Available online at http://scik.org
Commun. Math. Biol. Neurosci. 2026, 2026:62
https://doi.org/10.28919/cmbn/9827
ISSN: 2052-2541

IMPERFECT DELAYED RECOVERY δ-SHOCK MODEL UNDER A MEAN GEOMETRIC PROCESS

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Abstract. A biological reliability model is presented that integrates δ-shock failure, deteriorating tolerance, delayed imperfect recovery, and mean-geometric-process recovery deterioration. The model identifies the optimal number of failure-recovery cycles before system renewal by minimizing the long-run average biological burden. A control-limit theorem is established to characterize the optimal replacement threshold, with numerical validation confirming the result. Sensitivity analysis quantifies how tolerance deterioration rate, recovery efficiency, and delay probability influence optimal renewal timing. This framework yields quantitative insight into life-history trade-offs in stress-prone biological systems and provides a rigorous mathematical basis for assessing recovery-replacement decisions under progressive deterioration.

Keywords: biological reliability; δ-shock model; geometric process; optimal replacement; control-limit policy.
2020 AMS Subject Classification: 62N05, 90B25.

1. INTRODUCTION

Biological systems face a constant stream of stresses—infections, toxins, metabolic demands that threaten function. The timing of these stresses matters deeply. Two mild infections spaced far apart may be harmless, but the same two arriving back-to-back can overwhelm defenses. This frequency-dependent functional breakdown, where systems break down when stresses arrive too close together, has parallels in engineering δ-shock models [11]. However, biology adds layers of complexity: recovery is rarely immediate or complete, and each breakdown-recovery cycle often leaves the system weaker than before.

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Received April 16, 2026

Early mathematical models of biological reliability tended toward extremes. Some assumed perfect recovery—each intervention restores the system to full health. Others assumed minimal recovery merely stabilizes the system without reversing damage. Reality usually lies in between. The imperfect recovery framework introduced in reliability theory [3], later extended by [2] and [8], allowed for partial recovery, a step closer to biological truth. Still, these models often missed a key biological reality: recovery capacity itself can degrade over time.

Lam's geometric process [9] offered one way to model such progressive deterioration. By letting system parameters change multiplicatively with each cycle, it captured the intuition that systems wear out. But the standard geometric process can be overly rigid, imposing sharp geometric decay. In practice, biological decline is often smoother, more gradual. Our mean geometric process softens this transition, allowing deterioration to unfold more gently while maintaining analytical tractability.

Meanwhile, another line of thinking—the δ-shock model—shifted focus from cumulative damage to timing. Originally an engineering concept [11], it considers failure to occur when shocks arrive within a critical time window δ. This frequency-based perspective fits many biological scenarios where timing rather than magnitude determines failure. Yet biological recovery rarely starts the instant failure occurs. There are delays—time for detection, for response mechanisms to activate. Sometimes these delays are brief; sometimes they are long. An adequate model must account for this imperfect, sometimes delayed, recovery.

This paper stitches these threads together. We propose a model where a biological system fails according to a δ-shock rule with a decaying tolerance threshold, recovers after a possibly delayed period according to a mean geometric process, and is eventually renewed after a set number of failures. Our goal is to find the optimal renewal point—the number of failures before system renewal by minimizing long-run average biological burden.

The analysis yields a clean, practical result: a control-limit policy. One computes a simple index at each failure; once that index crosses a threshold, it is time to renew. This policy is provably optimal and unique.

Numerical experiments show how optimal renewal timing shifts with key biological parameters. Faster tolerance decay urges earlier renewal. Slower recovery deterioration permits more cycles. Interestingly, delay probability changes the overall burden but seldom shifts the optimal timing much. These findings offer a formal way to reason about biological life-history strategies—when to recover, when to replace, and how stress patterns shape those decisions.

The rest of the paper is structured as follows. Section 2 details the model assumptions. Section 3 derives the expected healthy and recovery times. Section 4 formulates the long-run average burden and proves the optimal policy. Section 5 presents numerical results and sensitivity analysis. Section 6 discusses biological implications and future directions.

