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Autophagy: The Principal Regulator of Vascular Function and Homeostasis

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ABSTRACT

Background: Vascular endothelium is more than just a selective barrier asit acts like a vast organ that functions continuously to regulate vascular tone, permeability, thromboregulation, leukocyte trafficking and angiogenesis. So, it is unsurprising that an early common abnormalityin conditions such asatherosclerosis, hypertension and diabetic vasculopathy is the loss of endothelial homeostasis. The lysosome-dependent recycling and quality-control mechanism known as autophagy has emerged as a crucial component in preserving endothelial stability.This review describes how autophagy is regulated in endothelial cells, summarizes its key physiological roles, and clarifies its context-dependent, reciprocal impacts on both health and disease. Our goal is torelatemolecular mechanisms with vascular functionand potential treatment options.

Methodology: We used the following MeSH terms and keywords to search PubMed, Scopus, and Web of Science for Englishpublications up until October 2023: "Autophagy," "Endothelial cells," "Endothelium," "Mitophagy," "Vascular homeostasis," "Endothelial dysfunction," "Atherosclerosis," "Hypertension," "Diabetes," "Ischemia–reperfusion," "Nitric oxide," "Shear stress," "mTOR" and "AMPK." Then, we manually went through the citation lists of significant primary research and reviews. To ensure that the information was current and accurate, emphasis was placed on recent high-impact studies, systematic reviews and meta-analyses.

Conclusion: Basal autophagy promotes redox balance, endothelial barrier integrity, Nitric Oxide (NO) signaling, mitochondrial quality control, and blood flow adaptation. Major vascular pathologies are significantly impacted by dysregulated (Excessive or insufficient) autophagic flux. As the effects of autophagy vary greatly depending on the circumstance and stage, the therapeutic modulation must be highly precise, with an emphasis on the endothelium, appropriate timing and solid clinical monitoring. Although selective pharmacologic approaches and lifestyle strategies (Exercise, diet restriction etc.) show promise, specificity and suitable flux biomarkers remain crucialunmet needs.

KEY WORDS

Atherosclerosis; AMPK; Endothelial autophagy; Hypertension; Mitophagy; mTOR; Nitric oxide; Oxidative stress; Therapeutic targeting; Vascular disease.

INTRODUCTION

While previously considered a passive 'Teflon-like' lining, the vascular endothelium is now considered a large and highly metabolically active organ. The single layer of endothelial cells provides an important interface between the circulation and all tissues of the body. The endothelial cell is not a passive cell; it is a sophisticated sensor that is continuously integrating a complex set of biochemical and hemodynamic inputs. Signaling mechanisms coordinate physiological mechanisms which are central to cardiovascular homeostasis such as regulation of vascular tone, leucocyte trafficking, angiogenesis (New vessel growth) and coagulation.[1]

Consequently, endothelial dysfunction is now recognized as a trigger event in the pathogenesis of most of the major disorders of the cardiovascular field. Impaired endothelium is present in a wide range of diseases ranging from hypertension to coronary artery disease to diabetic vasculopathy and the process begins early in disease (Years before a clinical manifestation is displayed).[2] This dysfunction is the phenotypic modification of endothelium from a state of quiescence and anti-inflammatory to activated and pro-inflammatory

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activity. Major processes involved in this pathological switch include a reduced bioavailability of a vasodilator, Nitricoxide (NO) the overproduction of Reactive Oxygen Species (ROS) and the up-regulation of the adhesion molecules that recruit inflammatory cells.[3]

The cellular quality control process of autophagy has recently been placed in a pivotal role for endothelial health. Once regarded as a primitive survival process in response to periods of starvation, it is now known to be a highly selective process for elimination of dysfunctional proteins, damaged organelles, and toxic biomolecules.[4,5] Endothelial resilience under varying shear stress, hypoxia-reoxygenation, and metabolic stressors like hyperglycemia depends on this housekeeping function.[6] Basal autophagy promotes the function of endothelial NO Synthase (eNOS) inhibits oxidative damage through mitophagy and aids in intercellular communication and barrier maintenance.[7,8] However, the pathway is not always protective: while prolonged or excessive activation can cause cell loss and tissue damage, insufficient flux encourages the buildup of cellular debris and senescence.[9] Comprehending this equilibrium is essential for logical therapeutic targeting.

SEARCH STRATEGY

To find studies published up until October 2023, we searched Web of Science, PubMed and Scopus. MeSH terms and keywords, such as "Autophagy," "Endothelial cells," "Endothelium," "Mitophagy," "Vascular homeostasis," "Endothelial dysfunction," "Atherosclerosis," "Hypertension," "Diabetes," "Ischemia–reperfusion," "Nitric oxide," "Shear stress," "mTOR" and "AMPK," captured endothelial autophagy from mechanism to clinical implications. Publications in English were the only ones included. The reference lists of significant reviews and seminal primary studies were manually checked. Evidence analyses and recent, high-impact work were emphasized.

