



Widespread Oxytocin Receptor Expression Suggests a Broader Role in Longevity Biology

Oxytocin receptor (OXTR) is expressed across brain, vascular, immune, musculoskeletal, and metabolic tissues, and converges on several core longevity pathways (inflammaging, vascular integrity, neuroprotection, and AMPK–mTOR–autophagy), which strongly suggests it is a systems-level node in healthspan biology rather than “just” a social bonding receptor.^{[1] [2] [3] [4] [5]}

Where OXTR is expressed

OXTR is widely expressed in the CNS (hypothalamus, limbic regions, cerebellar cortex, extended amygdala) and many peripheral tissues.^{[6] [5] [1]}

In humans, OXTR expression increases in late adulthood in cerebellar cortex and tracks with genes controlling learning, bone regeneration, and energy homeostasis, consistent with a compensatory, allostatic role during aging.^[1]

Mechanistic links to aging pathways

- Oxytocin signaling activates AMPK, inhibits mTOR, and promotes autophagy, which can reduce cellular senescence and preserve proteostasis, paralleling rapamycin/metformin-type effects.^[7]
- OXTR on microglia and astrocytes suppresses NF-κB and pro-inflammatory cytokines (IL-6, TNF-α), dampening inflammaging and neuroinflammation that drive neurodegeneration and cognitive decline.^{[3] [4]}
- Vascular OXTR modulates endothelial function and cerebral blood flow, making it relevant to stroke risk and vascular aging.^[5]

Experimental longevity data

- In old, frail male mice, a combined oxytocin + ALK5 (TGF-β) inhibitor protocol extended median lifespan from birth by ~14%, and remaining lifespan from treatment start by ~74%, with markedly lower hazard of death; females showed no lifespan benefit, underscoring strong sex-specificity.^{[8] [9]}
- Oxytocin has shown anti-aging effects on telomere maintenance under social isolation stress, suggesting a link between social environment, oxytocin signaling, and cellular aging.^[2]

CNS aging, cognition, and behavior

- Lifespan-wide analyses indicate dynamic OXTR expression, with late-life increases possibly buffering age-related cognitive circuit deterioration and supporting allostasis.^{[3] [1]}
- Oxytocinergic activation reduces microglial priming and central inflammatory tone, and an OT + ALK5 inhibitor combo halved CD68+ microglia in aged mouse brains, consistent with a neuroprotective, pro-healthspan role.^{[4] [3]}

Conceptual implications for longevity biology

- Natural variation in OXTR signaling alters transcriptional programs across multiple brain regions, including immune-related C-type lectin genes, implying that OXTR is a regulatory hub rather than a narrow “social” switch.^[10]
- Given its reach into autophagy, inflammaging, vascular function, bone and muscle maintenance, and cognitive resilience, OXTR looks like an upstream integrator of socioenvironmental signals into the classic hallmarks of aging, making it a plausible multi-target lever for healthspan extension rather than a single-axis intervention.^{[2] [7] [4] [5] [1] [3]}

Are you mainly interested in mechanistic mapping of OXTR onto specific hallmarks of aging (e.g., a pathways table), or in evaluating translational potential and risk profile of oxytocin/OXTR-targeted interventions in humans?

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Oxytocin declines with age. So do sleep quality, bone density, stress resilience, and appetite regulation.

That overlap raises a different question: What if these changes are not independent—but coordinated?

Oxytocin has traditionally been framed as a social hormone, associated with bonding and emotional connection. But that definition doesn't explain something more fundamental: its receptors are expressed across the brain, cardiovascular system, bone, adipose tissue, and gastrointestinal tract.

When a single signaling molecule is present across this many systems, it is not operating as a niche hormone. It is acting as a coordinating signal—one that helps regulate how the body adapts to stress, recovers during sleep, maintains structural integrity, and manages energy balance.

That shifts how its decline should be interpreted.

The reduction in oxytocin signaling across midlife doesn't produce one clear dysfunction. It produces many—distributed across the same systems where its receptors are expressed.

Changes we often group together under “aging” may, in part, reflect the loss of a shared regulatory signal.

So the question becomes: What changes when that signal fades?

In this week’s Research Review, Dr. Richard LaFountain examines what oxytocin’s receptor distribution reveals about its broader role in coordinating physiology—and what the gradual loss of that signal may mean across multiple systems at once.

The question is no longer whether oxytocin matters. It is how much of aging it helps orchestrate.