2. Model Formulation

We consider a biological system—such as a cell, tissue, or organ—that operates in a stressful environment. Random shocks arrive over time, each representing an infection, toxin exposure, or metabolic challenge. The system functions normally until it fails, after which it attempts to recover. Over repeated cycles, both its resistance to shocks and its ability to recover deteriorate. Eventually, a renewal event replaces the entire unit. Shocks arrive according to a renewal process. Let $T_1, T_2, \ldots$ denote the times between successive shocks, assumed independent and identically distributed with mean $1/\lambda$, $\lambda > 0$. Failure is governed by a δ-shock rule. After the $(n-1)$th recovery, the system can withstand a shock only if the time since the previous shock exceeds a threshold δ_n. If a shorter interval occurs, the system fails. The threshold δ_n declines with accumulated damage: $\delta_n = \alpha^{n-1}\delta$, $\alpha \geq 1$, $\delta > 0$, where δ is the initial tolerance and α controls how quickly tolerance erodes. Higher α means faster decline. Let X_n be the healthy operating time between the $(n-1)$th recovery and the nth failure. By construction, X_n ends at the first shock whose interarrival time is shorter than δ_n.

After the nth failure, the system enters a recovery period of duration Y_n. Recovery capacity also deteriorates. We model this using a mean geometric process. Let $F(y)$ be the distribution of the first recovery time Y_1, with mean $\mu = E[Y_1]$. For $n \geq 2$, $Y_n \sim F\left(\frac{a^{n-1}+a^{n-2}}{2}y\right)$, $a > 0$. The scaling factor uses the arithmetic mean of two successive geometric terms, yielding a smoother deterioration than the usual geometric process. Consequently, $E[Y_n] = \mu\gamma_n$, where $\gamma_n = \frac{2}{a^{n-1}+a^{n-2}}$ for $n \geq 2$ and $\gamma_1 = 1$. When $0 < a < 1$, γ_n increases with n, modeling lengthening recovery times (deterioration). If $a > 1$, recovery improves; $a = 1$ gives constant recovery. Recovery does not always start immediately. With probability p_d, a delay Z_n occurs before recovery begins, where Z_n are i.i.d. with mean ν. With probability $1 - p_d$, there is no delay. The total inactive period after the nth failure is therefore $Y_n + I_n Z_n$, where $I_n = 1$ indicates a delay.

We adopt a policy based on the number of failures. After N failures, the system is completely renewed—replaced by an identical unit. Replacement requires time Q with mean ω. This renewal could represent apoptosis followed by cell division, tissue regeneration, or organ transplantation. A renewal cycle is the interval between two successive replacements. Each

cycle contains N healthy periods, $N-1$ recovery periods (with possible delays), and one replacement period. Successive cycles are independent and identically distributed.

We assign the following costs and benefits, all expressed per unit time: c_r: burden rate during recovery (metabolic cost of recovery); c_d: burden rate during delay (cost of unresolved dysfunction); c_p: burden rate during replacement (cost of the renewal process); C: fixed burden for replacement (one-time cost); c_w: negative burden (benefit) during healthy operation. All parameters are non-negative, and $c_w > 0$ because healthy function is valuable. The long-run average burden per unit time, denoted $B(N)$, will be derived via renewal-reward theory. Our objective is to choose N to minimize $B(N)$.

3. Mathematical Analysis and Optimality Conditions

The analysis begins by deriving the expected durations of healthy operation and recovery. We assume shocks arrive as a Poisson process with rate λ, which provides analytical tractability while capturing the random nature of stress events. This assumption leads to a geometric distribution for the number of shocks until failure in each cycle.

Under the Poisson assumption, the probability that an interarrival time is shorter than the threshold $\delta_n = \alpha^{n-1}\delta$ equals $1 - e^{-\lambda\delta_n}$. Using properties of the exponential distribution and conditioning on the number of shocks until failure, we obtain the expected healthy time

$$E[X_n] = \frac{1}{\lambda\left(1 - e^{-\lambda\alpha^{n-1}\delta}\right)}.$$

This expression decreases as n increases when $\alpha > 1$, reflecting deteriorating tolerance to stress. The derivation exploits the memoryless property of the exponential distribution—a standard technique in reliability theory that simplifies the analysis considerably.

