



MURBURN CONCEPT (2025): ECOLOGY-BIODIVERSITY-EVOLUTION AND OTHER UPDATES

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*Although ecological processes have incorporated spontaneous chemico-physical processes resulting from stochastic interactions of molecules, biology has traditionally been interpreted through deterministic frameworks emphasizing ordered molecular interactions, tightly regulated enzymatic pathways, and hierarchical control mechanisms. However, increasing evidence across bioenergetics, bio-signaling, immuno/mechano/electro-physiology, xenobiotic metabolism, etc. indicates that stochastic, (non) equilibrium processes play a decisive role in shaping biological outcomes. Murburn concept, which posits that several life processes are also fundamentally driven by probabilistic interactions involving molecules, ions, radicals, and radiation, offers a unifying framework for understanding these phenomena. Originally developed to explain bioenergetic and metabolic processes, murburn principles naturally extend to ecological and biodiversity dynamics. This essay explores how murburn processes influence ecosystem-level turnover, elemental cycling, biodiversity generation, and ecological integration, arguing that murburn chemistry represents a foundational principle linking molecular randomness to macroscopic ecological order and evolution thereof. Further, I also present (i) a short snapshot of the various serious citations over the last several years on murburn, and (ii) discuss the ways to advance the research and outreach prospects of murburn concept. **Biomed Rev 2025; 36: 17-29***

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1. FROM MOLECULAR DETERMINISM TO EVOLVABLE BIODIVERSITY AND STOCHASTIC ECOLOGY

Classical biology has long relied on deterministic metaphors: lock-and-key enzymes, linear metabolic pathways, genetic blueprints, and tightly controlled regulatory circuits. Ecology, in turn, has inherited similar assumptions: predictable trophic cascades, equilibrium states, and stable cycles of matter and energy. While these models have been heuristically valuable, they increasingly fail to explain: (a) high variability in biological outcomes under similar conditions, (b) nonlinear responses of ecosystems to perturbations, (c) emergence of biodiversity from minimal genetic compositions/differences, and (d) resilience and adaptability in fluctuating environments.

Murburn concept challenges and complements deterministic reductionism by asserting that life is sustained by stochastic chemical interactions mediated by diffusible reactive species (DRS), particularly radicals and excited molecular states. It posits that these processes are not noise or side reactions but constitute the primary operational chemistry of living systems. If murburn chemistry underlies metabolism and physiology,

as a natural consequence, it must also influence ecology, which is ultimately the collective behavior of biochemical systems interacting with their environment. Also, ecological components are also made of molecules and ions, quite like biological systems. Although the carbon and halogen cycling in lignocellulosics and organohalogenics (respectively) are strongly attributed (as discussed in some earlier communications (1)) to murburn activity (in particular, heme peroxidases), the impact of murburn processes on biodiversity and ecology have not been discussed to any significant extent in any publications yet. This topic was touched upon in the National Conference on “Climate & Conservation: A dual approach to ecological resilience” (Figure 1) held at Govt. College, Chittur (Kerala, on 16th & 17th of October, 2025); in the plenary scientific lecture delivered by the author, “*Murburn concept & Life: The foundation of photosynthesis, biodiversity and ecology-dynamics*”. The first part of this writing elaborates upon the contents (and more) presented therein. The second and third parts shall briefly discuss the emerging discussions on murburn concept in some articles published and explore the scope/prospects of reaching out to the scientific community to enhance murburn-awareness.

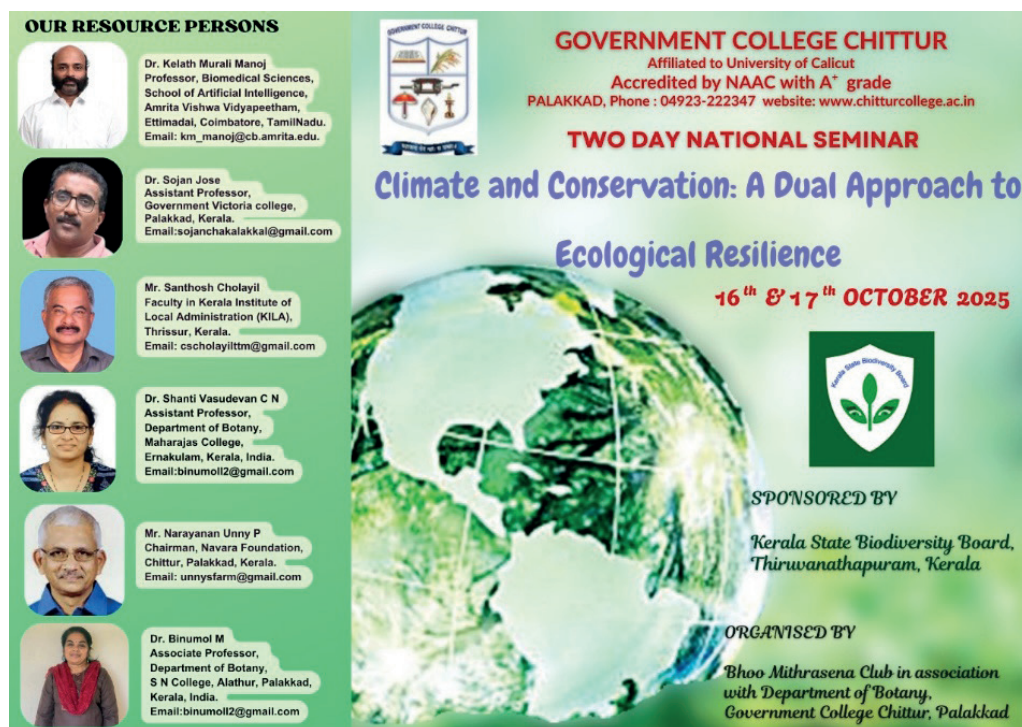


Figure 1. The flyer of the conference event at Chittur (Kerala) where murburn concept's relevance in biodiversity and ecology was originally propounded. The invitation to this meeting was extended to the author by the Organizing Secretary, Dr. Suresh Kumar KA, Head of the Department of Botany, Govt. College, Chittur, Palakkad.