Oxytocin’s receptor distribution makes it much more plausible that age-related “declines” in many domains are partially coordinated by a shared, gradually failing regulatory signal rather than a collection of unrelated failures.^{[16] [17] [18] [19] [20]}

Does oxytocin actually decline with age?

Evidence is mixed but converging on an age-linked erosion of oxytocin signaling capacity rather than a simple linear drop in one hormone level.^{[21] [22] [16]}

Work summarized by Maejima et al. shows progressive DNA hypermethylation of oxytocin neurons with age, creating a self-reinforcing decline that is reversible with intranasal oxytocin in animal models.^[21]

Human studies report complex, sex-specific patterns in circulating oxytocin (e.g., low in early adult males but higher in old age), but these do not rule out local receptor and signaling declines at the tissue level.^{[22] [23] [16]}

Region-specific reductions in Oxtr mRNA (e.g., ventromedial hypothalamus in aged female rats) support the idea that the system “thins out” unevenly across the brain, altering how social, stress, and metabolic inputs are integrated.^{[24] [25]}

How broad is OXTR expression?

OXTR is expressed in key CNS hubs (hypothalamus, limbic structures, hippocampus, brainstem nuclei) that regulate stress, arousal, appetite, and socioemotional behavior.^{[18] [26] [20]}

In rodents, detailed mapping shows dense OT fibers and OXTR-positive cell bodies in hippocampus, pons, substantia nigra, amygdala, and hypothalamus, with interesting mismatches (e.g., OT fibers in cortex, OXTR in hippocampus), consistent with a neuromodulatory broadcast role.^[18]

Peripheral expression includes uterus, mammary gland, endometrium, ovary, testis, epididymis, vas deferens, kidney, heart, and vasculature, illustrating that OXTR participates in cardiovascular regulation, reproduction, and fluid balance, not just parturition and lactation.^{[17] [19] [20]}

A coordinating signal for multiple aging phenotypes

Because OXTR is present across neural, cardiovascular, bone, and metabolic tissues, oxytocin is structurally positioned to act as a **coordinating** signal connecting social context and internal homeostasis.^{[19] [20] [16] [17] [18]}

Across the lifespan, oxytocin signaling modulates stress reactivity (HPA axis tone), sleep-related arousal and autonomic balance, feeding behavior and energy expenditure, bone remodeling through brain–bone circuitry, and cardiovascular function; all of these domains show characteristic mid- to late-life drifts.^{[25] [27] [20] [16] [22]}

If oxytocin tone and/or receptor availability erode, the downstream manifestation would be a set of modest, distributed deficits—poorer sleep, reduced stress resilience, slight shifts in appetite and body composition, slower bone turnover—exactly the pattern we lump together as “getting older” rather than a single catastrophic failure.^{[27] [16] [22] [21]}

Illustrative mapping of domains

Domain	OXTR site(s) most relevant	Aging-linked change plausibly influenced by loss of signal
Sleep/arousal	Hypothalamus, brainstem, limbic circuits	Fragmented sleep, blunted parasympathetic recovery ^{[16] [18]}
Bone and structure	Hypothalamus–bone axis, autonomic innervation	Declining bone density and slower remodeling ^[27]
Stress resilience	Hypothalamus, amygdala, hippocampus	Higher baseline cortisol, exaggerated stress responses ^{[16] [25]}
Appetite/energy	Hypothalamus, gut, adipose-linked circuits	Altered satiety, sarcopenic obesity, insulin resistance ^{[16] [28] [20]}
Cardiovascular	Heart, vasculature, renal tissues	Endothelial dysfunction, higher blood pressure variability ^{[29] [19] [20]}

What changes when the signal fades?

Conceptually, a fading oxytocin signal would reduce the coupling between social/sensory context and internal buffering mechanisms (anti-inflammatory tone, parasympathetic dominance during rest, anabolic support to bone and muscle, efficient nutrient partitioning).^{[28] [16] [27] [21]}

Epigenetic studies suggest that declining oxytocin is entangled with mitochondrial dysfunction and systemic inflammation, implying that the loss of this coordinating signal feeds into hallmarks of aging such as altered intercellular communication, epigenetic drift, and deregulated nutrient sensing.^{[28] [21]}

The result is not a discrete disease state but a broad loss of “physiological coherence”: individual systems can still function, but they no longer adapt to each other as smoothly across daily cycles of stress, feeding, and sleep.^{[16] [22] [21]}

How strong is the “orchestrator of aging” hypothesis?