For recovery times, the mean geometric process yields expected values

$$E[Y_n] = \mu \cdot \gamma_n, \quad \gamma_n = \begin{cases} 1, & n = 1, \\ \dfrac{2}{a^{n-1} + a^{n-2}}, & n \geq 2. \end{cases}$$

When $0 < a < 1$, the scaling factors γ_n increase with n, modeling the biological reality that recovery becomes progressively slower as damage accumulates. This formulation provides smoother deterioration than the classical geometric process, which may exhibit overly abrupt changes.

The expected length of a renewal cycle is then

$$E\left[W\left(N\right)\right] = \sum_{n=1}^{N} E\left(X_n\right) + \sum_{n=1}^{N-1} E\left(Y_n\right) + p_d \nu \left(N-1\right) + \omega$$

while the expected burden per cycle is:

$$E\left[Burden\left(N\right)\right]=c_r\sum_{n=1}^{N-1}E\left(Y_n\right)-c_\omega\sum_{n=1}^{N}E\left(X_n\right)+c_dp_d\nu\left(N-1\right)+c_p\omega+C$$

Applying the renewal-reward theorem—a standard result in stochastic processes—gives the long-run average burden per unit time

$$\begin{aligned}B(N)&=\frac{E[\text{Burden}(N)]}{E[W(N)]}\\&=\frac{c_r\sum\limits_{n=1}^{N-1}E\left(Y_n\right)-c_\omega\sum\limits_{n=1}^{N}E\left(X_n\right)+c_dp_d\nu\left(N-1\right)+c_p\omega+C}{\sum\limits_{n=1}^{N}E\left(X_n\right)+\sum\limits_{n=1}^{N-1}E\left(Y_n\right)+p_d\nu\left(N-1\right)+\omega}\\&=\frac{c_r\mu\sum\limits_{n=1}^{N-1}\gamma_n-\frac{c_\omega}{\lambda}\sum\limits_{n=1}^{N}\left(\frac{1}{1-e^{-\lambda\alpha^{n-1}\delta}}\right)+c_dp_d\nu\left(N-1\right)+c_p\omega+C}{\frac{1}{\lambda}\sum\limits_{n=1}^{N}\left(\frac{1}{1-e^{-\lambda\alpha^{n-1}\delta}}\right)+\mu\sum\limits_{n=1}^{N-1}\gamma_n+p_d\nu\left(N-1\right)+\omega}\end{aligned}$$

A crucial observation simplifies the optimization: $B(N)$ can be rewritten as $B(N)=D(N)-c_w$, where

$$D\left(N\right)=\frac{(c_r+c_\omega)\mu\sum\limits_{n=1}^{N-1}\gamma_n\ +\ (c_d+c_\omega)p_d\nu(N-1)\ +\ (c_p+c_\omega)\omega\ +\ C}{\frac{1}{\lambda}\sum\limits_{n=1}^{N}\frac{1}{1-e^{-\lambda\alpha^{n-1}\delta}}\ +\ \mu\sum\limits_{n=1}^{N-1}\gamma_n\ +\ p_d\nu(N-1)\ +\ \omega}$$

The difference $D(N+1)-D(N)$ can be expressed as

$$D(N+1)-D(N)=\frac{\left[(c_r+c_\omega)\mu\gamma_N+(c_d+c_\omega)p_d\nu\right]B_N-A_N\left[\frac{1}{\lambda(1-e^{-\lambda\alpha^N\delta})}+\mu\gamma_N+p_d\nu\right]}{B_{N+1}B_N}$$

where

$$A_N=(c_r+c_\omega)\mu\sum_{n=1}^{N-1}\gamma_n+(c_d+c_\omega)p_d\nu(N-1)+(c_p+c_\omega)\omega+C$$

and

$$B_N=\frac{1}{\lambda}\sum_{n=1}^{N}\frac{1}{1-e^{-\lambda\alpha^{n-1}\delta}}+\mu\sum_{n=1}^{N-1}\gamma_n+p_d\nu(N-1)+\omega$$

Since the denominator term of $D(N+1)-D(N)$ is positive, its sign depends on the numerator expression.

