

Physical Inputs, Internal Responses, and Repair in Medusozoa

A selective evidence synthesis with a descriptive *Aurelia* regeneration time course

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Literature synthesis and secondary analysis of public data. Unnumbered draft; not submitted, published, or independently peer reviewed.

Abstract

Background. Physical conditions, cellular signaling, and tissue organization influence cnidarian repair, but these findings do not necessarily establish a retained internal state after a separate environmental cue ends. We ask whether a brief, naturally realistic mechanical or electrical input can produce a measurable post-input change that affects closure, remodeling, or organ regeneration.

Methods. We integrate a supplied synthesis of 30 primary-research and associated-data records with a descriptive reanalysis of the public single-cell atlas of *Aurelia coerulea* rhopalium regeneration. Nine pooled post-amputation samples contain 205,747 captured cells. We distinguish cell-type proportions, within-cell-type normalized RNA, and transcript detection, without treating cells as independent animals.

Results. Published experiments connect mechanical stretch to Hydra organizer activity, electrical conditions to reversible morphogenesis, and wound-associated molecular functions to *Aurelia* blastema formation. Repair endpoints are separable: wound closure and remodeling need not entail replacement of a functional organ. In the deposited *Aurelia* atlas, the wound-induced-cell (WIC) cluster constitutes 5.246% of captured cells immediately after amputation, 38.341% at 0.5 days, 36.136% at day 1, and 0.663% at day 2. Its preselected 20-gene signature also has its highest within-cluster mean at 0.5 days, although individual candidate markers differ. Later estimates become sparse, reaching four wound-induced cells at day 7.

Conclusion. The combined evidence supports a temporally structured post-injury repair response and experimentally consequential interactions between physical conditions and cellular programs. It does not yet establish the complete separate-cue-to-retained-state-to-repair sequence, intracellular hydration as its mediator, or a naturally realistic heartbeat exposure. The synthesis identifies what is measured, what the new calculations add, and which causal connections remain open.

Keywords: Medusozoa; regeneration; mechanobiology; bioelectricity; wound-induced cells; single-cell RNA sequencing; post-input persistence.

1. Introduction

Regeneration requires more than survival after damage. Tissue boundaries can close, surviving structures can rearrange, and missing organs can be replaced; these outcomes need not share the same requirements. In cnidarians, mechanical stretch can influence organizer signaling, imposed electrical conditions can redirect morphogenesis, and wound-associated molecular programs contribute to organ rebuilding. These findings make the interaction between physical inputs and cellular responses a substantive research question. [1] [2] [3]

The central question is more specific than whether jellyfish respond to their surroundings: **can a brief, naturally realistic mechanical or electrical input produce a measurable internal change after the input stops, and does that change affect a defined repair outcome?** Injury itself must be distinguished from a separate cue. Amputation creates both a wound and the conditions for its response; it is not a clean test of a noninjurious pulse followed by a retained state.

The relevant internal variables also require definition. Cytoplasmic mechanics, calcium-probe activity, gene expression, cell-population composition, and tissue architecture are not interchangeable measurements. Mammalian-cell studies establish intracellular mechanical and membrane-repair components, whereas jellyfish extracellular-matrix measurements address a different compartment. Extracellular ATP effects on jellyfish epithelial closure do not, by

themselves, measure intracellular hydration. [4] [5] [6] [8]

This paper makes two linked contributions. First, it organizes the existing evidence by exposure, measured response, repair endpoint, and inferential limit. Second, it adds completed calculations from the Aurelia regeneration atlas to a question that the earlier synthesis left for future analysis. The resulting account is a selective synthesis with a descriptive secondary analysis, not a new biological experiment or a claim that one mechanism has been demonstrated across all included species.

2. Materials and methods

2.1 Literature scope and evidence classification

The literature component uses the supplied 18 September 2026 synthesis and its saved source register. Its 30 reference records include primary studies and associated data, not 30 independent replications. The original synthesis used focused searches across six questions; this compilation does not claim a new exhaustive search, systematic-review selection procedure, or meta-analysis. The uploaded 13-page PDF is byte-identical to the previously saved synthesis.

Medusozoan evidence spans freshwater Hydra polyps and marine medusae from different classes and life stages. Mammalian cellular studies and a colonial chordate study are retained as explicitly external comparisons, not as jellyfish experiments. Findings are classified as published experimental observations or perturbations, computational inferences, descriptive secondary-analysis results, or proposed connections. Absence of a required comparison is reported as unresolved in the located evidence, rather than as experimental refutation.