Current data are consistent with oxytocin as one of several higher-order integrators that influence aging trajectories, rather than a single master switch. [16] [21] [28]

We now have: (1) age-linked changes in oxytocin signaling (including epigenetic down-regulation), (2) wide OXTR distribution across aging-sensitive systems, and (3) initial evidence that restoring signaling can reverse epigenetic and inflammatory signatures in animals, collectively supporting the idea that part of “aging” is the loss of this shared regulatory layer. [21] [18] [28] [16]

The open questions are how much variance in human aging oxytocin actually explains compared with other axes (e.g., GH/IGF-1, sex steroids, sympathetic tone) and whether long-term modulation can shift hard outcomes like morbidity and mortality rather than just intermediate phenotypes. [22] [16] [21]

Would you like to take this into a more explicit hallmarks-of-aging framework (e.g., a table mapping oxytocin/OXTR to each hallmark) or focus on evaluating whether and how to ethically modulate oxytocin signaling in humans?



mapping of OXTR onto specific hallmarks of aging (e.g., a pathways table)

Oxytocin receptor (OXTR) signaling touches almost every major hallmark of aging; below is a concise mapping with key pathways and example findings. [32] [33] [34] [35] [36]

Hallmarks–OXTR pathways table

Hallmark of aging	OXTR-linked mechanisms	Representative evidence
Genomic instability & telomere attrition	Indirect via reduced oxidative stress and inflammation, protection from apoptosis, improved mitochondrial function lowers DNA damage burden. [34] [37] [36]	Oxytocin reduces LPS-induced ROS and inflammatory mediators in microglia/macrophages and limits NF-κB/NLRP3 activation, preserving neuronal viability. [34] [36]
Epigenetic alterations	OXT decline associates with reduced TET activity and 5hmC, increased DNA methylation; exogenous OXT reverses stress- and age-induced methylation changes at OXTR and other loci. [32] [37] [38]	Aging Cell work: age-related OXT decline → lower TET enzymes, aberrant methylation, mitochondrial dysfunction; 10-day intranasal OXT restores TET function and epigenetic marks in mice. [32] [37] [38]
Loss of proteostasis	AMPK activation and mTOR inhibition promote autophagy and clearance of damaged proteins/organelles. [39]	OXT activates AMPK, inhibits mTOR, enhances autophagic flux, theoretically aligning with rapamycin/metformin-like proteostatic benefits. [39]

Hallmark of aging	OXTR-linked mechanisms	Representative evidence
Mitochondrial dysfunction	OTR–mitochondria coupling maintains fusion–fission dynamics, mtDNA content, and limits neuroinflammation-induced mitochondrial injury. ^{[34] [36]}	OXT via OTR preserves mitochondrial homeostasis in hypoxic brain injury models, reducing neuronal apoptosis and microglial activation; effects are blocked by OTR antagonism. ^[36]
Deregulated nutrient sensing	OXT influences AMPK–mTOR axis, insulin sensitivity, energy expenditure, and feeding behavior via hypothalamic and peripheral OXTR. ^{[39] [40] [41]}	Reviews describe OXT as a modulator of appetite and energy balance; AMPK–mTOR modulation by OXT positions it squarely in nutrient-sensing circuitry. ^{[39] [40] [41]}
Cellular senescence	AMPK–mTOR–autophagy axis downstream of OXTR reduces senescent cell accumulation; anti-inflammatory actions blunt SASP signalling. ^{[35] [39] [34]}	Oxytocin’s AMPK activation reduces mTOR-driven senescence, and its suppression of IL-6, TNF- α , and iNOS in microglia/macrophages counteracts pro-senescent inflammatory milieu. ^{[35] [39] [34]}
Stem cell exhaustion	Direct support of muscle stem (satellite) cells; declined OT/OTR in aged muscle stem cells impairs regeneration and promotes fibrosis, reversible with OT. ^[33]	Elabd et al.: age-related drop in circulating OT and muscle OTR; OT is required for muscle regeneration, and short-term OT restores myogenesis in old mice while antagonism induces premature sarcopenia-like changes. ^[33]
Altered intercellular communication	OXTR broadly expressed in brain, immune, vascular, and metabolic tissues; OXT modulates cytokine release, neuroimmune crosstalk, and neurovascular coupling. ^{[35] [42] [43] [34]}	OXT dampens microglial and macrophage inflammatory responses to LPS, alters NF- κ B signalling, and changes glial OTR expression; reviews frame OXT as an anti-inflammaging signal. ^{[35] [42] [34]}
Inflammaging (low-grade chronic inflammation)	Strong anti-inflammatory hormone: suppresses IL-6, TNF- α , COX-2, iNOS, NF- κ B, and NLRP3 inflammasome activity in brain and periphery. ^{[35] [44] [34] [36]}	Microglial and macrophage studies show OXT pretreatment blocks LPS-induced inflammatory gene expression; intranasal OXT reduces brain inflammation in LPS-challenged mice. ^{[35] [34]}
Neurodegeneration / brain aging (downstream of multiple hallmarks)	OXTR in hippocampus, amygdala, and salience networks modulates neuroinflammation, mitochondrial integrity, and functional connectivity. ^{[44] [43] [45] [46]}	Intranasal OXT modulates salience-network connectivity in older adults; higher OXT associates with structural variation in hippocampus/amygdala, and OXT reduces neuroinflammatory markers relevant to cognitive aging. ^{[44] [45] [46]}