2. CORE PRINCIPLES OF MURBURN CONCEPT

Murburn is no hypothesis, but is a collection of ascertainable facts (2) on which models can be built for understanding/explaining specific cases of metabolic/physiological/organellar/cellular function (3-27). It emphasizes:

2.1. Stochasticity

Biological outcomes emerge from probabilistic molecular interactions rather than predetermined sequences and mandates.

2.2. Radical-mediated chemistry

Reactive oxygen, halogen, and nitrogen species act as functional intermediates, not merely toxic byproducts.

2.3. (Non)equilibrium dynamics

Living systems may operate far from thermodynamic equilibrium, but yet are subject to equilibrium forces/dictates. No molecule can violate such mandates of the established laws of physics! (Thereby questioning ideas such as ion pumps, proton motive force, bidirectional high-throughput selective ion-channels, etc.!).

2.4. Diffuse reaction zones (murzones)

Chemistry occurs in loosely confined spaces rather than rigid active sites, and murzymes (molecules that generate, modulate, buffer or utilize DRS) mediate the fundamental bioenergetic process of effective charge separation (ECS).

2.5. Energy dissipation as function

Heat, radiation, mechanical perturbations, electrical activity and entropy production are integral to biological work.

2.6. Biological intelligence as ‘emergent’ outcome

The ECS-DRS murburn mechanism results in a chemico-electromagnetic matrix (CEM) that enables the powering, coherence, electromechanical and sensing-response (PCHEMS) activities of cells, which work as simple chemical engines (SCE). The intelligence of life (from unicellular decision-making processes like that of motility to complex decisions like the ones made by the neuronal system in humans) is primarily electronic in this view (as opposed to ‘resident’ active and ionic in classical perception).

These principles naturally scale upward from molecular enzymatic reactions and lattice structure effects to macroscopic outcomes in tissues, organisms, populations, and ecosystems (22,23).

3. MURBURN PROCESSES AND ELEMENTAL CYCLING IN ECOSYSTEMS

The fundamental idea is that the murburn redox “tone” or “landscape” does not stop at the cell membrane. Diffusible reactive oxygen and electrophilic species (ROS/RES) can cross boundaries, influencing the immediate extracellular environment. This creates a web of chemical communication and stress that connects organisms to each other and to their abiotic surroundings.

3.1. Oxygen and redox ecology

Oxygen is simultaneously both, life-sustaining and destructive. Traditional models struggle to reconcile this duality. Murburn chemistry resolves this paradox by recognizing oxygen as a controlled radical mediator, whose partial reduction generates reactive intermediates essential for energy transduction, signaling, environmental detoxification, organic matter turnover, etc. At the ecological level, oxygen gradients shape soil microbiomes, aquatic stratification, sediment chemistry, plant-microbe interactions, etc.

Murburn-driven redox chemistry explains why micro-oxic and fluctuating oxygen niches often exhibit the highest biodiversity. A step-by-step (from chemistry → physiology → ecology → biodiversity → evolution) understanding is provided below, along the murburn framework.

A. Empirical ecological observation (starting point): Across ecosystems, the highest biodiversity is often observed not at oxygen extremes, but in micro-oxic or fluctuating oxygen (and ion) niches, such as: soil aggregates, rhizospheres, wetland sediments, estuarine interfaces, microbial mats, biofilms, animal gut microenvironments, etc. This pattern is robust and well documented, yet poorly explained by classical models.

B. Why classical explanations are insufficient: Classical redox / respiration models assume that oxygen is either present or absent and metabolism switches cleanly between aerobic respiration, anaerobic respiration and fermentation. These models predict competitive exclusion, not diversity and they do not explain coexistence of aerobes, facultative anaerobes, microaerophiles, obligate anaerobes, rapid adaptation to oxygen fluctuations, high mutation and innovation rates in these niches, etc. Thus, the diversity peak at intermediate oxygen remains mechanistically underexplained.

C. Murburn’s core chemical insight: Oxygen is not a binary terminal electron acceptor; it is a stochastic redox mediator whose partial reduction produces a spectrum of reactive species. In micro-oxic or fluctuating niches, (i) oxygen

concentration is low but non-zero, (ii) diffusion maybe limited, and (iii) redox gradients are steep and dynamic. This would generate a continuous production of oxygen-centric and other-element centric DRS, which act as localized and probabilistic reaction drivers. Under murburn logic, micro-oxia leads to-

(a) maximum redox heterogeneity, multiple oxidation states coexist, no single redox pathway dominates, radical lifetimes are short but frequent, delocalized reactions occur outside strict enzyme active sites and environmental contexts can strongly influence outcomes.

(b) energetic non-linearity results from small oxygen changes giving large functional effects, which could lead to non-monotonic “fitness landscapes”. This creates many viable chemical strategies, not one optimal solution.

D. Physiological consequences (organism level): As a consequence, organisms in micro-oxic niches experience- (a) multiple viable metabolic modes (partial aerobic metabolism, mixed redox coupling, opportunistic electron sinks, and no single metabolism fully dominates. Thus, micro-niche partitioning could result. (b) some organisms can develop tolerance and utilization of different types of DRS, exploit them for signaling or metabolism and others specialize in scavenging, and yet others avoid or compartmentalize! Each strategy is locally adaptive, none globally optimal. (c) elevated but survivable DRS-induced mutagenesis could also result at spatially restricted regions, accelerating phenotypic exploration without lethality.