2.2 Aurelia data and experimental units

The article-linked processed-data/code deposit was verified at Zenodo record 19466460, version 3. Its Seurat object contains nine raw RNA count layers and 29,766 genes in their union. The BioProject accession PRJNA1224934 corroborates organism and study identity; its public run metadata were inspected without downloading sequencing reads. The official supplementary PDF and Dataset S1 workbook were obtained through Europe PMC. [3] [31] [32] [33]

The processed samples are H0, H12, H24, H48, H72, H96, H120, H144, and H168, corresponding to 0, 0.5, 1, 2, 3, 4, 5, 6, and 7 days after amputation (dpa). The methods pool tissue from three medusae before single-cell capture at each stage. This yields one measured pool per stage, not three independently measured animal replicates. Day 0 is already post-amputation. Archive records share one BioSample and do not provide animal-level identities. A day-14 single-cell-classified run exists in the archive but is absent from this processed nine-stage atlas and was not added to the analysis. [3] [32]

The separate bulk experiment describes 51 libraries: three biological replicates for intact controls and each of 16 post-amputation stages. No usable bulk expression matrix was located in the inspected materials. Bulk replication was therefore not transferred to the single-cell results. [3] [33]

2.3 Label audit and descriptive calculations

The stored 16-group clustering, `RNA_snn_res.0.1`, was used instead of the object's default 62-group clustering. Cluster 2 in this solution was used as the authors' wound-induced-cell (WIC) group. Across 77 preselected atlas markers, all 154 recomputed within/outside-cluster detection fractions matched Dataset S1 to its three-decimal rounding. Every stage's captured-cell total matched Table S2, summing to 205,747 cells. RNA unique molecular identifier (UMI) totals and detected-gene totals also matched deposited metadata for every cell. These are technical consistency checks on the same dataset, not independent biological validation. [31] [33]

Captured-cell composition was calculated as WIC-assigned cells divided by all retained captured cells at a stage. For each gene and cell, RNA expression was normalized as $\log_{10}(10000 \times \text{gene UMIs} / \text{total cell RNA UMIs})$. Means retain zero-count cells. Detection is the fraction of cells with at least one gene UMI and is reported separately.

The 20-gene signature is the equal-gene mean of the top 20 positive cluster-2 markers ranked by Dataset S1 fold change before inspecting their time courses. It is a descriptive signature, not a validated pathway-activity or stemness score. Three other audit-panel genes absent from some feature lists were excluded rather than assigned artificial zeros; all signature genes and named annotation candidates remained available across stages.

Only maxima at actual sampled times are reported. No animal-level confidence intervals, cell-level significance tests, interpolated onset times, or decay constants were estimated. Stored

gene annotations are treated as candidates unless an authoritative gene-ID/symbol correspondence is available. Pseudotime is not substituted for elapsed sampling time.

3. Literature evidence: inputs, internal responses, and distinct repair endpoints

3.1 Mechanical conditions can influence organizer activity

In Hydra tissue spheroids, suppressing osmotic inflation prevented normal Wnt3 organizer activity and regeneration, while sustained Wnt3 expression or pre-existing organizer tissue could bypass the usual mechanical requirement. This provides evidence for a functional connection between mechanical conditions and organizer state. Restoring normal medium, however, restores continuing water influx and stretch. Measurements during that renewed regime are not observations after a completed finite stretch pulse. [1]

Other experiments extend the mechanical picture without completing that post-input comparison. Microaspiration characterizes deformation, flow, and rupture of regenerating Hydra tissue, but does not establish subsequent organizer activation by a brief alternative stimulus. Repeated local strain events occur near future head sites during regeneration, with strain-to-morphogen coupling partly represented by a model. Altered contraction rates in Aurelia can change stable body shape, supporting an active mechanical contribution to form rather than a demonstrated retained state after an external cue. [9] [10] [11]

Intracellular water remains relevant as a possible physical component. In mammalian cultured cells, osmotic volume changes alter cytoplasmic mechanical behavior, and much of the measured indentation relaxation occurs on a sub-second scale under the reported conditions. This is evidence for intracellular poroelastic behavior, not a measured lifetime for regenerative memory in a jellyfish. Mesogleal rheology addresses extracellular material and cannot replace an intracellular measurement. [4] [8]