Conceptual takeaways

- OXTR is positioned at a convergence of epigenetic regulation, mitochondrial health, inflammasome activity, autophagy, and stem-cell maintenance, rather than acting in a narrow behavioural niche.^{[33] [34] [35] [36] [32]}
- The most robust links so far are to inflammaging, stem cell exhaustion in muscle, mitochondrial dysfunction, and epigenetic aging; evidence for direct effects on genomic instability and global proteostasis is promising but still more inferential.^{[34] [37] [35] [36] [32] [33]}

Would it be most useful if I now build a parallel table that overlays specific tissues (brain, muscle, bone, vasculature, immune) on these same hallmarks with OXTR-driven mechanisms?

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Yes map hallmarks by tissue systems

OXTR maps onto the hallmarks in a tissue-specific way, with especially strong roles in brain, muscle, bone, vasculature/heart, and immune–metabolic interfaces. [\[51\]](#) [\[52\]](#) [\[53\]](#) [\[54\]](#) [\[55\]](#)

Brain / CNS

Hallmark cluster	OXTR-linked CNS mechanisms	Key evidence
Mitochondrial dysfunction & neurodegeneration	OXTR on neurons and microglia maintains mitochondrial dynamics, reduces apoptosis, and limits neuroinflammation. [56] [57]	OT via OTR preserves mitochondrial homeostasis and reduces neuronal death in hypoxic brain injury; OTR antagonism reverses protection. [56] Intranasal OT attenuates microglial activation and corrects memory deficits in APP/PS1 AD mice. [57]
Inflammaging & altered intercellular communication	OXTR+ microglia/astrocytes show suppressed NF-κB/NLRP3 and reduced IL-6, TNF-α, COX-2, iNOS after OT, lowering chronic neuroinflammatory tone. [55] [58] [56] [57]	OT pretreatment blunts LPS-induced microglial activation and cytokine production; intranasal OT reduces plaque-associated microglial activation in AD models. [55] [58] [57]
Epigenetic aging	OT modulates stress-linked epigenetic drift (including OXTR methylation) in social/affective circuits, linking social environment to brain epigenetic age. [59] [60] [61]	Human data show age- and stress-dependent OXTR methylation patterns in brain-related regions; experimental OT can reverse some stress-induced epigenetic changes. [59] [60] [61]

Skeletal muscle

Hallmark cluster	OXTR-linked muscle mechanisms	Key evidence
Stem cell exhaustion	Muscle satellite cells express OTR; OT is required for normal activation/proliferation and regeneration with age. [51] [62] [63]	Elabd et al.: plasma OT and OTR in muscle stem cells decline sharply with age; short-term systemic OT restores regeneration in old mice, while genetic OT loss causes premature sarcopenia-like phenotype. [51] [62] [63]
Loss of proteostasis & cellular senescence	Improved satellite-cell function and reduced fibrotic, non-regenerative repair indirectly reduce senescent cell accumulation and mis-repaired tissue. [51] [62] [63]	OT rescues proliferative capacity of old satellite cells to near-young levels without hyperproliferation in young, suggesting restoration rather than overdrive. [51] [62] [63]

Hallmark cluster	OXTR-linked muscle mechanisms	Key evidence
Mitochondrial dysfunction	Better regenerative capacity and lower chronic inflammation in muscle support healthier mitochondrial turnover and function, though data here are more inferential. [51] [63]	Reviews on muscle stem cell aging cite OT as one of the few systemic factors that directly rejuvenate aged satellite cells and their metabolic profile. [63]

