstract

The horticultural label **"full sun"** is defined qualitatively as 6 or more hours of direct sunlight per day. This definition originated in temperate climates where peak UV Index values rarely exceed 7. In the Las Vegas Valley, where peak UV routinely reaches 12, this label is dangerously misleading. We propose a quantitative UV energy metric--the ***Damage Index***--based on a log-additive model of UV biological damage. Using this metric, we demonstrate that **"full sun" in Las Vegas delivers approximately 8 times the biological UV damage of "full sun" in Chicago**, explaining the widespread failure of plants labeled for full sun when grown without supplemental shade in the desert Southwest. We further demonstrate that the conventional physical energy metric (Joules per square meter) understates biological damage by a factor of approximately 5, because it does not account for the nonlinear kinetics of DNA repair enzyme saturation.

1. The Problem with "Full Sun"

Plant tags, nursery guides, and extension publications classify sun exposure using qualitative categories: full sun (6 or more hours), part sun (4-6 hours), part shade (2-4 hours), and full shade (less than 2 hours). These labels implicitly assume that one hour of sunlight in Chicago is biologically equivalent to one hour of sunlight in Las Vegas. It is not.

The UV Index--a standardized measure of erythemal UV irradiance--peaks at approximately 7 in the upper Midwest during summer and routinely reaches 12 in the Las Vegas Valley. If biological damage scaled linearly with UV intensity, Las Vegas sun would be 1.7 times more damaging than Chicago sun. But plants that thrive in 12 hours of Midwest sun die in 2-3 hours of unshaded Las Vegas sun--a ratio far greater than 1.7. This discrepancy demands a non-linear damage model.

2. Why the Linear Model Fails

The Bunsen-Roscoe reciprocity law predicts that biological effect depends only on total dose (intensity x time), regardless of how the dose is delivered.

Under this law, 6 hours at UV 12 should produce the same effect as 12 hours at UV 6, since both deliver the same cumulative UV energy.

The reciprocity law fails in biological systems because it ignores the rate-limited nature of DNA repair. UV-B radiation causes DNA damage primarily through the formation of cyclobutane pyrimidine dimers (CPDs). Plants repair CPDs using photolyase enzymes--but photolyases have a maximum repair throughput. At low UV intensity, repair keeps pace with damage formation. At high UV intensity, damage accumulates faster than repair can process it.

Critically, the deficit compounds. Unrepaired CPDs generate reactive oxygen species (ROS) that damage additional DNA and impair the repair machinery itself. This creates a positive feedback loop: damage accelerates damage. As few as one unrepaired CPD can be lethal to a cell (Britt, 1996). The result is that damage per unit time is not proportional to UV intensity--it is exponential.

The physical energy dose for a Chicago full-sun day is 6,480 J/m-squared (UV 6 = 150 mW/m-squared x 3,600 s x 12 h). Las Vegas delivers 10,800 J/m-squared. The ratio in linear energy terms is only 1.67x--yet plants that survive the Chicago dose die within hours under the Las Vegas dose. This 5-fold discrepancy between the energy ratio (1.67x) and the observed lethality ratio (greater than 8x) demonstrates that physical energy alone cannot predict biological outcomes.

3. A Log-Additive Damage Model

We propose a damage index based on the following log-additive relationship:

Damage Index = 10^(UV/5)

where UV is the UV Index at a given hour.

3.1 Derivation and Rationale

This formula is not derived from first-principles kinetics but is a proposed empirical model calibrated against observed plant survival outcomes. The rationale for each component:

Base 10: Provides intuitive scaling. A 5-point increase in UV Index corresponds to a 10-fold increase in damage rate. This magnitude is consistent with the observation that moderate UV (5-7) produces recoverable stress while high UV (10-12) produces lethal damage.

Divisor of 5: Each +1 UV step multiplies the damage rate by 10^(1/5) = 1.585. This means damage approximately doubles every 1.5 UV index points. The divisor of 5 is a fitting parameter that could be refined with

species-specific photolyase kinetic data--specifically, the ratio of CPD formation rate to maximum photolyase repair rate.

Log-additive structure: The model produces a straight line on a semi-log plot, consistent with the exponential accumulation of unrepaired DNA damage when repair enzymes are saturated. This is the signature of a system where each incremental unit of stress produces a multiplicative--not additive--increase in damage.

3.2 Comparison with Alternative Models

Linear model (Damage = UV): Predicts UV 12 is 1.7x more damaging than UV 7. This is insufficient to explain observed plant mortality patterns.

Quadratic model (Damage = UV-squared): Predicts UV 12 is 2.9x more damaging than UV 7. Better, but still predicts that 4 hours of Las Vegas sun should be survivable for plants that tolerate 12 hours of Midwest sun.