3.2 Electrical conditions alter tissue state; withdrawal matters

Prolonged alternating electrical stimulation can reversibly drive regenerated Hydra tissues toward a spheroid-like state. Calcium-probe activity increases while Wnt3 organizer signal declines during reversal; setting voltage to zero permits renewed Wnt3 expression and regeneration. The response depends on stimulus conditions: changing frequency at the same reported voltage can alter the outcome. These observations establish electrical control and a genuine post-removal regrowth outcome, but do not isolate a lasting intracellular mediator produced by a brief naturalistic pulse. [2]

Calcium activity alone is not an adequate marker of regenerative benefit. The same study's extracellular-potassium comparison increased calcium-probe activity and caused tissue folding without the corresponding recoverable regenerative outcome. Initial wound sealing also preceded electrical exposure in the spheroid experiments. A change in activity must therefore be interpreted alongside tissue state and the endpoint actually tested. [2]

Implanted-electrode pacing in Aurelia establishes control of contractions and swimming during stimulation. Later embodied-computing work extends analysis of stimulated body dynamics, but neither supplies a post-off repair assay. Electrode voltage cannot be equated with ambient field strength at a naturally exposed animal, especially across different electrode geometries and freshwater versus seawater conditions. [12] [13]

An informative external comparison is *Botryllus schlosseri*, a colonial chordate. A finite electrical-treatment regimen was associated with later differences in normal budding, colony output, and other traits. The 35-day budding comparison used seven colonies per group. RNA samples labeled two and 24 hours post-treatment provide reported molecular differences, but the main text does not unambiguously assign them to a particular session schedule or define time zero relative to its first or final session. Separate treatment/control tanks and the absence of a reported electrode-connected zero-current sham leave alternatives unresolved. This is neither a jellyfish wound-regeneration experiment nor a demonstration that the measured RNA changes mediate the later outcome. [23]

3.3 Moving water permits regrowth under some conditions, but the mechanism is unresolved

In amputated Aurelia ephyrae, continuing water motion in a permissive setup was associated with arm regeneration over the recovery period. Outgrowth could contain canals, muscle, neurons, and sometimes a rhopalium, with regenerated arms participating in coordinated pulsing. Frequencies and completeness varied. These findings concern more than sealing, but

not complete restoration in every animal. [14] [15]

Water motion, swimming, posture, loading, feeding access, and transport were not independently isolated. Reduced oxygen could also promote regeneration in the permissive setup, so increased oxygen supply alone is not an adequate explanation of the current effect. Nutritional interventions provide further candidates without resolving their interaction with flow. The earlier mechanically driven symmetrization experiments concern rearrangement of surviving tissue, a different endpoint from arm replacement. The current-screen data can describe tested associations, but cannot supply unmeasured intracellular variables or absent factorial controls. [14] [15] [16]

3.4 Closure, remodeling, and functional replacement are separable

The distinction between repair endpoints is experimentally consequential. In *Clytia*, hydroxyurea treatment impaired manubrium outgrowth without preventing initial closure or umbrella remodeling. This pharmacological contrast is useful without assuming every drug effect is exclusively proliferation-specific. Extracellular ATP also affects *Clytia* epithelial closure and actin organization, but was not established as a pulse-and-washout demonstration of a retained state. Mammalian calcium- and ATP-dependent membrane resealing concerns the barrier of individual cells, not the same compartment or experiment. [17] [6] [5]

Table 1. Selected repair endpoints and their interpretation.

Organism and context		Observed endpoint	Important distinction
Clytia medusae		Closure by about 12 h; feeding-functional manubrium by day 4 in 43/44 untreated animals. [17]	Closure/remodeling can occur without normal organ outgrowth.
Cladonema	bulb-retaining	Closure by 24 h; prey capture in 55% at 48 h and 100% at 72 h, n = 36 per reported time-point assay. [18]	Functional replacement exceeds sealing; contributions of repair-specific and resident cells differ.
Halicystus tentacles, preprint		Both tested types sealed by 48 h; secondary tips regrew by 168 h while primary tips did not within that window. [7]	Developmental/tentacle contexts differ; this is not permanent or class-wide incapacity.
Tripedalia	rhopalia, abstract-only evidence	Rhopalial/neural regrowth, renewed pacemaking, and lens-eye light responses were reported. [19]	Structural and functional recovery were assessed; learned-state control of repair was not.