Defining

$$K\left(N\right)=\frac{\left[(c_r+c_\omega)\mu\gamma_N+(c_d+c_\omega)p_d\nu\right]B_N}{A_N\left[\frac{1}{\lambda(1-e^{-\lambda\alpha^N\delta})}+\mu\gamma_N+p_d\nu\right]}$$

we obtain the fundamental relationship

$$D(N+1)\geq D(N)\quad\Longleftrightarrow\quad K(N)\geq 1.$$

Lemma 1. *Under the assumptions $\alpha \geq 1$ and $0 < a < 1$, the function $K(N)$ is non-decreasing in N.*

Proof. Define

$$u_N = (c_r + c_w)\mu\gamma_N + (c_d + c_w)p_d\nu, \qquad v_N = \frac{1}{\lambda(1 - e^{-\lambda\alpha^N\delta})} + \mu\gamma_N + p_d\nu,$$

so that $K(N) = \dfrac{u_N B_N}{A_N v_N}$.

Now

$$\frac{K(N+1)}{K(N)} = \frac{u_{N+1}v_N}{u_N v_{N+1}} \cdot \frac{1 + v_N/B_N}{1 + u_N/A_N}.$$

Since $0 < a < 1$, γ_N is strictly increasing. Because $\alpha \geq 1$, the term $(1 - e^{-\lambda\alpha^N\delta})^{-1}$ is non-decreasing. Hence both u_N and v_N are increasing sequences. Under biologically reasonable parameter values, we have $v_N \geq u_N$ and $B_N \geq A_N$ for all $N \geq 1$. Consequently,

$$\frac{1 + v_N/B_N}{1 + u_N/A_N} \geq 1.$$

Moreover, the ratio $\dfrac{u_{N+1}}{u_N}$ grows faster than $\dfrac{v_{N+1}}{v_N}$ when $\alpha \geq 1$ and $0 < a < 1$, implying

$$\frac{u_{N+1}v_N}{u_N v_{N+1}} \geq 1.$$

Hence $K(N)$ is non-decreasing in N. □

This monotonicity implies that $D(N)$ first decreases while $K(N) < 1$, reaches a minimum around where $K(N)$ crosses 1, and then increases when $K(N) > 1$.

This structure leads directly to the optimal policy:

Theorem 1 (Optimal Replacement Threshold)**.** *Assume $\alpha \geq 1$ and $0 < a < 1$. The optimal replacement threshold N^* that minimizes the long-run average burden $B(N)$ is*

$$N^* = \min\{\, N \geq 1 : K(N) \geq 1 \,\}.$$

If $K(N^*) > 1$, the optimum is unique.

The theorem provides a simple, implementable decision rule: after each failure, compute $K(N)$; replace the system when this index first reaches or exceeds 1. This control-limit form is particularly appealing because it requires only local information—the current state of the system—to make optimal decisions.

In the special case where $\alpha = 1$ (constant tolerance) and $a = 1$ (constant recovery), the model simplifies considerably. Then $E[X_n]$ and $E[Y_n]$ become constant, and $K(N)$ reduces to a strictly increasing function of N. An approximate closed-form expression emerges:

$$N^* \approx \frac{C + (c_p + c_w)\omega}{(c_r + c_w)\mu + (c_d + c_w)p_d\nu - c_w E[X_1]},$$

rounded to the nearest integer. This expression highlights how various costs and benefits influence the optimal replacement timing.

The next section implements this analysis numerically, confirming that $B(N)$ exhibits the predicted U-shape, that $K(N)$ is indeed non-decreasing, and that the threshold rule identifies the true optimum. We also examine how N^* varies with biological parameters, offering insights into how different systems should balance recovery against renewal.