Bone / skeletal system

Hallmark cluster	OXTR-linked bone mechanisms	Key evidence
Stem cell exhaustion & altered intercellular communication	OXTR is expressed on osteoblasts and osteoclasts; OT promotes osteoblast differentiation and mineralization, while cyclically modulating osteoclastogenesis and resorption. [52] [54]	OT stimulates osteoblast formation and matrix mineralization; in ovariectomized mice, OT reduces osteoclast number and increases bone density. [52] [54]
Endocrine/inflammaging interface	OT is now recognized as a hypothalamic determinant of bone remodeling, interacting with estrogen and leptin signals; low OT links to low BMD in post-menopausal women. [52] [54] [64]	Clinical data: higher OT associates with higher hip BMD, especially in women with low estradiol or high leptin; OT and OTR are positively regulated by estrogen in bone. [54] [64]

Heart and vasculature

Hallmark cluster	OXTR-linked cardiovascular mechanisms	Key evidence
Mitochondrial dysfunction & genomic/oxidative stress	Cardiac OXTR activation improves mitochondrial protection, reduces oxidative stress, and limits cardiomyocyte hypertrophy. [53] [65]	OT increases glucose uptake, reduces oxidative stress, and protects cardiac mitochondria; blockade of cardiac OXTR worsens outcomes after MI. [53] [65]
Stem cell exhaustion & regeneration	OXTR on epicardial progenitor cells (EpiPCs) is required for their activation and heart regeneration. [66] [53]	OT robustly activates EpiPCs in vitro and in vivo, enhancing cardiomyocyte and vascular cell production; OTR expression rises 20–30× in activated EpiPCs. [66]
Inflammaging & altered intercellular communication	OXTR in heart and vasculature regulates cytokine expression, immune cell infiltration, vasodilation, and blood pressure. [53] [65]	OT reduces pro-inflammatory cytokines and immune infiltration in cardiac injury, while promoting angiogenesis and improving endothelial function. [53] [65]

Immune–metabolic axis (adipose, liver, systemic inflammation)

Hallmark cluster	OXTR-linked immune/metabolic mechanisms	Key evidence
Inflammaging & altered intercellular communication	OXTR-expressing macrophages and other immune cells reduce NF-κB and cytokine output when stimulated by OT, dampening chronic low-grade inflammation. [55] [58] [67]	Studies show OT lowers IL-6, TNF-α, and other inflammatory mediators in immune cells, framing OT as an anti-inflammatory hormone in systemic circulation. [55] [58] [67]

Hallmark cluster	OXTR-linked immune/metabolic mechanisms	Key evidence
Deregulated nutrient sensing & metabolic aging	OXTR in hypothalamus, adipose-linked circuits, and peripheral tissues modulates appetite, insulin sensitivity, energy expenditure, and body composition. [68] [69] [70]	Reviews and animal work: OT reduces food intake in some contexts, shifts substrate utilization, and improves metabolic parameters linked to insulin resistance and obesity. [68] [69] [70]
Epigenetic aging	Social/psychological stress alters OXTR methylation and OT system activity, feeding into epigenetic age acceleration; restoring OT can normalize some stress-induced methylation patterns. [59] [71] [61]	Human epigenetic studies link chronic stress and low OT signaling to accelerated DNAm aging; OT-centered interventions in animals reverse epigenetic drift at stress-sensitive loci. [59] [71] [61]

High-level pattern

Across tissues, OXTR sits at nodal points where:

- **Stem-cell maintenance**, mitochondrial resilience, and local inflammation intersect (muscle satellite cells, osteoblasts, epicardial progenitors, microglia). [\[62\]](#) [\[66\]](#) [\[52\]](#) [\[57\]](#) [\[63\]](#) [\[51\]](#)
- Systemic signals (social environment, stress, reproductive hormones, nutritional state) are transduced into changes in regeneration, remodeling, and inflammatory tone, i.e., classic “altered intercellular communication” and “inflammaging”. [\[52\]](#) [\[53\]](#) [\[54\]](#) [\[65\]](#) [\[55\]](#) [\[59\]](#)

For your purposes, would a next step be a visual schema (even just in text) connecting “social stress → OT/OXTR → specific hallmarks in each tissue,” or do you want to move straight into how one might safely probe or modulate these axes in humans?



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