E. Ecological consequences (community level): The above processes lead to breakdown of competitive exclusion as no single redox strategy is optimal across spatio-temporal barriers. Environmental fluctuations continually reshuffle advantages. The creation of redox-defined micro-niches/gradients allow for different levels of scale specialization and coexistence of closely related species that work along the feedback between organisms operating under networked chemistry, by modalities including oxygen consumption, DRS production and metabolite release. This self-organizes dynamic ecological mosaics.

F. Evolutionary consequences (longer timescales): Ultimately, the above consequences lead to accelerated diversification, higher mutation rates (environmentally biased mutagenesis) and rapid fixation of adaptive variants. Also, fluctuating selection prevents allele fixation and balancing selection emerges naturally, resulting in the maintenance of genetic diversity, and emergence of novelty from the CEM (radical/radiative chemistry- unusual chemical reactions, non-canonical pathways, new metabolic linkages, etc.). Innovation

is chemically enabled, not just “genetically permitted”.

In stark contrast, let us see how the extremes of oxygen availability reduce diversity. In fully oxic environments, strong oxidative pressure would favor robust antioxidant systems with high metabolic viability with fewer viable strategies. In strictly anoxic environments, limited oxygen-water recycling would lower metabolic rates/diversity and afford limited redox repertoire. Thus, biodiversity peaks between extremes. In short, murburn-driven redox chemistry provides a mechanistic explanation for the empirical observation that micro-oxic and fluctuating oxygen niches harbor maximal biodiversity. In such environments, partial and stochastic reduction of oxygen generates a heterogeneous spectrum of reactive intermediates that create dynamically shifting redox landscapes. These conditions preclude the dominance of a single metabolic strategy, promote niche partitioning at fine spatial and temporal scales, and elevate adaptive mutagenesis without catastrophic damage. Consequently, micro-oxic systems function as evolutionary incubators, where chemical heterogeneity sustains ecological coexistence and accelerates diversification. Therefore, biodiversity peaks where oxygen is sufficient to generate redox complexity but insufficient to enforce metabolic uniformity.

3.2. Halogen cycling and chloroperoxidase

Halogens play a disproportionate role in ecological chemistry despite their low abundance. Enzymes such as chloroperoxidase catalyze reactions that DRS through murburn mechanisms and are responsible for more than 90% of the naturally formed organohalogenics in the environment (1). The ecological consequences include- antimicrobial defense in marine and terrestrial organisms, modulation of microbial community structure, halogenated organic compound turnover, atmospheric chemistry via volatile halogen release, etc. These reactions are not tightly substrate-specific but depend on local electron source and ionic availability, pH, and environmental context; which are typical hallmarks of murburn processes.

3.3. Carbon-cycling and lignocellulosic degradation

Besides murburn being the fundamental logic of photosynthesis and nitrogen fixation (24), its roles in carbon cycle is also well documented. The degradation of lignin and cellulose is among the most complex processes in nature. Classical enzymology fails to fully account for the reach/invasion of microbial growth into the dense macromolecular frameworks, non-specific bond cleavage, high efficiency under mild conditions and synergistic microbial consortia that participate. Murburn chemistry explains lignocellulosic decay as a radical-driven

process, where diffusible reactive species generated by fungi and bacteria mediate extracellular oxidative depolymerization. Ecologically, this enables relatively rapid carbon turnover, nutrient release, enriched soil formation, and atmospheric CO₂ regulation. Thus, murburn processes underpin the global carbon cycle, linking molecular chaos to planetary homeostasis.

4. MURBURN DYNAMICS, BIODIVERSITY GENERATION AND EVOLUTION

Traditional evolutionary biology explains biodiversity through genetic mutations and natural selection. Murburn does not replace this but adds a crucial, dynamic, and immediate layer to it. It explains biodiversity by providing a mechanism for rapid, non-genetic phenotypic diversity upon which natural selection can act. The traditional view is that the genotype (DNA) rigidly dictates the phenotype (the observable traits). Murburn suggests that the phenotype is not a direct, one-to-one readout of the genotype. Instead, the genotype sets the boundaries for a “reaction chamber” where murburn dynamics play out. The actual realized phenotype is a result of the interplay between the genetic blueprint and the stochastic redox chemistry happening within the cell.

4.1. Mutation as a stochastic outcome

Stochasticity as an engine of variation: Mutations are often portrayed as rare replication errors. The very nature of murburn is probabilistic. The generation of DRS and other diffusible reactants is not perfectly controlled. Their interactions with cellular components are random “hits.” This means that even in two genetically identical cells in the same environment, the murburn-mediated processes will have slightly different outcomes. This intrinsic stochasticity introduces phenotypic variation at a fundamental biochemical level. DRS generated during murburn processes can also induce base modifications, strand breaks, transposable element activation, etc.; changes that are environmentally contingent, linking ecological stressors directly to genomic variability. Thus, murburn chemistry provides a mechanistic bridge between environment and evolution.

The diffuse, network-based nature of murburn regulation is assisted with a complex system of buffers, antioxidants, and repair mechanisms. This could explain the evolution of immense genomic complexity. A lot of non-coding DNA might code for RNAs and proteins that fine-tune this redox landscape, manage its stochasticity, and repair the damage it causes. Therefore, biodiversity is not just in the “working

parts” but also in the vast and varied “control systems” that manage murburn chemistry.