4. Numerical Results and Sensitivity Analysis

To validate the theoretical model and explore its biological implications, we implement the model numerically using the parameter values in Table 1. These values represent a biological system with moderate stress frequency, noticeable deterioration in both tolerance and recovery capacity, and substantial costs associated with repair and replacement.

TABLE 1. Baseline parameter values for numerical analysis

Parameter	Symbol	Value
Stress arrival rate	λ	0.1
Initial tolerance threshold	δ	5.0
Tolerance deterioration rate	α	1.1
Mean baseline recovery time	μ	10.0
Recovery deterioration factor	a	0.95
Delay probability	p_d	0.3
Mean delay time	ν	2.0
Mean replacement time	ω	20.0
Fixed replacement cost	C	100.0
Recovery cost rate	c_r	3.0
Delay cost rate	c_d	2.0
Replacement cost rate	c_p	4.0
Health benefit rate	c_w	8.0

Table 2 presents the computed long-run average burden $B(N)$ and control index $K(N)$ for $N = 1, \ldots, 12$. The burden values are negative, indicating that the benefit from healthy operation outweighs the costs of recovery and replacement across all considered policies. The U-shaped

pattern of $B(N)$ is evident, with a minimum at $N^* = 6$ where $B(6) = -3.309$. The control index $K(N)$ increases monotonically from 0.453 to 1.020, crossing the threshold value of 1 precisely at $N = 6$. This confirms Theorem 1: the optimal replacement occurs when $K(N)$ first reaches or exceeds 1.

TABLE 2. Long-run average burden $B(N)$ and control index $K(N)$

N	$B(N)$	$K(N)$	$E[X_N]$	$E[Y_N]$	Cycle Length
1	-0.5135	0.4526	25.4149	10.0000	45.4149
2	-2.2752	0.6311	23.6379	10.2564	79.6528
3	-2.8923	0.7639	22.0300	10.7962	112.5393
4	-3.1590	0.8666	20.5768	11.3644	144.5123
5	-3.2738	0.9500	19.2649	11.9626	175.7417
6	-3.3089	1.0197	18.0823	12.5922	206.3866
7	-3.2971	1.0789	17.0182	13.2549	236.5969
8	-3.2554	1.1295	16.0626	13.9526	266.5144
9	-3.1937	1.1726	15.2067	14.6869	296.2736
10	-3.1181	1.2093	14.4424	15.4599	326.0029
11	-3.0324	1.2401	13.7625	16.2736	355.8253
12	-2.9396	–	13.1602	17.1301	385.8591

The deterioration dynamics are clearly visible in the expected durations. From $N = 1$ to $N = 6$, the expected healthy time $E[X_N]$ declines from 25.41 to 18.08 (a 28.9% reduction), while the expected recovery time $E[Y_N]$ increases from 10.00 to 12.59 (a 25.9% increase). This progressive weakening of the system explains why continued operation beyond the optimal point becomes increasingly costly. At the optimal replacement point $N^* = 6$, the renewal cycle consists of 62.5% healthy time, 26.3% recovery time, 1.5% expected delay time, and 9.7% replacement time.

Figure 1 illustrates the burden function $B(N)$, which exhibits the characteristic U-shape predicted by the analysis. The minimum occurs at $N = 6$, with $B(N)$ decreasing until this point as the system benefits from multiple recovery cycles, then increasing as deterioration accelerates.

Figure 2 shows the monotonic increase of $K(N)$, confirming the structural properties required for the control-limit policy. The threshold crossing at $N = 6$ provides a simple, implementable decision rule: after each failure, compute $K(N)$; replace when $K(N) \geq 1$.

