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Four Modifiable Levels of Replicative Capacity: Centrioles, Progenitors, Gametes, Nutrition

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Abstract

Ageing research is rich in candidate mechanisms and poor in operational readouts. The hallmarks framework organises damage into a coherent list [1,2], yet a laboratory that wants to slow replicative decline still has to choose which quantity it will actually measure, and a list of twelve mechanisms does not indicate which one should be perturbed first. We argue that replicative capacity — the number of divisions a cell lineage can complete before it loses function — is set at four partly independent levels, and that each level has a distinct, already available intervention and a distinct way of being wrong. The four are: structural inheritance of the mother centriole, resilience of tissue-resident progenitors to senescence, reconstitution of gametes from somatic cells, and bioprocess-based modification of nutrition. For each level we state three things: the intervention, the readout that would count as success, and the observation that would falsify the claim. That triad is what separates a declaration from an experiment. Our operational definition of replicative capacity is deliberately narrower than "healthspan" and wider than the Hayflick limit: it is measured by population doubling level, clone size distribution, or the fraction of cells retaining a lineage marker after serial passage, and it excludes organism-level endpoints, which we do not address. We then give a single multicentre-style test matrix that could be run with existing methods, with a realistic time to a first result for each level, and we add two design rules: no level is scored on a marker alone, and a negative result at one level is informative rather than fatal because the levels are claimed to be partly independent — which is also the matrix's main vulnerability, since partial independence is easy to assert and hard to demonstrate. We are also explicit about how thin the published evidence base still is at three of the four levels, and about the self-citation that enters through the authors' own institution. The purpose of the paper is not to propose a new theory of ageing; it is to make the existing claims testable in the same units.

Keywords: replicative capacity; centriole inheritance; mesenchymal progenitors; cellular

senescence; in vitro gametogenesis; bioprocessed food; ageing biomarkers

1. Introduction

Two decades of work have turned ageing from a vague observation into a list of mechanisms: genomic instability, telomere attrition, epigenetic alteration, loss of proteostasis, disabled macroautophagy, mitochondrial dysfunction, cellular senescence, stem-cell exhaustion, altered intercellular communication, chronic inflammation and dysbiosis [1,2]. Stem-cell exhaustion in particular now has a mechanistic literature of its own [20]. The list is useful for orientation and unhelpful for measurement. A list of twelve mechanisms does not tell an experimenter what to perturb first, nor how to recognise success when it appears.

The problem is not theoretical but administrative. Cell senescence has a working definition, a validated marker panel and a recommended reporting standard [3], and senolytic interventions have a discovery pipeline with a documented translation path [4,5]. Molecular clocks give a numerical readout with quantified error [6]. These are exceptions. For most of the mechanisms in the list, "improvement" is asserted rather than measured, and the assertions rarely share units with one another.

This review takes a deliberately narrow question: **at how many independent points can replicative capacity be modified with methods that exist today, and what would count as evidence at each point?**

We identify four such levels. Each has a different physical substrate, a different timescale, and — importantly — a different failure mode. We do not claim that these four exhaust the biology. We claim that each is currently actionable, and that treating them as separate levels rather than as one aggregated "anti-ageing" claim makes the field's disagreements resolvable.

1.3.1. *1.1 What "replicative capacity" means here*

We use the term operationally: the number of complete divisions a lineage can perform under defined conditions while retaining its functional phenotype, measured by population doubling level, clone size distribution, or the fraction of cells retaining a lineage marker after serial passage. This is deliberately narrower than "healthspan" and deliberately wider than the Hayflick limit. It excludes organism-level endpoints, which we do not address.

2. Level 1 — Structural inheritance of the mother centriole

2.1 The case for a structural counter

The centriole pair is not reproduced by fission of an existing structure; a new (daughter) centriole is assembled orthogonally on the wall of the mother, and the two resulting centrosomes differ in age, in protein composition and in their fate at division [7,8]. Of all cellular structures, this makes the centriole pair the most plausible physical carrier of a division count that cannot be reset by simple dilution.

Direct evidence that structural age is differentially apportioned exists for mitochondria: aged components are sorted asymmetrically between daughter cells, and blocking that sorting reduces

stemness [10]. The parallel argument for centrioles is older but weaker. It rests on three observations: that mother and daughter centrioles differ in maturation state and in recruitment of surrounding material during the cycle [8,9]; that the geometry of the assembly reaction constrains what can be inherited; and that manipulating the assembly machinery changes both centrosome number and cell-cycle re-entry [7].