4.2. Epigenetic and post-translational modulations

DRS can also bring about DNA methylation patterns, histone modifications, and chromatin accessibility. Because murburn processes respond to environmental conditions, epigenetic changes become context-sensitive memory systems, enabling organisms to adapt across generations without altering DNA sequence. This has profound implications for phenotypic plasticity, rapid adaptation, and ecosystem resilience. Also, oxidation of moieties, phosphorylations and glycosylations of proteins can result from murburn modifications (19), thereby altering protein activity (and metabolic alterations thereof!) in the cellular context.

A genetic mutation might take generations to spread. However, a change in the murburn “tone” or “landscape” of a cell (e.g., due to a change in diet, oxygen levels, or toxin exposure) can immediately alter the rates of thousands of reactions simultaneously. This can lead to rapid physiological adaptation within a single generation. Populations with a wider range of murburn-mediated responses are pre-adapted to handle environmental shifts, increasing their chances of survival and diversification.

4.3. Speciation and ecological niches

The constant, low-level stochastic damage and modification of biomolecules by murburn products create a diverse pool of slightly altered molecules within a population. This is like the cell continuously running millions of tiny experiments. Most have no effect, but some lead to a protein with a slightly new function or a metabolic pathway with a slightly different efficiency. This increases the raw material upon which natural selection can act. Stochastic biochemical variation allows populations to explore phenotypic space efficiently. Over time, ecological selection stabilizes certain variants, leading to niche specialization, reproductive isolation and speciation. Thus, biodiversity emerges not from deterministic design but from murburn-driven exploration constrained by ecological feedback.

Different species have different metabolic rates, lifespans, and environmental niches. Murburn provides a plausible mechanism for this. For instance: A species that evolves a slightly more efficient mitochondrial complex might generate a different profile of ROS, leading to a different set of regulated genes and metabolic outcomes. This could nudge a population towards a new dietary preference or a different tolerance

for oxygen, eventually leading to reproductive isolation and speciation. Table 1 captures the basic essence of the murburn contributions towards biodiversity and evolution.

Table 1. Contribution of murburn to biodiversity and evolution

Aspect	Traditional explanation	Murburn contribution
Source of variation	Primarily random genetic mutations.	Adds stochastic biochemical variation from redox-based reactions.
Level of action	Focus on modulation of genes to influence proteins.	Focus on systems-level network dynamics, bioenergetics and diffusible chemical messengers.
Role of genetics	DNA is the direct blueprint for the phenotype.	DNA sets the stage, but murburn dynamics influence the real-time performance of the phenotype.
Pace of change	Slow, over generations.	Provides a mechanism for rapid, within-generation physiological adaptation.

In short, murburn theory contributes to our understanding of biodiversity by proposing that the inherent randomness and diffusibility of fundamental life processes are not a bug, but a feature. This stochasticity at the core of cellular energetics provides a continuous and rich source of non-genetic variation, accelerating evolutionary processes and contributing to the magnificent diversity of life we see today.

5. ECOSYSTEM-LEVEL INTEGRATION VIA MURBURN PROCESSES

5.1. Distributed control and robustness

Ecosystems lack centralized control, yet display remarkable stability. Murburn chemistry explains this via redundant pathways, distributed reaction networks and feedback through reactive intermediates. Such systems are robust precisely because they are noisy, allowing adaptation rather than collapse.

5.2. Energy dissipation and trophic structure

Energy flow through ecosystems is not perfectly efficient. Heat loss, radical quenching, and entropy production are not waste but essential features that prevent runaway reactions, enable trophic stratification, and maintain ecosystem boundaries.

Symphony Orchestra Vs. Jazz Band

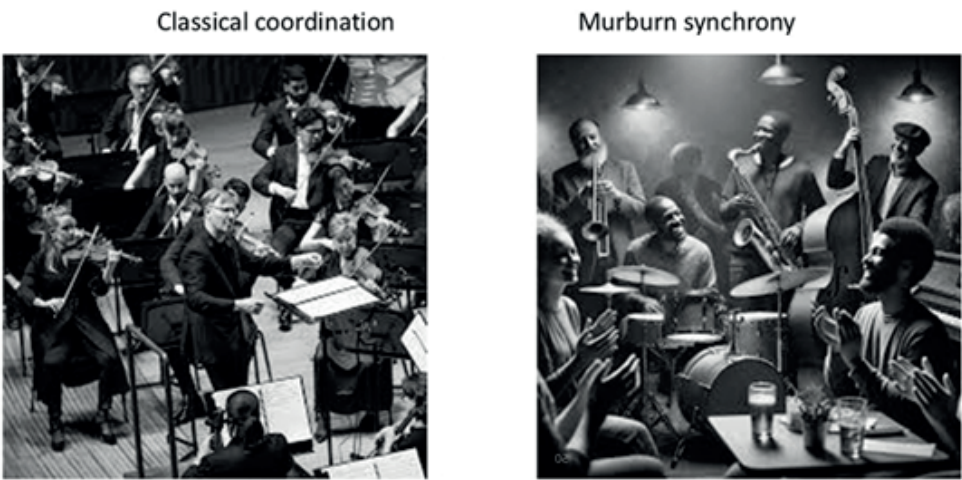


Figure 2: On the left panel is an analogy to the traditional view (lock-and-key) of cellular metabolism/physiology. The cell is like a classical symphony orchestra. Every musician (enzyme) has a specific, written part (substrate) to play, and the conductor (DNA) ensures perfect coordination. Variation comes from changing the sheet music (mutation). **The jazz band on the right panel is the murburn view (ECS-DRS) of cellular function.** There's a basic chord progression (the genetic framework). The drummer (murburn complexes) sets a stochastic rhythm (redox tone). The soloists (other cellular components) improvise based on the rhythm and what they hear around them. The final performance (phenotype) is a unique, emergent property that is never played the same way twice, even with the same musicians and chord chart. Biodiversity is the sum of all these different jazz performances across all of life.