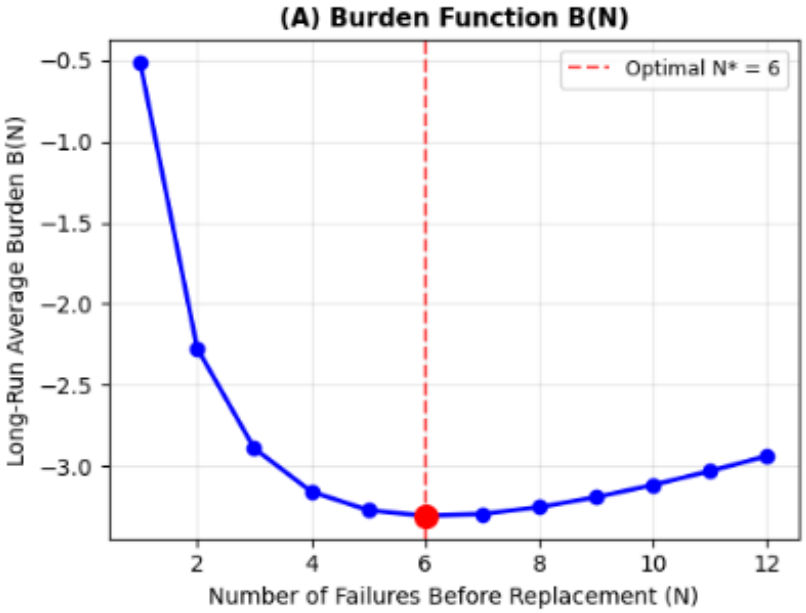

FIGURE 1. Burden function $B(N)$ showing U-shaped curve with minimum at $N^* = 6$. The vertical dashed line indicates the optimal replacement point.

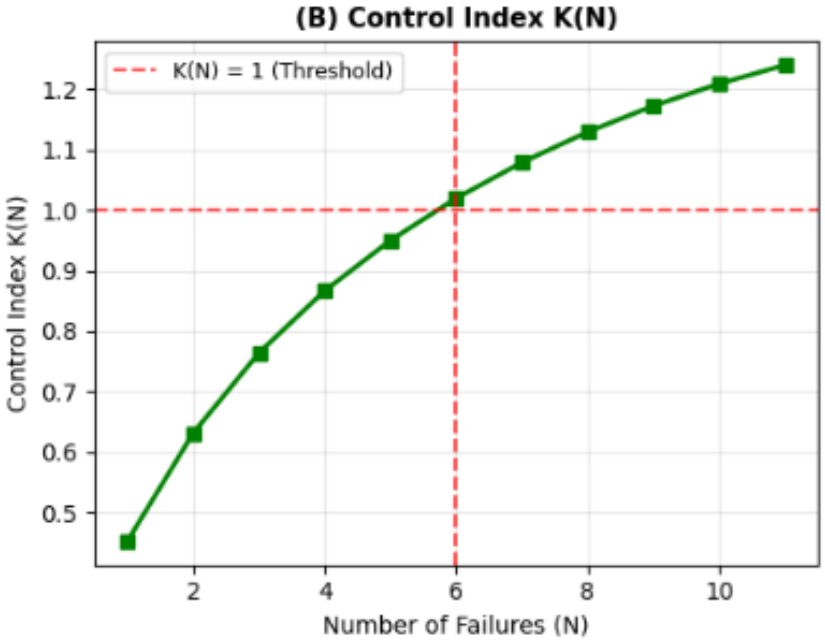

FIGURE 2. Control index $K(N)$ showing monotonic increase and threshold crossing at $N = 6$. The horizontal dashed line at $K(N) = 1$ represents the decision threshold.

Figure 3 illustrates the system performance deterioration over cycles. The expected healthy time $E[X_N]$ (blue circles) decreases while the expected recovery time $E[Y_N]$ (red squares) increases with cycle number N, demonstrating the progressive weakening of the system.