The geometric-persistence proposal formalises the third observation: if daughter centrioles are constrained to inherit the mother's dimensions within a tolerance set by the assembly reaction, then shape acts as a heritable, slowly drifting quantity and can stand in for a division count [16]. This is a hypothesis with a numerical prediction — drift per division bounded by assembly tolerance — and it is falsifiable by measuring daughter centriole dimensions across a known number of divisions in a lineage-traced system.

2.2 Intervention and readout

The intervention at this level is not a drug but a perturbation of assembly fidelity: partial depletion or overexpression of core duplication factors, or control of the timing between centriole disengagement and the next S phase [7]. The readout is a joint distribution of daughter centriole dimensions and cumulative divisions, with an explicit test of whether the dimensions drift monotonically and irreversibly.

2.3 How this level can be wrong

Three ways. First, drift may be a passive consequence of damage rather than a cause of decline; distinguishing them requires an intervention that changes drift without changing damage burden, which we do not currently have. Second, the tolerance may be far larger than any biologically meaningful division count, making the counter useless over a lifetime. Third, the measurement may be confounded by fixation artefacts, since centriole dimensions are at the edge of conventional light microscopy resolution — correlative light and electron microscopy is required, which caps throughput.

3. Level 2 — Resilience of tissue-resident progenitors

3.1 The case for intervention at the progenitor

Cellular senescence is the best-characterised damage response in the field: it has a consensus definition, a marker panel, and a reporting standard [3]; senescent cell clearance produces measurable functional improvement in animal models, the pharmacology of senolytics has a documented translation path [4,5], and a first human study reported a measurable reduction of senescent cell burden together with clinical improvement [21]. Two consequences follow. First, senescence is the one level at which intervention is already clinically arguable. Second, the effect size is limited by what the surviving progenitors can do.

This reframes the question: instead of only removing senescent cells, ask what makes a progenitor resistant to entering senescence in the first place. Reported work on mesenchymal progenitors describes subpopulations that retain proliferative capacity and tissue-supporting activity under conditions where sibling populations arrest, and treats that resilience as a selectable property rather than an accident [11,12]; the wider mesenchymal literature documents the same decline and several proposed rejuvenation routes [22,23]. If resilience is heritable within a lineage, it is a

target; if it is a transient state determined by the niche, it is not.

3.2 Intervention and readout

The intervention is selection and expansion of resilient progenitors, or conditioning of the niche so that resilience is favoured. The readout is the fraction of progenitors that remain marker-positive and division-competent after a fixed number of passages under a fixed stress, together with the composition of their secretome. The latter matters because a "resistant" progenitor that secretes a pro-inflammatory programme is not an improvement [3].

3.3 How this level can be wrong

The dominant risk is selection artefact: expansion in culture selects for whatever grows in culture, which may correlate with resilience in a dish and not in a tissue. A secondary risk is definitional — if "resilience" is inferred from the absence of a single marker, it inherits every problem of single-marker senescence detection [3]. Any test at this level must therefore report at least two independent senescence markers plus a functional division endpoint.

4. Level 3 — Reconstitution of gametes from somatic cells

4.1 Why this level is the hardest test of the same variables

Germ-cell development is the one process where replicative capacity and structural inheritance are deliberately reset rather than maintained: the germline re-acquires totipotency, and the centriole is eliminated or remodelled in a sex-specific way, and that elimination is an actively regulated process rather than an incidental loss [13,15,24]. Reconstituting gametes from somatic cells therefore compresses the entire problem into one experimental system. If a somatic nucleus can be returned to a state competent for meiosis, the reset is possible in principle; if it cannot, the reset has a barrier that the first two levels do not touch.

Progress in vitro has been genuine but bounded. Reconstitution of germ-cell development from pluripotent stem cells has been achieved in mice through defined stages [18], and the disposal of the maternal centrosome is now understood as an ordered, experimentally tractable step [25]. The species gap is widening quickly: the developmental timing, the somatic support cells and the meiotic entry requirements differ enough that mouse protocols do not transfer directly, and each stage has to be re-established rather than translated [19]. Reports of oocyte-like cells derived from somatic or stem sources must be read against that background — the burden of proof is not the presence of a marker but the completion of meiosis with correct chromosome segregation and the production of viable offspring, or at minimum a defensible meiosis-specific readout.

4.2 Intervention and readout

Intervention: staged differentiation with defined morphogens, plus deliberate control of centriole elimination and of the maternal-to-zygotic transition timing. Readouts: meiosis-specific recombination markers, chromosome spreads with normal synapsis, and — the decisive one — ploidy and imprinting status of the resulting cells.

