Is Aubry de Grey conducting any research on restoring elastin

There is no clear evidence that Aubrey de Grey is personally running a specific experimental program focused on **restoring** elastin right now, but elastin and loss of tissue elasticity fall under one of the damage categories he has long targeted, and he has helped spin out work in that area.[11][12]

How elastin fits into his framework

- In the SENS/"damage repair" model, the loss of elasticity in arteries, skin, and other tissues is classed as "extracellular matrix stiffening," driven by crosslinking and other changes in structural proteins like collagen and elastin.[12][11]
- De Grey has described supporting a company specifically aimed at "the extracellular stiffening problem of restoring elasticity," though elastin is discussed as part of the broader extracellular matrix rather than as a narrow, elastin-only program.[11]

Current research he is involved in

- As of 2025–2026, his main hands-on work at the LEV Foundation is large multi-intervention mouse rejuvenation studies (rapamycin, senolytics, telomerase, stem cells, etc.), which are systemic aging-damage experiments and not targeted elastin-restoration trials.[13][14]
- Earlier SENS Research Foundation–funded work on tissue elasticity has focused more on understanding and potentially breaking harmful extracellular crosslinks (mainly in collagen) rather than directly rebuilding elastin fibers, although this is mechanistically related to restoring overall tissue elasticity.[15][12]

What this means practically

- If you are specifically asking whether he is running or advertising a lab program like "elastin replacement therapy" or "elastin regeneration project," there is no public indication of such a dedicated project under his direct leadership right now.[14][13][12]

- Elastin biology itself is an active research area (outside his own groups), for example studies on elastin structure, crosslinking, and elastin-derived fragments in aging and disease, but these are not described as de Grey–run programs.[16][17]

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as elastin production stops when we are young, it is essential to fix this problem to increase life span beyond 120 years, this nearly impossible task of restoring elastin is always overlooked, while interventions that will never increase maximal life span are still the focus, this indicates a significant lack of knowledge in the longevity field.

Your point about elastin highlights a real, very hard problem in aging biology, but the situation is a bit more nuanced than "no elastin, no >120 lifespan" or "the field is ignorant."[26][27][28]

What actually happens to elastin

- Elastin is laid down mainly in fetal and early postnatal life; its production drops sharply after maturity and turnover is extremely low, so accumulated damage is hard to reverse.[27][28][26]
- With age, elastic fibers undergo enzymatic degradation, oxidative damage, glycation, calcification, and other modifications, contributing to vascular stiffness, lung and skin changes, and loss of mechanical resilience.[28][26][27]

Is elastin "ignored"?

- In dermatology and cosmetic science, elastin loss is widely discussed but mostly at the appearance level (wrinkles, sagging), not as a systemic life-span determinant.[29][30][26]
- In geroscience, extracellular matrix (ECM) and stiffness are active topics, but most work still focuses on collagen crosslinking, senescent cells, nutrient signaling, and inflammation; elastin-specific regeneration is a smaller niche.[31][32][28]

Why elastin is so technically hard

- You cannot simply “add” elastin: correct assembly requires precise control of tropoelastin expression, crosslinking enzymes, and matrix architecture; mis-deposition can worsen tissue structure.[26][31][28]
- Because adult elastin synthesis is very low, even boosting gene expression may have limited impact, which is why researchers are exploring recombinant tropoelastin scaffolds and targeted nanotherapies to damaged elastin.[33][31][26]

Does ignoring elastin cap lifespan?

- Arterial stiffening and ECM deterioration clearly contribute to cardiovascular risk and age-related frailty, so repairing elastin and related ECM damage would likely be important for pushing healthy lifespan further.[32][28][26]
- However, maximum lifespan is also constrained by many other processes (genomic instability, stem-cell exhaustion, proteostasis failure, immune aging, etc.), so elastin is probably necessary to address but not sufficient alone to “unlock” life beyond 120.[32][28]

Is it a “lack of knowledge” or a prioritization issue?