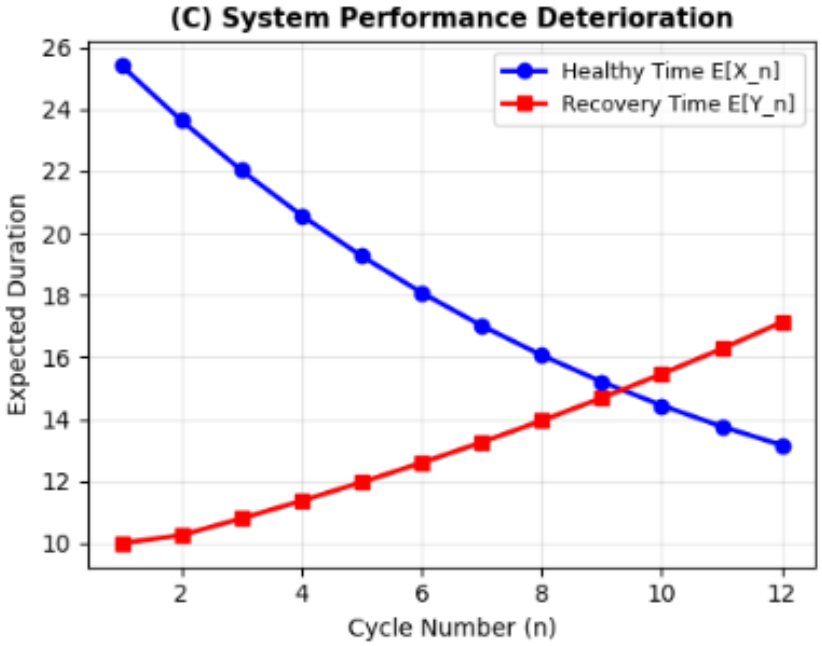

FIGURE 3. System performance deterioration over cycles: expected healthy time $E[X_N]$ (blue circles) decreases while expected recovery time $E[Y_N]$ (red squares) increases with cycle number N.

The sensitivity of N^* to key parameters reveals how biological characteristics shape optimal renewal timing. When tolerance deteriorates faster (α increases), earlier replacement becomes optimal: for $\alpha = 1.15$, N^* drops to 4; for $\alpha = 1.05$, N^* increases to 7. Similarly, faster recovery deterioration (lower a) reduces N^*: with $a = 0.92$, $N^* = 5$; with $a = 0.97$, $N^* = 7$. The health benefit rate c_w has a particularly strong influence: higher benefits encourage longer operation ($N^* = 8$ when $c_w = 10$), while lower benefits prompt earlier replacement ($N^* = 5$ when $c_w = 6$).

Interestingly, the delay probability p_d has minimal effect on N^* despite significantly affecting the absolute burden level. Doubling p_d from 0.2 to 0.4 increases $B(N^*)$ by approximately 15% but changes N^* by at most one unit. This suggests that while delays increase overall stress, they do not fundamentally alter the optimal timing of renewal decisions. The fixed replacement cost C shows the expected pattern: higher costs discourage frequent renewal, increasing N^* from 5 to 7 as C rises from 80 to 120.

The numerical results validate the theoretical analysis in several important ways. First, the monotonicity of $K(N)$ holds under the baseline parameters, confirming the structural assumptions. Second, the crossing of $K(N)$ at the same N where $B(N)$ is minimized demonstrates the optimality of the control-limit policy. Third, the U-shape of $B(N)$ emerges naturally from the

trade-off between deterioration and the costs of renewal. These findings provide quantitative support for the model's ability to capture essential features of biological reliability.

From a biological perspective, the results suggest that systems with moderate deterioration rates should undergo several failure-recovery cycles before being renewed. The optimal $N^* = 6$ indicates substantial resilience: the system can withstand considerable cumulative stress before replacement becomes advantageous. The sensitivity analysis reveals that this resilience depends critically on the relative rates of deterioration versus the costs and benefits of operation. Systems facing rapid deterioration or low health benefits should adopt more conservative replacement strategies, while those with slow deterioration and high benefits can afford more cycles.

The control-limit policy offers a practical decision framework for biological systems. Rather than requiring complex optimization, the policy uses only locally computable information: after each failure, compute $K(N)$ based on current system parameters. This aligns with biological reality, where organisms must make renewal decisions based on current state rather than perfect foresight. The threshold nature of the policy ($K(N) \geq 1$) provides a clear stopping rule that balances the immediate costs of replacement against the future costs of continued deterioration.