4.3 How this level can be wrong

Two failure modes dominate. Marker-only claims: a cell expressing a germ-cell marker is not a gamete, and the literature has repeatedly confused the two. And species extrapolation: a protocol validated in one mammal does not transfer to another without re-validating each stage [18]. We therefore place this level last in any test matrix, not first, despite its conceptual centrality.

5. Level 4 — Bioprocess-based modification of nutrition

5.1 Why a nutrition level belongs in a mechanistic review

If the first three levels act on cells, the fourth acts on the substrate supply. Bioprocessing — controlled germination, fermentation, enzymatic hydrolysis — changes the bioavailable fraction of a food without changing its genetics, and the resulting products can be standardised in a way that raw agricultural material cannot. This is the only level at which intervention in humans can be argued with existing regulatory frameworks, and therefore the only level that can currently generate population-scale evidence.

A concrete case is controlled germination of wheat, in which the protocol is the intervention: timing, temperature and humidity determine the accumulation of free amino acids, γ-aminobutyric acid and phenolic compounds and the activity of hydrolytic enzymes, and therefore the measured bioactivity [26]. A published protocol describes production of a shelf-stable activated wheat with nutritional profiling and bioactivity assessment [17]. Two features make it methodologically interesting rather than merely applied: the product is stable enough to be distributed (so a trial is logistically possible), and the bioactive content is a function of a documented process (so the intervention can be replicated and, if needed, dialled up or down).

5.2 Intervention and readout

Intervention: a standardised bioprocess with specified parameters. Readout: composition (a defined panel, not "antioxidant capacity"), then a functional endpoint in a controlled setting — glycaemic response, satiety, or a marker of low-grade inflammation — with pre-registration of the endpoint before the trial.

5.3 How this level can be wrong

The characteristic failure is over-attribution: a food product accumulates multiple changes at once, and any single measured benefit is confounded by the rest of the diet. The second failure is assay inflation — in vitro antioxidant assays have poor predictive value for in vivo outcomes and should not be used as primary evidence. Both failures are avoidable by pre-registration and by reporting composition and functional endpoint separately.

6. A four-level test matrix

The value of separating the levels is that they can be tested in parallel with a shared reporting standard. Table 1 states, for each level, the intervention, the primary readout, the falsifying observation, and the realistic time to a first result.

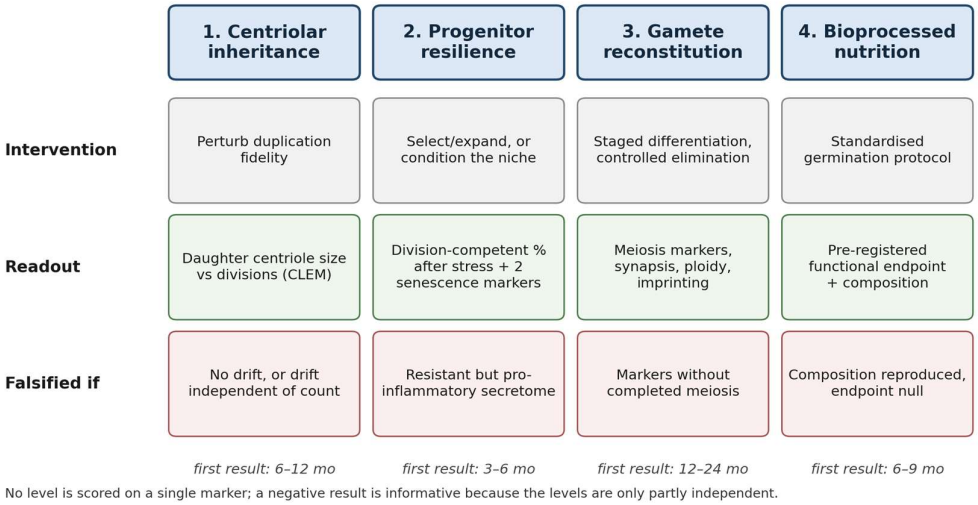


Figure 1. The four levels as a test matrix: intervention, primary readout, falsification criterion and a realistic time to a first result. The panels restate Table 1 in the form used in the study-design discussion; no new data are shown.

Table 1. Test matrix.