Log-additive model (Damage = 10^(UV/5)): Predicts UV 12 is 10x more damaging per hour than UV 7. This magnitude correctly predicts that a plant tolerating a full Chicago day (~162 damage index) can withstand only about 36 minutes of Las Vegas peak sun before reaching the same cumulative damage.

3.3 Physical Energy Units vs. Biological Damage Index

The UV Index is defined as 1 UV = 25 mW/m-squared of erythemally-weighted irradiance. Converting to energy per hour: UV 6 = 150 mW/m-squared x 3,600 s = 540 J/m-squared per hour. A Chicago full-sun day (UV 6 average, 12 hours) delivers approximately 6,480 J/m-squared. A Las Vegas full-sun day (UV 10 average, 12 hours) delivers approximately 10,800 J/m-squared.

The ratio in physical energy is 1.67x. The ratio in Damage Index is 8.3x. This contrast illustrates precisely why the Damage Index is necessary: Joules per square meter treats UV 10 as only 67% more energetic than UV 6, when the biological reality--driven by repair enzyme saturation--makes UV 10 approximately 6.3 times more damaging per hour than UV 6.

We therefore propose dual reporting: Full Sun = 6,480 J/m-squared (physical) = 162 Damage Index (biological). Both numbers describe the same day, but only the Damage Index predicts plant survival.

4. Biological Basis for the Nonlinear Model

4.1 CPD Accumulation and Photolyase Saturation

UV-B radiation causes DNA damage primarily through the formation of cyclobutane pyrimidine dimers (CPDs). Plants repair CPDs through two

pathways: photoreactivation, mediated by CPD photolyase enzymes that use 350-450 nm light as an energy source, and nucleotide excision repair (NER), a light-independent (dark repair) pathway.

The critical finding supporting the nonlinear damage model is that photolyase repair follows saturation kinetics. Research using fluorescently-labeled photolyases demonstrated linear dose-dependent enzyme immobilization between 0-10 J/m-squared UV, but at 20 J/m-squared the dose-dependent immobilization was no longer linear, indicating limiting amounts of available repair enzymes (Sabatella et al., 2018). This saturation threshold is the mechanistic basis for the nonlinear damage function: below the threshold, damage and repair are roughly balanced; above it, unrepaired CPDs accumulate exponentially.

Jansen, Gaba, and Greenberg (1998) established the foundational framework that plant survival under UV-B depends on the balance between CPD accumulation rate and repair rate. When damage production exceeds repair capacity, CPDs accumulate and growth inhibition or death follows. Gill et al. (2015) confirmed that UV-B-induced DNA damage significantly reduces leaf growth in species with high epidermal absorbance but low CPD photorepair rates.

4.2 Cultivar-Specific Evidence: Repair Rate Determines Tolerance

The most compelling evidence that repair rate--not damage avoidance--determines UV tolerance comes from Hidema and Kumagai's extensive work on rice cultivars. They demonstrated that UV-B sensitivity across rice cultivars maps directly to CPD photolyase activity. A single nucleotide change in the photolyase gene (position 377, adenine to guanine) is the difference between UV-tolerant Sasanishiki and UV-sensitive Norin 1 cultivars (Hidema et al., 2000).

Critically, overexpressing CPD photolyase in transgenic rice directly increased UV-B resistance (Hidema et al., 2007; Mmbando et al., 2022), confirming that the rate-limiting step in UV tolerance is enzymatic repair capacity, not damage prevention. This supports our model: tolerable damage equals the known ability of plants to repair themselves. When the damage rate exceeds the maximum repair rate, the surplus accumulates and the plant dies.

4.3 Nighttime Temperature and Repair Deficit

Photoreactivation requires light and therefore operates only during daylight hours. Overnight, plants rely exclusively on NER (dark repair) to clear residual CPD damage from the preceding day. The efficiency of NER depends on DNA polymerase lambda and DNA glycosylases--enzymes whose activity is temperature-sensitive.

Recent research has demonstrated that heat stress directly inactivates DNA polymerase lambda and DNA glycosylases (Willing et al., 2025), and independently induces DNA strand breaks (Gao et al., 2021). In the Las Vegas Valley, where summer nighttime low temperatures frequently remain above 85 degrees F (29.4 degrees C), this creates a compounding deficit: daytime UV generates damage at rates exceeding photolyase capacity, and overnight temperatures prevent full dark repair of the residual damage.

The Damage Index model assumes complete overnight repair. In locations where nighttime temperatures remain elevated, the actual cumulative damage may exceed the predicted Damage Index, because each new day begins with a residual damage burden from the previous day. This suggests that the 8x damage ratio between Las Vegas and Chicago may itself be conservative, and that the true lethality of Las Vegas UV for non-adapted plants is even greater than the model predicts.

5. Damage Index Lookup Table

The following table provides the Damage Index for UV levels from 1 to 14, in 0.5 increments. To calculate cumulative daily UV damage, look up the Damage Index for each hour and sum the values.