The *Halicystus* result remains preliminary: primary and secondary assays used small animal groups, with within-animal comparisons, and proliferation was not isolated by a selective blockade/rescue. Life-cycle reversal is another distinct endpoint. The historical *Turritopsis nutricula* report describes medusa-to-hyroid reversal, not a demonstrated water mechanism or indefinite survival of every individual. [7] [30]

4. Aurelia rhopalium regeneration: published perturbations and new descriptive results

4.1 What the original experiments establish

Li and colleagues identified an early wound-induced-cell population associated with rhopalium regeneration and tested molecular requirements for blastema formation. After knockdown, blastemas formed in 0/16 Sox2-treated individuals and 0/16 Otx5b-treated individuals, compared with 15/16 controls; Wntless knockdown yielded 4/16. These are the authors' animal-level perturbation results, not new experiments or tests from the present analysis. They support gene functions under the reported conditions without establishing selective necessity of the entire WIC population, which was not specifically ablated. [3]

Regenerating rhopalium recovered anatomical structures and supported progressive swimming recovery. The article describes wound healing at 0–1 dpa, blastema development at 1–3 dpa, later statocyst and ocellus formation, and continued growth to day 14. Swimming recovery is a functional outcome, but not an assay of complete recovery of every sensory capacity. Inferred

cell trajectories and expression-based communication analyses remain distinct from direct lineage tracing. [3]

4.2 Observed captured-cell time course

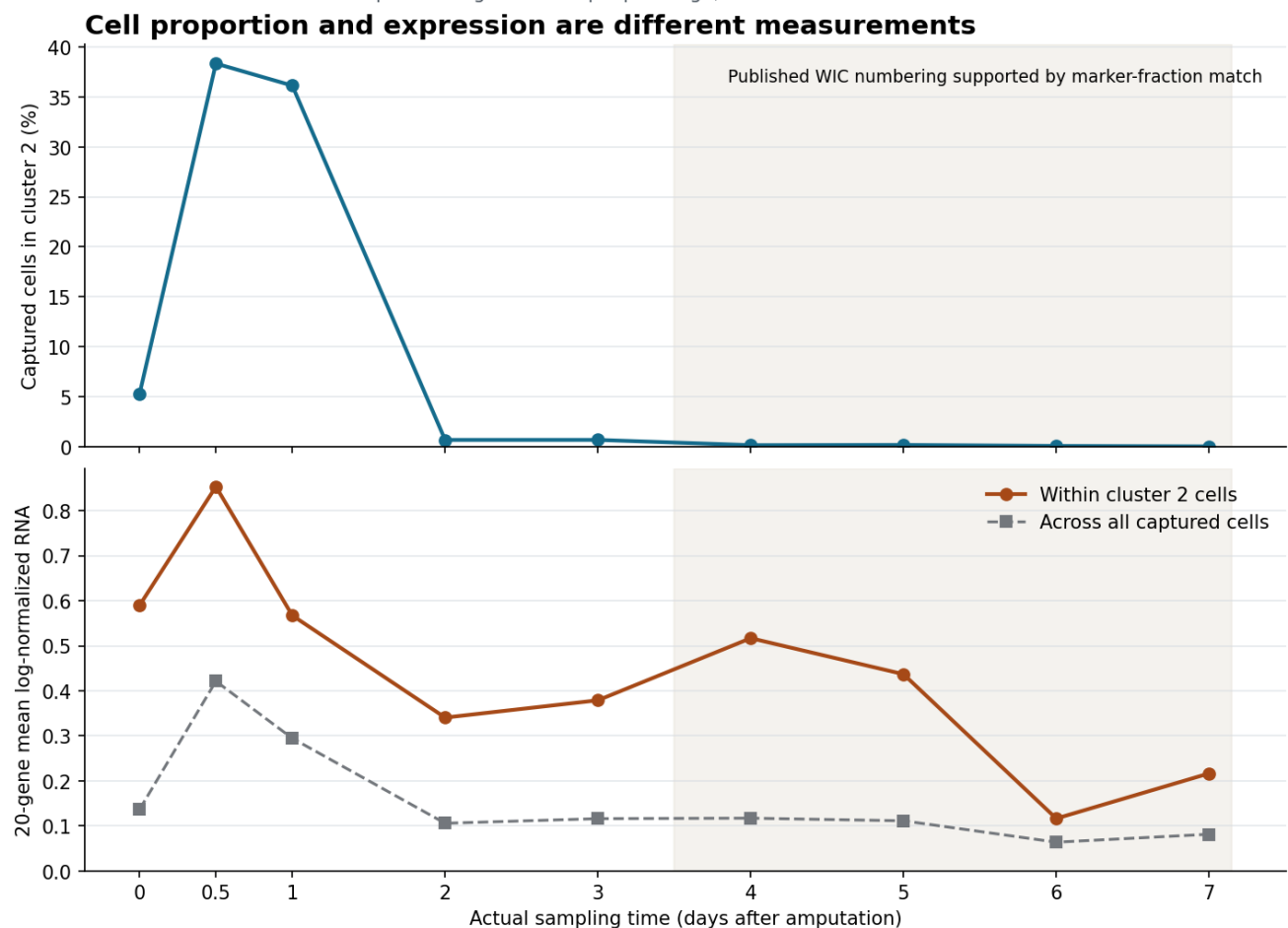
The reanalysis resolves the early dynamics in actual sampled time rather than only broad phase labels. The maximum WIC **fraction** occurs at 0.5 dpa, while the maximum captured WIC **count** occurs at day 1. These are different summaries because total captured cells also vary. Neither is an absolute tissue abundance estimate. Results derive from the authors' processed atlas and supplementary marker reference. [31] [33]