Murburn processes frame energy dissipation as functional ecology, not inefficiency.

5.3. Cross-scale coupling

Murburn chemistry naturally couples molecular events → physiological responses, organismal behavior → population dynamics and population activity → ecosystem chemistry. This multi-scale integration explains emergent phenomena such as (A) algal blooms, (B) forest succession and (C) microbiome-driven ecosystem shifts. I shall elaborate upon this as follows:

A. ALGAL BLOOMS AS A MURBURN-DRIVEN EMERGENT PHENOMENON

Classical ecology explains algal blooms as a result of nutrient enrichment (N, P), favorable light and temperature, reduced grazing pressure. This explains when blooms occur, but not well

(i) why blooms are explosive, (ii) why certain taxa dominate, (iii) why collapse is sudden and toxic, and (iv) why micro-oxic zones precede blooms.

a. Murburn mechanism affords a molecular → physiological deontological succession.

In step 1, oxygen and light generate radical flux in surface waters due to photosynthesis (photochemical ROS like $^1\text{O}_2$, $\text{O}_2^{\bullet-}$, H_2O_2), and this also leads to O_2 supersaturation therein. In conjunction, metal ions (Fe, Mn) catalyze radical cycling, creating a murburn field leading again to partial oxygen reduction and diffusible radicals with fluctuating redox microdomains.

In step 2, the physiological asymmetry among algae start manifesting. Different algal taxa differ in ROS tolerance/anti-oxidant capacity and utilization (signaling, redox metabolism). Under moderate ROS, growth is stimulated whereas under high ROS, damage occurs. Thus,

Growth = $f(\text{ROS})$ (non-monotonic)

b. Then, the organismal → population scale dynamics follow.

In step 3, a positive feedback loop may result. Fast-growing algae increase O_2 production and such ROS selectively suppress competitors; dominant species accelerates further. This is autocatalytic dominance, not simple nutrient competition.

In step 4, sudden and massive blooms emerge once ROS-tolerant taxa dominate. Population growth becomes super-linear and toxins (often ROS-related secondary metabolites) accumulate.

In step 5, eventually, ROS exceeds tolerance threshold, oxidative stress damages even dominant taxa and massive

die-off results, leading to microbial decomposition, which consumes O_2 . This leads to hypoxia. Thus, bloom collapse is a chemically triggered phase transition, not gradual resource depletion.

The key murburn insight is that algal blooms are redox runaway phenomena, stabilized and destabilized by murburn chemistry, not merely nutrient abundance. (Let us not forget to mention that the utilization of key elemental plant nutrients “NKP” is also subject to murburn mechanisms!) Quite like vacillating redox reactions, population dynamics are subject to electron-transfer dynamics!

B. FOREST SUCCESSION THROUGH MURBURN ECOLOGY

Classical models see the succession as pioneer → intermediate → climax species; this is driven by shading, soil nutrients, competition. The missing elements are roles of oxidative chemistry, soil radical dynamics, and microbial mediation of succession.

Murburn chemistry in soil and litter: Early successional soils are oxidative. After disturbance (fire, logging), exposed soil affords high oxygen penetration, enabling metal-catalyzed ROS generation and photochemical radical formation. Organic matter breakdown is radical-mediated, not purely enzymatic. Thereafter pioneer species exploit oxidative niches, as these plants have high ROS tolerance, fast metabolism, lignin-poor tissues and rapid turnover. They thrive where radical flux is high and nutrients are rapidly mineralized. As vegetation increases

cross-scale feedback of plants ↔ soil chemistry becomes operative, and litter accumulates and oxygen diffusion decreases. ROS production shifts from oxidative to moderated and microbial communities change from oxidative decomposers to reductive/specialized consortia. Here, succession is to be seen as a redox trajectory wherein pioneer are oxic and radical-rich, intermediates are micro-oxic and balanced, and mature forest are redox-buffered and relatively low ROS. In this purview, species replacement occurs because each stage constructs the redox environment that undermines its own dominance. The emergent outcome in forest succession is not merely species replacement, but ecosystem self-redox regulation; and murburn chemistry provides the mechanism behind observed successional patterns.

C. MICROBIOME-DRIVEN ECOSYSTEM SHIFTS

This analysis applies equally well to gut, soil, coral reefs,

sediments, and rhizospheres. The classical view is ridden with the puzzle of how/why microbiome shifts occur rapidly, are often non-linear, and are disproportionate to external forcing. Classical competition models are inadequate because microbes interact chemically, not just trophically.

In the murburn model of microbial systems, microbes produce ROS deliberately and release redox-active metabolites that modulate local oxygen and radicals. The microbiome creates a self-generated chemical field from which stepwise phenomena emerge. In step 1, small perturbation (say antibiotic, diet change, temperature shift, etc.) alters ROS balance slightly. In step 2, redox tipping results as ROS crosses tolerance thresholds for some taxa, leading to the decline of some sensitive species, whereas tolerant species expand. Mutation rates increase locally due to radical exposure. In step 3, a new stable state emerges as the community reorganizes around new metabolic loops and steady states, with altered host or environmental interaction. Such processes have ecosystem-scale consequences. Examples are:

gut dysbiosis → host inflammation → altered oxygen leakage

soil microbiome shift → nutrient cycling change

reef microbiome shift → coral bleaching

Each one of the above is a chemically driven regime shift, not gradual replacement. These are emergent phenomena because of the nonlinearity, radical effects are non-monotonic, governed by feedback (organisms alter the chemistry that governs their fitness), multi-scale coupling (molecules influence populations, populations reshape chemistry), and threshold behavior (small changes → large ecological outcomes). It can be seen that classical ecology lacks these analytical and synthetic ingredients. Murburn chemistry supplies the missing causal glue that links molecular stochasticity to ecological order. Algal blooms, forest succession, and microbiome-driven ecosystem shifts are not anomalies to be patched into ecology! They are natural consequences of redox-driven, (non) equilibrium systems operating across scales.