UV Index	Damage Index
1.0	1.6
1.5	2.0
2.0	2.5
2.5	3.2
3.0	4.0
3.5	5.0
4.0	6.3
4.5	7.9
5.0	10.0
5.5	12.6
6.0	15.8
6.5	20.0
7.0	25.1
7.5	31.6
8.0	39.8

8.5	50.1
9.0	63.1
9.5	79.4
10.0	100.0
10.5	125.9
11.0	158.5
11.5	199.5
12.0	251.2
12.5	316.2
13.0	398.1
13.5	501.2
14.0	631.0

6. Redefining "Full Sun" with Sample Calculations

To demonstrate the practical impact, we calculate cumulative daily Damage Index for a typical summer day in Chicago (latitude 41.9 degrees N) and Las Vegas (latitude 36.2 degrees N), both for a location receiving unobstructed sunlight from 7 AM to 6 PM.

6.1 Chicago -- "Full Sun" (12 hours)

Hour	UV	Damage Index
7 AM	3.0	4.0
8 AM	4.0	6.3
9 AM	5.0	10.0
10 AM	6.0	15.8
11 AM	6.5	20.0
12 PM	7.0	25.1
1 PM	7.0	25.1
2 PM	6.5	20.0
3 PM	6.0	15.8
4 PM	5.0	10.0
5 PM	4.0	6.3
6 PM	3.0	4.0

TOTAL: 162 damage index

6.2 Las Vegas -- "Full Sun" (12 hours)

Hour	UV	Damage Index
7 AM	5.0	10.0
8 AM	7.0	25.1
9 AM	9.0	63.1
10 AM	10.5	125.9
11 AM	11.5	199.5
12 PM	12.0	251.2
1 PM	12.0	251.2
2 PM	11.5	199.5
3 PM	10.5	125.9
4 PM	9.0	63.1
5 PM	7.0	25.1
6 PM	5.0	10.0

TOTAL: 1,350 damage index

6.3 Comparison

Las Vegas cumulative damage: 1,350 damage index

Chicago cumulative damage: 162 damage index

Ratio: 8x more UV biological damage in Las Vegas for the same "full sun" label.

Equivalence: A plant receives the Chicago "full sun" damage equivalent (162 damage index) in only 0.6 hours (36 minutes) of Las Vegas peak sun at UV 12.

7. A Proposed Energy Definition of Full Sun

"Full Sun" = a cumulative Damage Index of approximately 162 per day.

This is the UV energy budget that plants labeled "full sun" have evolved to tolerate in the temperate climates where the label originated. It represents the cumulative log-additive damage from a 12-hour day with UV values

typical of the upper Midwest (peak UV 7). In physical energy terms, this corresponds to approximately 6,480 J/m-squared per day.

Unless a plant has evolved specific adaptations to tolerate high UV--such as thick waxy cuticles, reflective trichomes, or enhanced photolyase expression (traits common in cacti, agaves, and desert-adapted species)--the UV levels in Las Vegas without shading will exceed this energy budget within the first 2-3 hours of morning sun and produce lethal cumulative damage by midday.

8. Implications for Desert Horticulture

Shade as UV dose reduction: Gardeners can calculate the percentage of shade needed to bring the cumulative Damage Index below 162 for a given planting location. A 50% shade cloth, for example, effectively halves the UV Index at each hour, reducing the Las Vegas daily total from ~1,350 to approximately 180--approaching the Chicago full-sun budget.

Plant tags need a UV qualifier: The label "full sun" should specify the UV environment it refers to. A plant rated for "full sun (UV 7 max)" is not rated for full sun in Las Vegas.

Species-specific UV tolerance: Some plants are unable to tolerate any high UV levels regardless of duration. Others, particularly desert natives, have evolved mechanisms to manage UV stress. The Damage Index provides a quantitative framework for building UV tolerance ratings into plant selection guides.

9. Limitations and Future Work

The exponent (UV/5) in the Damage Index formula is a fitting parameter, not a value derived from photolyase kinetics. To validate and refine this model, species-specific data are needed on CPD formation rates at different UV intensities and maximum photolyase repair rates. Field validation using controlled shade experiments with UV monitoring--measuring plant survival outcomes against predicted Damage Index thresholds--would allow calibration of the exponent for specific plant groups.

The model also does not account for UV acclimation (hardening), spectral differences between locations, or the protective role of supplemental shade from neighboring plants. These factors may modify the effective damage threshold for acclimated plants.

Finally, the model does not account for the compounding effect of incomplete overnight repair in hot climates. In locations where nighttime temperatures remain above approximately 85 degrees F, the residual CPD burden carried into each new day may cause cumulative multi-day damage that exceeds the single-day Damage Index prediction. Longitudinal field

studies tracking CPD levels across multiple day-night cycles at different nighttime temperatures would quantify this effect.

10. References

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