Table 2. Captured-cell composition and within-WIC RNA signature.

Days	Captured total	WIC count	WIC share (%)	Within-WIC 20-gene mean
0	10,904	572	5.246	0.589
0.5	21,079	8,082	38.341	0.853
1	29,796	10,767	36.136	0.567
2	17,943	119	0.663	0.341
3	20,291	135	0.665	0.379
4	15,207	21	0.138	0.517
5	25,300	41	0.162	0.437
6	37,454	23	0.061	0.116
7	27,773	4	0.014	0.217

The article's narrative describes cluster-2 proportions as peaking during blastema formation. The deposited stage summaries put the numerical maximum at 0.5 dpa, with a similarly high fraction at day 1. The new draft therefore reports the exact sampled stages and flags the broad phase wording for clarification; it does not silently move the samples or reinterpret captured fractions as tissue cell counts. [3] [31]

One pooled single-cell sample per stage; no animal-level error bars



Shaded: only 4–41 WICs per stage. Lines connect observations; they are not inferred trajectories.

Figure 1. Captured WIC fraction and the preselected 20-gene signature. Expression means within WICs are separated from means across all captured cells. Lines connect observations, not inferred trajectories. Shaded late stages contain only 4–41 WICs each. There are no animal-level error bars because separately measured replicate pools are unavailable.

The signature's highest within-WIC mean is at 0.5 dpa. Later apparent rebounds have small denominators: the day-4 mean describes 21 cells and the day-7 mean four. The article's neural cluster 5 has a captured share of 3.26% at day 0, a sampled maximum of 11.32% at day 4, and 9.09% at day 7. Sensory hair-cell cluster 8 changes from 5.36% to 1.01% between days 0 and 7. These shifts do not demonstrate WIC-to-neuron conversion. [3] [31]

4.3 Marker expression is not interchangeable with population representation

Dataset S1 contains protein-description annotations, not a certified Aurelia gene-symbol crosswalk. The gene IDs below identify the measured rows; their biological names remain candidates. The published gene-symbol knockdowns and these RNA rows should not be treated as an independently verified gene-specific causal chain without that crosswalk. [3] [33]

Table 3. Candidate-marker mean log-normalized RNA within WICs.

Gene	Mean 0 d	Mean 0.5 d	Mean 2 d	Within-WIC sampled maximum
Aco03402	0.347	1.869	0.120	0.5 d
Aco10552	0.293	0.778	0.175	0.5 d
Aco03861	0.168	0.331	0.093	0.5 d
Aco07511	0.778	2.248	1.226	0.5 d
Aco20833	1.698	1.152	0.256	0 d

Candidate labels are Col6a5 (Aco03402), Otx5b (Aco10552), Fzd10 (Aco03861), Wntless (Aco07511), and Plg (Aco20833). Table 3 abbreviates the row identifiers by omitting the gene-prefix.