Level	Intervention	Primary readout	Falsified if	Time to first result
1. Centriolar inheritance	Partial perturbation of duplication fidelity	Daughter centriole dimension vs cumulative divisions (CLEM)	No monotonic drift, or drift independent of division count	6–12 months
2. Progenitor resilience	Selection/expansion, or niche conditioning	Fraction division-competent after fixed stress, with two senescence markers + secretome	"Resilient" cells show pro-inflammatory secretome or lose function in vivo	3–6 months
3. Gamete reconstitution	Staged differentiation with controlled centriole elimination	Meiosis-specific recombination, synapsis, ploidy and imprinting	Correct markers without completed meiosis	12–24 months
4. Bioprocessed nutrition	Standardised germination protocol	Pre-registered functional endpoint + composition panel	Composition reproduced but functional endpoint null	6–9 months

Two design rules follow from the matrix. First, no level is scored on a marker alone; each needs a functional endpoint. Second, a negative result at any level is informative rather than fatal, because the levels are claimed to be partly independent — which is also the matrix's main vulnerability, since partial independence is easy to assert and hard to demonstrate. Demonstrating it requires a two-level intervention with a factorial design, and we are not aware of such a study.

7. Limits of the present evidence

We state these plainly, because the field's credibility problem is not a shortage of ideas.

The evidence base for levels 1–3 is thin and largely pre-clinical; for level 4 it is thin and largely product-specific. Five of the works we cite most directly [11–15] were published by the open-

access journals operated by the authors' own institution, which is a conflict we disclose rather than conceal (see Conflicts of Interest). Their claims should therefore be treated as hypotheses with a described method, not as independent confirmation. For level 1, the central measurement is near the resolution limit of the instruments most laboratories own. For level 3, the gap between mouse and human developmental timing is not a technical detail but a barrier that has not been crossed. For level 4, the strongest available study designs are still small.

There is also a framing risk. Calling four things "levels" implies an ordering and a completeness that we cannot defend. They are four actionable points, chosen because each has an available intervention and a measurable failure mode. Other points exist, and some of them may be more important.

8. Conclusions

Replicative capacity can be modified at four separable points with methods that exist today: the fidelity of centriolar inheritance, the resilience of tissue progenitors, the reconstitution of gametes from somatic cells, and the bioprocess by which food is made bioavailable. Each point has a specific intervention, a specific readout and a specific way of being wrong, and stating the three together is what turns a claim into an experiment. The immediate implication for practice is procedural: pre-register the endpoint, report a functional readout alongside any marker, and use the same units across levels so that results can be compared. The immediate implication for the field is less comfortable: at three of the four levels, the published base is too thin to support clinical language, and the fastest way to strengthen it is to run small, well-reported studies rather than to add further frameworks.

Author Contributions

Conceptualization: Gabro Gakely, Laura Gegelia, Alexander Taktakidze. Methodology: Gabro Gakely, Alexander Taktakidze. Formal analysis: Laura Gegelia. Investigation: Gabro Gakely, Laura Gegelia, Alexander Taktakidze. Writing — original draft preparation: Gabro Gakely, Laura Gegelia, Alexander Taktakidze. Writing — review and editing: Gabro Gakely, Laura Gegelia, Alexander Taktakidze. Project administration: Gabro Gakely. All three authors have read and agreed to the published version of the manuscript.

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Data Availability Statement

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Conflicts of Interest

Gabro Gakely, Laura Gegelia and Alexander Taktakidze are all affiliated with Georgia Longevity Alliance (GLA) — a non-entrepreneurial (non-commercial) legal person registered in Georgia (NAPR reg. No. 404506520), 47 I. Javakhishvili St., Tbilisi 0170, Georgia — the organisation that operates the open-access journals in which references [11–15] were published. The authors declare no financial interest in any product discussed. GLA is not a commercial publisher: neither journal charges article processing fees. Readers should weigh the self-citation accordingly; we have kept it to the minimum needed to represent the described protocols.

Use of Generative AI

No generative AI tools were used to draft or edit the scientific content of this manuscript; the authors are responsible for all statements.

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Note on transliteration. References 16 and 17 name the same co-author, Jaba Tqemaladze (ORCID 0000-0001-8651-7243). The two spellings are exactly those registered in the metadata of the respective publishers — Tqemaladze for the Cytoskeleton article (10.1002/cm.70222) and Tkemaladze for the Preprints.org record (10.20944/preprints202508.1997.v1) — and each entry has been kept as published so that both resolve to the deposited record.