6. MURBURN ECOLOGY AND ENVIRONMENTAL PERTURBATIONS

Among other parameters, planetary climate change primarily alters temperature, oxygen solubility and radiation exposure. These changes directly modulate murburn chemistry, leading to: altered metabolic rates, shifts in species composition, and novel ecological states. Understanding ecosystems as murburn-driven systems allows prediction of tipping points.

Anthropogenic activity like release of xenobiotics also interact with murburn pathways, often amplifying radical fluxes. This explains endocrine disruption, bioaccumulation, and ecotoxicological nonlinearity. Risk assessment must therefore move beyond classical dose-based models toward reactivity-based frameworks.

7. IMPLICATIONS FOR ECOLOGICAL AND EVOLUTION THEORIES

Adopting murburn principles necessitates rethinking of ecosystems as continuous but yet discretized (non)equilibrium chemical reactors. Biodiversity should be seen as stabilized stochasticity and evolution as environmentally modulated chemical exploration. This perspective dissolves false dichotomies between order and chaos, showing that ecological order arises from managed chemical disorder. Table 2 shows the new implications.

Table 2. Murburn's implications in ecological concepts

Ecological concept	Murburn contribution
Competition & Allelopathy	Explains it as a natural consequence of organisms broadcasting their redox chemistry, creating zones of inhibition or facilitation.
Symbiosis	Framed as the successful integration of disparate redox systems into a compatible, mutually beneficial network.
Succession	Viewed as a predictable progression in the redox-landscape of an environment, from harsh and oxidized to mild and reduced.
Nutrient Cycling	Identified as being driven by large-scale oxidative (murburn) processes in decomposers and primary producers.
Systemic Health	Proposes that the stability of an ecosystem is reflected in the stability of its collective redox chemistry.

Murburn theory provides a powerful, bottom-up lens for ecology. It posits that the seemingly chaotic and random chemistry of life at the cellular level is, in fact, the very engine that also drives the structure, dynamics, and intricate interconnectedness of life and non-life at the global scale. Here is the breakdown:

Micro scale: Enables flexible metabolism and energy transduction.

Macro scale: Links all living and non-living systems via diffusible redox couplings.

Ecological scale: Explains nutrient cycling, adaptability, and dynamic equilibrium.

Planetary scale: Describes a self-organized, non-equilibrium energy network sustaining life.

Following is a succinct analysis of how murburn shapes evolution:

Introduces built-in randomness: Murburn injects stochastic, non-genetic variation directly into cellular physiology through diffusive reactive molecules, creating phenotypic diversity even in genetically identical populations.

Decouples phenotype from genotype: The organism's realized traits emerge from the interplay between its genetic blueprint and the probabilistic redox chemistry, meaning the same genome can yield different phenotypes in fluctuating environments.

Enables rapid physiological adaptation: Changes in environmental factors (like oxygen or toxins) immediately alter the intracellular redox landscape, allowing for swift, within-generation metabolic adaptation without requiring genetic mutation.

Acts as a selective filter: An organism's ability to manage its own murburn chemistry (e.g., through antioxidants, repair systems) becomes a direct target of natural selection; inefficient management leads to oxidative damage and reduced fitness.

Drives evolutionary innovation: The constant, low-level oxidative modifications to proteins, lipids, and DNA serve as a source of molecular "experiments," occasionally producing novel functions upon which selection can act.

Facilitates speciation: Divergence in murburn system components (like mitochondrial complexes) can create irreversible physiological and metabolic incompatibilities between populations, contributing to reproductive isolation.

Scales to ecological interactions: An organism's redox "halo" influences competitors, predators, and symbionts, making the evolution of one species a direct selective pressure on the murburn physiology of others.

Unifies micro- and macro- evolution: The same stochastic redox principles that cause cellular variation also underpin larger-scale evolutionary trends in metabolism, complexity, and niche specialization over deep time.

8. SUMMATIVE STATEMENTS ON ECOLOGY-BIODIVERSITY-EVOLUTION

If life is sustained by controlled chaos, then biodiversity is its most elegant expression; and murburn chemistry its hidden architect. Its salient features are:

Distributed (not fixated!) redox/charge dynamics: Probabilistic, locally adaptive system permits divergent metabolic solutions, enabling diversity at molecular, cellular, and organismal levels.

Energetic decentralization: Radiations, mutations, lateral gene transfers, or environmental changes can more easily produce viable new metabolic routes.

Microenvironmental niches: Distinct reaction environments foster cradles of new metabolic pathways and species, from bacteria in vents to complex/coherent multicellular life!

Molecular diversity from safe randomness: DRS can cause modifications in DNA, proteins, and metabolites, causing genetic and phenotypic variability, fueling natural selection and adaptation.

Ecosystem-level integration: Biogeochemical cycles (C, N, S, O, X, etc.) self-sustain through redox coupling mediated by microbes and plants by thermodynamic optimization via the all-pervasive ecological CEM.

In short, ecology and evolution are the emergent behavior of countless murburn reactions distributed across scales, from molecules to biospheres.