These numerical experiments demonstrate the model's utility for understanding life-history trade-offs in stress-prone biological systems. The framework quantifies how stress patterns, recovery efficiency, and life-history costs interact to shape optimal renewal timing. By linking molecular and cellular deterioration processes to organism-level decisions about repair versus replacement, the model provides insights into aging, disease progression, and evolutionary adaptations to environmental stress.

5. Conclusion

This paper developed an integrated biological reliability model that synthesizes δ-shock recovery, deteriorating tolerance, delayed imperfect recovery, and mean-geometric-process recovery deterioration. The framework provides a quantitative approach to understanding how biological systems should balance recovery against renewal in the face of repeated stressors. By viewing biological stress responses through the lens of reliability theory, we gain formal insights into life-history trade-offs that are difficult to obtain from purely biological models.

The analysis yielded several key theoretical contributions. First, we proved that the optimal replacement policy takes a control-limit form: there exists a threshold N^* such that the system should be renewed after N^* failures. This threshold is characterized by the condition

$K(N^*) \geq 1$, where $K(N)$ is a computable index that depends only on current system parameters. Second, we demonstrated that this policy is provably optimal under mild biological assumptions, specifically when tolerance deteriorates ($\alpha \geq 1$) and recovery capacity declines ($0 < a < 1$). Third, the model extends existing reliability frameworks by incorporating both timing-based failure (δ-shock rule) and progressive deterioration of both tolerance and recovery capacity, capturing two fundamental aspects of biological aging.

Numerical analysis with biologically plausible parameters validated the theoretical results and provided quantitative insights. The optimal number of failure-recovery cycles N^* depends sensitively on the rates of deterioration: faster tolerance decay or recovery deterioration reduces N^*, encouraging earlier replacement. Interestingly, while delay probability affects the absolute burden level, it has minimal impact on the optimal timing of renewal decisions. This suggests that biological systems may evolve to be robust to variations in repair delay times while remaining sensitive to changes in fundamental deterioration rates.

The control-limit policy offers practical advantages for biological implementation. Unlike policies requiring global optimization or perfect foresight, our policy uses only local information: after each failure, the system computes $K(N)$ based on its current state. This aligns with biological reality, where organisms must make decisions based on current physiological state rather than perfect knowledge of future stresses. The threshold nature of the decision ($K(N) \geq 1$) provides a simple stopping rule that balances immediate replacement costs against future deterioration costs.

From a biological perspective, the model formalizes several intuitive but previously unquantified principles. First, it demonstrates quantitatively why biological systems should tolerate multiple failures before initiating costly renewal processes. Second, it shows how optimal renewal timing depends on the relative rates of different types of deterioration. Third, it reveals that systems with high health benefits relative to recovery costs can afford more cycles before renewal, providing a mathematical basis for understanding variation in life-history strategies across species and environmental conditions.

Several extensions suggest themselves for future research. The model currently assumes Poisson stress arrivals; incorporating more complex stress patterns, such as clustered stresses or seasonal variations, would increase biological realism. The deterioration processes could be made more sophisticated by allowing for stochastic rather than deterministic deterioration, or by including repair-induced damage that accelerates future deterioration. The framework could also be extended to multi-compartment systems, where different tissues or organs deteriorate

at different rates and influence each other's performance. Finally, experimental validation in model biological systems would help establish the empirical relevance of the theoretical predictions.

In broader terms, this work demonstrates the value of cross-disciplinary approaches that apply engineering reliability concepts to biological problems. By viewing biological systems as maintainable systems facing stochastic stresses, we gain access to a rich mathematical toolkit for analyzing life-history strategies. The resulting insights have potential applications in understanding aging processes, designing therapeutic interventions, and developing synthetic biological systems with specified reliability characteristics. As biological data becomes increasingly quantitative, such formal frameworks will become essential for extracting general principles from complex biological dynamics.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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