The Plg-annotated candidate gene-Aco20833 demonstrates why the distinction matters: its highest within-WIC mean occurs at day 0, whereas its mean across all captured cells is highest at day 0.5. A whole-sample maximum need not represent the same expression timing inside a cell type. Conversely, the Col6a5, Otx5b, Fzd10, and Wntless candidates listed here have their highest within-WIC mean at day 0.5.

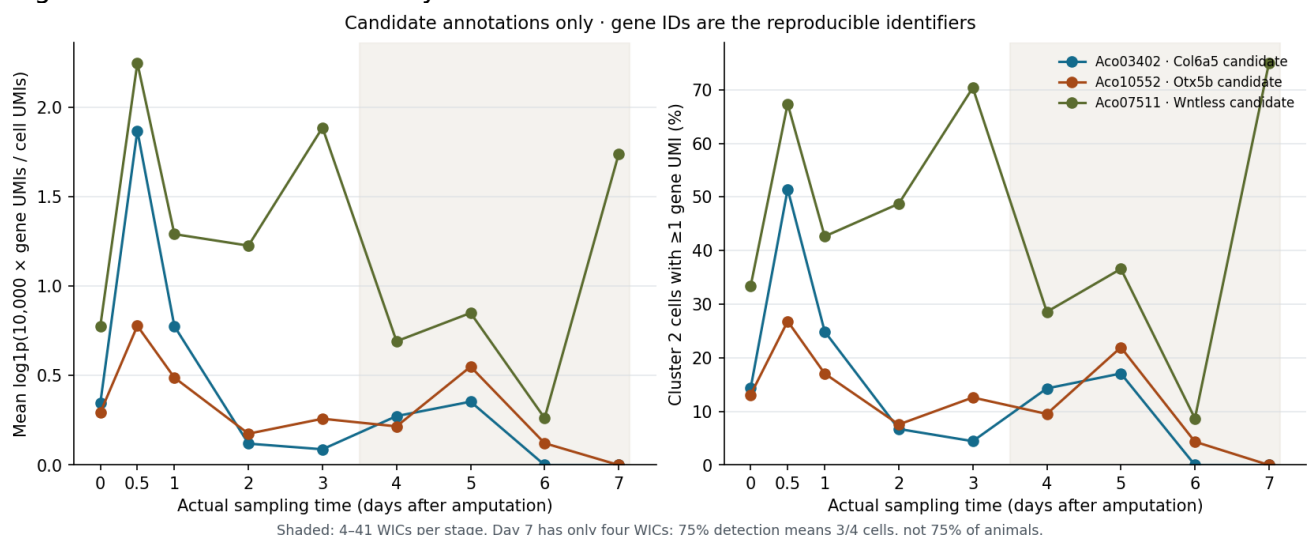


Figure 2. Candidate-marker normalized means and detection fractions are separate measurements. At day 0.5, Col6a5-, Otx5b-, and Wntless-annotated candidates are detected in 51.4%, 26.8%, and 67.4% of WICs. The day-7 Wntless-candidate detection of 75% represents only 3/4 captured cells, not 75% of animals or a reliable late activation estimate.

Both Plg candidates and both Piwi-like candidates are retained in the complete result tables because their timing differs. SoxF2/SOX18 annotations do not establish equivalence to the supplement's Sox2 label. Wnt1 and Wnt3 lacked exact description matches in the inspected workbook. An opsin candidate has rare day-0 UMIs, but an uncertified gene/subcluster mapping

cannot independently confirm or refute the paper's reported photoreceptor onset. [33]

5. Integrated interpretation

5.1 A changing repair program is not yet a same-cell persistence measurement

The combined Aurelia evidence is stronger than a morphological description alone: published gene perturbations identify functional requirements, while the reanalysis quantifies a rapidly changing early captured population and its RNA profile. Their combination supports a temporally structured post-injury response. It does not show that one identified cell retains a state across days, because sequencing destructively samples different cells at each stage.

This distinction is compatible with active maintenance. A post-input state need not be passive or require the organism to become inactive. It would need an identified variable that remains different from an appropriate control after a verified external cue ends. A lesion, continuing exposure, recurrent muscle activity, a transcriptional program, and altered neural responsiveness can all last for different reasons; duration alone does not identify a common mechanism.