- Researchers are aware that elastin is a bottleneck and that current interventions to “improve elasticity” (topicals, mild ECM remodeling) are mostly cosmetic and unlikely to shift maximum lifespan.[34][31][26]
- The limited focus reflects difficulty, risk, and funding priorities rather than total ignorance: elastin restoration is recognized as a missing piece, but it is technically demanding and only a few groups and companies are seriously tackling it so far.[35][31][33]

If you’d like, I can walk through what a plausible elastin-restoration strategy might look like (e.g., tropoelastin scaffolds plus targeted removal of damaged fibers) and how close current research is to that.

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Role of elastin in longevity beyond 120 years

Elastin is a major determinant of how long our arteries, lungs, and other elastic tissues can function well, so preserving or restoring **elastin** is likely important for pushing healthy human lifespan upward—but current evidence does not yet let us say it is "the" key that unlocks life beyond 120 years.[41][42][43]

Why elastin matters for survival

- Elastin-rich large arteries buffer each heartbeat, reducing cardiac workload and smoothing blood flow; when elastin degrades, arteries stiffen, blood pressure rises, and heart failure risk increases.[43][41]
- Increased arterial stiffness (driven in part by loss or damage of elastin and a lower elastin-to-collagen ratio) is an independent predictor of cardiovascular mortality in older adults and high-risk groups.[44][45][41]

Elastin, aging, and extreme old age

- Elastin is an ultra-long-lived protein with extremely low turnover; over decades it accumulates fractures, proteolytic damage, calcification, and glycation, which progressively shift load-bearing from elastin to much stiffer collagen.[42][46][41]
- Degeneration of arterial elastic fibers is now recognized as a primary driver of age-related arterial stiffening and inflammatory remodeling, which are central to cardiovascular disease—the main cause of death in later life.[47][42][43]

Could elastin repair be necessary for >120?

- Because cardiovascular disease is the dominant late-life killer and elastin damage is foundational in vascular aging, robust preservation or repair of elastic fibers is very likely one of the prerequisites for safely extending maximum healthy lifespan beyond ~110–120.[41][42][47]
- However, even perfect vascular elastin would not prevent deaths from cancer, neurodegeneration, immune failure, or other systemic aging processes, so elastin repair would need to be combined with multiple other damage-control strategies to enable reliable survival far beyond current human limits.[48][49][50]

How elastin connects to known longevity pathways

- Work in model organisms shows that longevity pathways (for example reduced insulin/IGF-1 signaling) actively remodel the extracellular matrix and help maintain tissue mechanics, supporting the idea that ECM integrity, including elastic elements, is part of the mechanism of extended lifespan.[49][50][48]
- Recent reviews argue that elastic fiber–derived signaling and extracellular matrix dynamics are not just passive damage but active regulators of vascular aging and thus logical targets for next-generation longevity interventions.[47][49]

What we still don't know

- No human intervention has yet been shown to regenerate large-scale arterial elastin and thereby extend maximum lifespan, so the quantitative contribution of elastin repair to living beyond 120 remains speculative, even though the mechanistic logic is strong.[42][43][41]
- Current research is focusing on protecting existing elastic fibers, understanding elastin-fragment signaling, and exploring ways to rebuild or replace damaged elastic matrices; whether these can scale to true life-span extension in humans is still an open question.[49][42][47]

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While the elastin problem is not the key and there are many other problems to solve, if the elastin problem is not solved, what we are left with is increasing health span, not increasing maximum life span.