9. RECENT ASCENDANCY OF MURBURN IN LITERATURE: DISCUSSIONS BY OTHER WORKERS

I have been very negative about the reception the global scientific community meted out to murburn. But there is a silver lining emerging at the periphery of the dark clouds that loomed large.

9.1 Zhang et al.'s exaltation of murburn as a pivotal founding principle of life

In their comprehensive review (28), "Nrf1 as determinant of redox homeostasis" published in *Cell Stress* (2025), Zhang et al. position Nrf1 (nuclear factor erythroid 2-related factor 1) as an indispensable "determinon" for maintaining robust redox homeostasis across evolutionary and developmental scales. The 78-page article synthesizes Nrf1's roles (drawing from eco-evo-devo perspective) in cytoprotection, proteostasis, lipid metabolism, energy regulation, and organ integrity, contrasting it with the more adaptive Nrf2. They stratify redox stress quantitatively and introduce novel concepts like "zero theory" for redox set points and the Grand Redox-Unifying Theory (GRUT) of life.

Murburn is exalted here as a foundational element of GRUT, with the authors listing it as a keyword and citing approximately 18 publications from my group. They portray

murburn as a “novel paradigm shift in redox-integrated bio-physico-chemical sciences,” integrating stochastic processes via DRS, Murzymes and murzones networks. DRS are reframed as “versatile elixirs” essential for respiration, immunity, and metabolic flexibility, operating at the “edge of chaos” to balance stochasticity and determinism. In relation to Nrf1, murburn underpins redox homeostasis by enabling multi-hierarchical signaling and gene reprogramming, where Nrf1 senses thiol-based redox changes to prevent oxidative damage and pathologies like neurodegeneration or cancer. The article favorably quotes: “The ‘murburn concept’, introduced by Indian biochemist Manoj, K.M. [497-499], represents a novel paradigm shift... ubiquitously implicated in all cellular life processes.” It links murburn to OSTeOL: abiogenesis via DRS-catalyzed prebiotic synthesis in hydrothermal vents, evolution through redox-driven self-organization (e.g., Fe-S clusters in murzymes for CO₂ fixation), sustenance via homeodynamic thresholds, perpetuation through stochastic fidelity, and termination via cumulative DRS dysregulation. This portrayal underscores murburn’s universality, aligning it with Nrf1’s basal roles while addressing anomalies in classical models, such as Q-cycle flaws in respiration or photochemical activation in photosynthesis. By citing murburn works on electron carriers, cytochrome interactions, and ATP synthesis, Zhang et al. affirm its thermodynamic validity, suggesting it resolves long-standing paradoxes in redox biology. This China-centric international collaboration signals murburn’s global traction, elevating it from a challenger hypothesis to a “pivotal founding principle of life.”

9.2. Yuan et al’s discussion on murburn’s role in ROS heterogeneity and carcinogenesis

Yuan et al.’s article (29), “Spatiotemporal heterogeneity of ROS in hepatitis-driven carcinogenesis” in *Cell & Molecular Biology Letters* (2025), cites 5 murburn papers and explores how ROS dynamics evolve during the progression from viral hepatitis to hepatocellular carcinoma (HCC). The review details oxidative stress escalation leading to chronic inflammation, fibrosis, and malignancy, with spatiotemporal heterogeneity influencing ROS patterns across organelles and disease stages. In early hepatitis, ROS from mitochondria and ER promote viral replication and immune evasion; in cirrhosis, they drive fibrosis via stellate cell activation; in HCC, elevated ROS support proliferation and metastasis while antioxidant systems (e.g., Nrf2, GSH) enable tumor survival. The authors discuss ROS-regulated pathways like NF-κB and MAPK, and therapeutic implications of targeting ROS heterogeneity.

They first portray what murburn is and how/why it is different from classical perceptions of seeing ROS in negative light. In hepatitis-driven carcinogenesis, this portrayal suggests murburn explains ROS’s dual role: facilitating metabolic reactions in normal liver function while contributing to spatiotemporal dysregulation in malignancy. The article notes: “According to this model, dROS such as superoxide anion and H₂O₂ are not merely metabolic waste; they actively mediate reactions... through free radical mechanisms.” However, it tempers enthusiasm by stating, “Despite its explanatory potential, the murburn model remains a subject of vigorous debate, necessitating rigorous experimental validation.” This favorable yet cautious integration highlights murburn’s relevance to cancer biology, where ROS heterogeneity mirrors murburn’s stochastic processes. By linking it to HCC progression, Yuan et al. imply murburn could inform targeted therapies, such as modulating dROS in immune microenvironments. This citation extends murburn’s outreach to oncology, portraying it as a innovative lens for understanding ROS in disease etiology.

9.3. Analyses of other favorable citations that broaden murburn’s applications

Francati et al.’s (30) “The Janus face of oxidative stress in health and disease” in *Biomedical Reviews* (2023) cites 4 murburn papers and examines ROS/RNS/DRS duality: beneficial for signaling and defense at low levels, pathological in excess (e.g., in cancer, neurodegeneration). They discuss sources, antioxidants, and therapeutic modulation. Murburn is portrayed as an alternative enzyme mechanism involving “closed burning” with DRS, where murzymes (e.g., hemoglobin, cytochrome c) and murzones enable oxidative processes under aerobic/anaerobic conditions. They note murburn’s deviation from standard kinetics, favoring multiple equilibria for metabolic functions like respiration and homeostasis. Favorably, it reframes DRS as crucial for physiology, aligning with oxidative stress’s Janus nature: “Enzymes carrying out the Murburn system are defined as ‘Murzymes,’ and the region where DRS interact... is termed ‘Murzone.’”