Recent Hydra cell-state work supports tracking changing positional identities and organizer organization over time rather than treating recovery of shape as the only endpoint. A separate calcium/polarity study supplies mechanochemical context but was available here only through abstract and figure captions. Neuromechanical and neural-network models generate additional hypotheses; computational aftereffects must remain identified as predictions rather than measured post-input cellular memory. [21] [22] [28] [29]

5.2 Learning, natural exposure, and repair require an explicit connection

Tripedalia learning experiments establish associative changes in behavior and isolated-rhopalium responsiveness. The reported post-training response is relevant, but the study does not define a stimulus-free delay demonstrating retention for hours or days, nor does it test repair. Combining evidence that a species learns with separate evidence that it regenerates a rhopalium does not establish that learned state controls regeneration. [20] [19]

Naturalistic exposure is a separate measurement problem. Prolonged low-frequency playback produced later statocyst lesions in two scyphozoan species, demonstrating a post-exposure tissue effect under those conditions. It was high-level exposure with an injury endpoint, not evidence of a brief beneficial cue; particle motion at the animal was not measured. Aglantha circuit physiology addresses internal signaling, not a calibrated remote heartbeat-detection threshold. [24] [25]

Marine bioelectric source measurements and immersed human ECG recordings also do not establish the electric gradient received by a jellyfish. Source geometry, distance, conductive medium, duration, and receiver sensitivity are required. Heartbeat-associated mechanical motion and cardiac electrical activity must be considered separately. The located evidence supplies no defensible heartbeat-to-jellyfish detection distance or repair effect. [26] [27]

5.3 The remaining causal comparison

The strongest open question is not whether physical conditions matter; they already do in the scoped experiments. It is whether a **separate, completed, naturally realistic cue** produces an identified post-off state that changes a specified repair outcome in the same biological comparison.

That connection would require verified receiver exposure and cessation, a post-input internal measurement, an outcome that distinguishes closure from remodeling or functional replacement, and evidence that the internal change contributes to the outcome rather than merely accompanies it. These are evidential requirements, not a laboratory protocol or a claim that the missing experiment has been performed. Intracellular hydration remains one possible variable to measure, not an inferred cause supplied by a lumen, calcium probe, gene signature, or mesogleal measurement.

6. Limitations

The literature is selective, biologically heterogeneous, and includes single-study findings, historical primary evidence, an unreviewed Haliclystus preprint, and access-limited records.

Tripedalia regeneration, Aglantha circuitry, and historical Turritopsis reversal are abstract-only in this source set; the 2026 Hydra polarity record adds figure captions, not a full-text assessment. Thirty-three cited research/data records are not thirty-three independent studies or replications. [7] [19] [22] [25] [30]

For the secondary analysis, one pooled sample per stage prevents estimation of between-animal or between-pool variation. Dissociation, capture efficiency, stage-associated batch differences, and sequencing depth may affect comparisons. Median UMIs vary across stages; normalization does not replace a coverage-matched sensitivity analysis, which was not performed. Candidate annotations and very small late WIC groups further limit marker interpretation. RNA measurements do not directly establish protein activity.

Neither the single-cell atlas nor the located literature completes a pulse-withdrawal, intracellular-hydration, or heartbeat-to-repair comparison. A missing variable cannot be recovered by reinterpretation of a different measurement. These limits constrain the proposed connection without erasing the directly observed mechanical, cellular, and regenerative findings.

7. Conclusion

Physical conditions and cellular programs have experimentally consequential roles in medusozoan tissue behavior, and closure, remodeling, and organ replacement are separable outcomes. The Aurelia reanalysis adds a reproducible early repair-cell time course: captured WIC share and the selected within-WIC signature are highest at 12 hours, with high representation still present at 24 hours and a sharp fractional decline by 48 hours. Individual marker timing is not uniform.

The resulting research model is specific: physical conditions, internal activity, cell composition, and tissue reconstruction can interact, but each must be measured on its own terms. The complete separate-cue-to-retained-state-to-repair sequence remains a testable open connection rather than a demonstrated common mechanism.

Data availability and manuscript status

External data and supplements are linked below. Reproducible extraction, audit, calculation, and test instructions are retained in research/aurelia-timecourse/README.md; the compilation folder contains the evidence ledger, reference crosswalk, input hashes, and rendering instructions. The supplied synthesis and original analysis remain unchanged. No raw-read alignment, new laboratory experiment, corpus revision, app change, or publication was performed. This manuscript is an AI-assisted working compilation, not an independently peer-reviewed paper.

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