You are basically right that if we never solve vascular/ECM aging (which heavily involves elastin), most progress will look like "better healthspan within the same rough lifespan ceiling," not a big jump in maximum lifespan.[56][57][58]

Why this intuition is largely correct

- Age-related arterial stiffening, driven by elastin fragmentation, calcification, and replacement by collagen, is a major causal driver of hypertension, stroke, heart failure, and other cardiovascular deaths.[57][58][56]
- Vascular aging is so central that classic clinical aphorisms like “you are as old as your arteries” now have strong mechanistic backing: in later life, cardiovascular disease remains the dominant cause of death.[59][58][60]

Healthspan vs maximum lifespan without elastin repair

- If we improve many hallmarks of aging (better metabolic control, fewer senescent cells, improved proteostasis, etc.) but leave large-artery elastin and ECM to continue degrading, we likely shift disease onset later (healthspan) but still hit a hard wall of vascular events.[61][58][62]
- Observational data (for example “Blue Zone” populations with slower vascular stiffening) suggest that protecting vascular elasticity aligns with more people reaching their eighties and nineties in good health, but it has not yet produced humans consistently living far beyond current maximum ages.[60][57]

Why elastin/ECM is a bottleneck, not the only one

- Recent work frames ECM and mechanotransduction as active regulators of aging: distorted matrix mechanics feed back into cell signaling, inflammation, and stem-cell behavior, so failing to address ECM (including elastin) caps how far other interventions can go.[63][61]
- At the same time, maximum lifespan is also limited by cancer, neurodegeneration, immune failure, and other systemic processes, so elastin repair is best seen as a **necessary but not sufficient** component of any strategy that truly pushes maximum lifespan, not just average healthspan.[62][64]

Where your critique is spot-on

- Major reviews on vascular aging explicitly note that arterial stiffness is a key determinant of cardiovascular mortality, yet translational work on actually rebuilding or truly preserving elastic fibers lags far behind more fashionable targets.[58][56][57]
- That gap is more about technical difficulty and incentives than pure ignorance: restoring a long-lived load-bearing protein network like elastin is much harder than, say, modulating a signaling pathway, but from a lifespan standpoint it is likely just as fundamental.[56][61][62]

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Vascular stiffness impact on maximum lifespan

Vascular stiffness is one of the central physiological processes that pushes people toward the current human lifespan ceiling, so if it is not prevented or reversed, it will strongly limit maximum lifespan even if other aspects of health improve.[72][73][74]

How vascular stiffness shortens late-life survival

- With age, large arteries stiffen because elastin fibers break and calcify and are progressively replaced by stiffer collagen, leading to higher pulse pressure and systolic hypertension.[73][75][72]
- This stiffness drives myocardial strain, stroke, kidney damage, and vascular dementia, making cardiovascular disease the leading cause of death in older adults.[74][73]

Evidence that stiffness predicts mortality

- Measures of arterial stiffness such as carotid-femoral pulse wave velocity or brachial-ankle pulse wave velocity independently predict all-cause and cardiovascular mortality, even after adjusting for traditional risk factors.[76][77][78]
- In large cohorts, each 1 m/s increase in estimated pulse wave velocity is associated with a substantial relative increase in all-cause mortality risk, highlighting stiffness as more than a cosmetic aging change.[77]

Implications for maximum lifespan

- Reviews of vascular aging emphasize that arterial stiffness is a core mechanism underlying cardiovascular diseases that dominate deaths beyond about age 65, so uncorrected stiffness places a de facto upper bound on survival for most people.[72][73][74]
- In practical terms, if we correct many aging pathways but leave arteries stiff and fragile, we mainly delay disease onset (healthspan) but still hit a late-life wall of cardiovascular and microvascular events, limiting any big extension of maximum lifespan.[79][74][72]

Why solving stiffness is necessary but not sufficient

- Normalizing vascular elasticity would likely lower late-life cardiovascular and even cancer mortality (stiffness correlates with cancer deaths as well) and is therefore a necessary component of any strategy to push human lifespan much beyond current limits.[80][73][74]

- However, even perfect arteries would not prevent deaths from cancer, neurodegeneration, or immune collapse, so meaningful increases in maximum lifespan require combining vascular-stiffness solutions (including elastin/ECM repair) with interventions targeting multiple other aging mechanisms.[74][79]

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