Chaldakov et al.’s (31) “Trackins (Trk-targeting drugs): A novel therapy for different diseases” in *Pharmaceuticals* (2024) reviews neurotrophins (e.g., NGF, BDNF) and Trk receptors for treating cardiometabolic and neurodegenerative disorders. Murburn is invoked to explain ROS/RNS interference in NGF-TrkA-microtubule-lipid raft signaling in Alzheimer’s. Citing a few murburn papers, they query: “We wonder whether the murburn concept of the biology of oxygen [89,90]

could explain such an association between *Trk*, *ROS*, *RNS*, *MT*, and *AD*.” This favorable speculation positions murburn as a framework for oxidative disruptions in neurometabolic pathways, complementing trackin therapies.

Dalbanjan et al.’s (32) “Electrochemical techniques in protein engineering” in *International Journal of Biological Macromolecules* (2025) focuses on redox-active protein design for bioelectronics. While no explicit “murburn” mentions appear in accessible content, the article’s emphasis on electron transfer mechanisms aligns with user-noted citations of murburn works in photosynthesis contexts (e.g., potentially referencing Manoj’s on Q-cycle alternatives). It portrays related ideas favorably through innovations in ET efficiency, indirectly supporting murburn’s stochastic redox views.

Abadie’s solo review (33), “A two-genome portrayal of mitochondrial disorders” in *Frontiers in Bioscience (Scholarly Edition)* (2024), covers mtDNA/nDNA genetics, clinical syndromes (e.g., MELAS, Leigh), and diagnostics. Murburn is highlighted as a “thermodynamically and kinetically valid model” superseding proton-centric ETC views, with DROS as metabolic intermediates. Citing 2 of my papers critiquing ETC-CRAS views, Abadie states: “*However, a more recent... model based on the Murburn concept was explained by Manoj.*” This portrayal favors murburn for interpreting OXPHOS defects, suggesting DROS enable direct substrate-ADP coupling, reframing them as “essential intermediates... instead of merely agents of chaos.”

9.4. Murburn’s growing impact and future trajectory

Portrayed as a paradigm-shifting, unifying framework, these recent publications portray murburn favorably for resolving classical anomalies and redefining DRS/ROS as functional elixirs. With multiple citations of murburn in a single paper and papers that span across disciplines, it signals resilience and philosophical depth in explaining OSTeOL. As debates continue, continued empirical validation could cement murburn’s everlasting impact, rivalling other biological theories (like Darwinian evolution) in biological unification. The works cited above are not passive references; they are active participants in constructing a new, expanded paradigm. They use multiple papers on murburn to: reframe foundational principles (29), address clinical paradoxes (33; 30), unify mechanisms of stress and signaling (30,29) and inform therapeutic and engineering design (31-33). With the publication/acceptance of works that quantitatively ground murburn in mathematical treatments and identify murzymes/murburn systems with algorithmic/AI-ML

methods, murburn is set to greater outreach, beyond the sphere of influence of those that are bent on subduing it.

10. STRATEGIES AHEAD FOR ENHANCING MURBURN’S OUTREACH/ACCEPTANCE

When Sabine Hossenfelder says that “Research is broken!”, it is nothing but the truth ([youtube.com/watch?v=9yPy3DeMUyI&vl=en](https://www.youtube.com/watch?v=9yPy3DeMUyI&vl=en))!

When I submit pre-submission queries to popular biochemistry/biophysics journals, it is indeed a frustration to note the dismissive responses from the editors therein (when they say that a more specialized journal could suit it better!). Assurances were given to me by some seniors that system will come around in a few years, but none of that happened. They said that I should refrain from frontal attack, as it is unproductive. Now, I understand totally that paradigm-challenging concepts do not advance by frontal assault on orthodoxy (because the leadership of the scientific community is more concerned with saving their faces than serving those that they lead!). They advance by solving problems orthodoxy cannot solve; quietly, repeatedly, and reproducibly. Therefore, my group shall continue to demonstrate that murburn explains existing anomalies better than prevailing models, with more literature mining, pattern extraction, meta-analysis, ideas clustering and concrete actions. For example, the recent Q1 (SCOPUS) publications in 2025 list a dozen or more fields wherein murburn pips the classical perceptions. Particularly, in mitochondrial oxidative phosphorylation and hepatocyte endoplasmic reticulum mediated xenobiotic/drug metabolism, more than three dozens of theoretical, experimental and structural aspects are pointed out! Via these works, we are not asking for investment of belief, but we are showing explanatory economy and power! Further, multiple enzymes and reaction systems are now capable of being parsed into murburn/murzyme system using algorithmic and AI-ML logic, breaking the monotonous dominance of enforced consensus! More computational modeling, predictive and simulative works shall follow. Experiments shall be suggested to demarcate the murburn logic and specific agenda shall be charted out, as done in the ends of recent published papers. Small wet lab experiments with UG/PG experiments shall be undertaken to make the young ones see the logic and evidence of the new idea. The recurring motifs of diffuse intermediates, non-specific reactivity and unusual context-dependent outcomes shall be demonstrated, making it inevitable for the scientific community to disregard murburn. Whatever I do, the tone will be argumentative and confrontational. For, science

and scientists must accept this as a way of doing research, it is based in claims and arguments/evidence to support them. We must discourage overly cautious attitudes that resist engaging with radical scientific claims. Most importantly, the focus would shift to building a “murburn research ecosystem” and decentralize validation from the self-appointed authorities and their eager appeasers. Although AI does not substitute human intelligence, we can now have students indulge in real-time debate with AI portals (which are more amenable to introspective inspections and genuine prompting!) to see that reasoning can critically acclaim murburn concept, and slowly break the authority-enforced consensus!

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