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დაბერების ბიოლოგია მექანიზმების სიმრავლეს გვთავაზობს, მაგრამ ექსპერიმენტს ხშირად არ აქვს ერთი, წინასწარ შეთანხმებული სიდიდე, რომლითაც „გაუმჯობესება“ გაიზომება. ჰოლმარკების სქემა ზიანს ერთ სიად აწყობს [1,2], თუმცა ამ სიიდან არ გამომდინარეობს, რომელი პარამეტრი უნდა შეიცვალოს პირველად და რა უნდა ჩაითვალოს წარმატებად. მიმოხილვაში ვსვამთ ვიწრო და ოპერაციულ კითხვას: რამდენ დამოუკიდებელ წერტილში შეიძლება რეპლიკაციული უნარის შეცვლა დღეს არსებული მეთოდებით და რა ჩაითვლება მტკიცებულებად თითოეულ მათგანში.

რეპლიკაციულ უნარს ვიგებთ ოპერაციულად: ეს არის სრული დაყოფების რაოდენობა, რომელსაც უჯრედული ხაზი განსაზღვრულ პირობებში ასრულებს ფუნქციური ფენოტიპის შენარჩუნებით — იზომება პოპულაციური გაორმაგების დონით, კლონის ზომის განაწილებით ან ხაზის მარკერის შემნარჩუნებელი უჯრედების წილით. ასეთი განსაზღვრება განზრახ უფრო ვიწროა, ვიდრე „ჯანსაღი სიცოცხლის ხანგრძლივობა“, და უფრო ფართო, ვიდრე ჰეიფლიკის ზღვარი.
გამოვყოფთ ოთხ დონეს, რომელთაც განსხვავებული ფიზიკური სუბსტრატი,

განსხვავებული დროითი მასშტაბი და — რაც მთავარია — განსხვავებული მარცხის რეჟიმი აქვთ: დედა ცენტრიოლის სტრუქტურული მემკვიდრეობითობა; ქსოვილის პროგენიტორების რეზისტენტობა სენესცენციის მიმართ; გამეტების რეკონსტიტუცია სომატური უჯრედებიდან; კვების ბიოპროცესული მოდიფიკაცია. თითოეული დონისთვის ვაყალიბებთ სამ რამეს: ჩარევას, იმ მაჩვენებელს, რომელიც წარმატებად ჩაითვლება, და იმ დაკვირვებას, რომელიც მტკიცებას უარყოფს. სწორედ ეს სამეული ქმნის განსხვავებას დეკლარაციასა და ექსპერიმენტს შორის. ჩვენი განსაზღვრება არ მოიცავს ორგანიზმის დონის საბოლოო წერტილებს, რომლებსაც ამ მიმოხილვაში არ განვიხილავთ.

შემდეგ ვაძლევთ ერთ სატესტო მატრიცას, რომლის გაშვებაც არსებული მეთოდებით შესაძლებელია: ჩარევა, პირველადი მაჩვენებელი, უარმყოფელი დაკვირვება და პირველ შედეგამდე რეალისტური ვადა თითოეული დონისთვის. ვაცხადებთ ორ საპროექტო წესს: ვერც ერთი დონე არ შეფასდება მხოლოდ მარკერით — აუცილებელია ფუნქციური მაჩვენებელი; და უარყოფითი შედეგი ერთ დონეზე საინფორმაციოა და არა ფატალური, ვინაიდან დონეები ნაწილობრივ დამოუკიდებლად მიიჩნევა — რაც ამავე დროს მატრიცის მთავარი სისუსტეა, ვინაიდან ნაწილობრივი დამოუკიდებლობის დაშვება ადვილია, ხოლო დამტკიცება — ძნელი.

ბოლოს ღიად ვამბობთ, რამდენად თხელია მტკიცებულებათა ბაზა: სამი დონისთვის ის ძირითადად პრეკლინიკურია, მეოთხესთვის — პროდუქტსპეციფიკური; ნაწილი ციტირებული ნაშრომი ავტორთა საკუთარი ინსტიტუტის ღია წვდომის ჟურნალებშია გამოქვეყნებული, რაც ინტერესთა კონფლიქტად ცხადდება. მიმოხილვა განკუთვნილია უპირველესად იმ ლაბორატორიებისთვის, რომლებსაც ერთი, წინასწარ შეთანხმებული საზომი სიდიდე სჭირდებათ. მიმოხილვის მიზანი ახალი თეორიის შეთავაზება არ არის — მიზანია არსებული მტკიცებების ერთსა და იმავე ერთეულებში შემოწმებადობა. ახალი ექსპერიმენტული მონაცემი არ არის წარმოდგენილი.

საკვანძო სიტყვები: რეპლიკაციული უნარი; ცენტრიოლის მემკვიდრეობითობა; მეზენქიმური პროგენიტორები; უჯრედული სენესცენცია; in vitro გამეტოგენეზი; ბიოპროცესული საკვები; დაბერების ბიომარკერები