DISCUSSION

The Core Machinery and Regulation of Endothelial Autophagy

The multi-step, conserved process of autophagy transports cytoplasmic material to lysosomes for recycling and degradation. Shear stress, endoplasmic reticulum stress, oxidative load, nutritional status, and other metabolic cues all affect endothelial autophagy.[4,6] The four stages of autophagy are carried out by a coordinated group of autophagy-related (Atg) proteins.

i) Initiation and nucleation: The gatekeeper is the Unc-51-like kinase 1 (ULK1) complex. ULK1 is phosphorylated and suppressed by mTORC1 when nutrients are sufficient. When AMP-activated protein kinase (AMPK) is activated by energy stress, it phosphorylates and activates ULK1 while opposing mTORC1, turning on autophagy.[10] In order to scaffold downstream factors, activated ULK1 recruits the class III PI3K complex (Beclin1/Vps34) which produces phosphatidylinositol-3-phosphate (PI3P) at the isolation membrane (Phagophore).[5]

ii) Elongation and closure: The phagophore is expanded by two ubiquitin-like conjugation systems. The Atg12–Atg5–Atg16L1 complex enhances both autophagosome leaflets by facilitating LC3 lipidation (LC3-I -> LC3-II) with phosphatidylethanolamine. One of the functional markers of autophagosome biogenesis is LC3-II [4,11].

iii) Selective cargo capture and fusion: Adaptor proteins like p62/SQSTM1 that connect ubiquitinated cargo to LC3 provide selectivity preventing bulk cytoplasm from being ingested. Mitophagy receptors (BNIP3, NIX/BNIP3L, FUNDC1) and the PINK1/Parkin pathway, which mark weakened organelles for elimination, are responsible for clearing mitochondria.[8] Mature autophagosomes merge with lysosomes to form autolysosomes after moving along microtubules.

iv) Degradation, recycling and flux: Membranes and cargo are broken down by lysosomal hydrolases into nucleosides, fatty acids and amino acids that can re-enter energetic and biosynthetic pathways.[5] Most importantly, true activity of autophagy is defined by autophagic flux, which is the throughput from initiation to lysosomal degradation; a mere increase in the number of autophagosomes may indicate either induction or a distal block.[11]

Physiological Roles of Autophagy in the Endothelium

A precise and baseline autophagy is required for healthy endothelial structure and function and for quick adaptation to stress (Table I).

i) Control of vascular tone and nitric oxide

The NO produced by eNOS is crucial for vasodilation, inhibition of platelet adhesion and control of inflammation.[3] By removing ROS-producing mitochondria that would otherwise quench NO, autophagy preserves NO signalling.[7,8] It also encourages caveolin-1 turnover (Relieving eNOS inhibition) and places eNOS within suitable membrane domains for stimulus-coupled activation.[12]

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ii) Responses to mechanical forces

Endothelial phenotype is influenced by shear stress. While oscillatory/turbulent flow at curvatures and bifurcations is atheroprone, unidirectional laminar flow is atheroprotective.[1] Laminarflow increases Krüppel-like factor 2 (KLF2) and activates AMPK, which improves autophagic flux and anti-inflammatory signaling.[6,13] Whereas autophagy is compromised in areas subjected to disrupted flow, which leads to oxidative stress and the expression of adhesion molecules that cause lesions.[6]

iii) Mitochondrial quality control via mitophagy

Damaged mitochondria weaken NO signaling and cause inflammation by leaking electrons and increasing ROS. Through PINK1/Parkin and receptor-mediated pathways, endothelial mitophagy eliminates these dysfunctional organelles, thereby reducing NLRP3-inflammasome activation and senescence. A pro-oxidant, pro-inflammatory state is maintained when mitophagy fails, leading to overt dysfunction.[14]

iv) Barrier integrity and angiogenesis

Vascular permeability is controlled by junctional complexes, such as VE-cadherin. In order to preserve barrier integrity, autophagy replaces oxidized or misfolded junctional proteins. Faulty autophagy destabilizes junctions, promotes leakage, and makes leukocyte infiltration easier.[6] Migrating and proliferating endothelial cells need enough energy and substrates during angiogenesis. Integrated autophagy/mitophagy reduces ROS linked to this high metabolic flux ensuring proper energy, substrates and environment for orderly sprouting.[15]

Table I Autophagy in endothelial physiology and disease: comparative view

Domain	Physiological role (Protective)	Pathological shift when dysregulated
NO signaling and vascular tone	Preserves eNOS function, removes ROS sources to maintain NO bioavailability[3,7,8,12]	ROS accumulation quenches NO, caveolin 1–mediated eNOS inhibition, impaired vasodilation
Hemodynamic adaptation	Laminar shear ⇝ AMPK/KLF2 ⇝ enhanced autophagic flux, anti-inflammatory phenotype [6,13]	Disturbed flow ⇝ reduced flux, oxidative/inflammatory signaling at atheroprone sites
Mitochondrial quality (Mitophagy)	PINK1/Parkin and BNIP3/NIX pathways clear depolarized mitochondria, restrain NLRP3 and senescence [8,14]	Defective mitophagy ⇝ mitochondrial ROS, inflammasome activation, endothelial dysfunction
Barrier integrity	Autophagic renewal of junctional proteins (e.g. VE cadherin) controlled permeability[6]	Junctional instability, leakage, leukocyte infiltration
Angiogenesis	Supplies energy and substrates during migration/proliferation, limits oxidative burden[15]	Metabolic/oxidative stress impairs sprouting and vessel quality
Atherosclerosis	Early protection against lipid deposition, apoptosis, inflammation[1,9,16]	Advanced lesion: stalled or maladaptive autophagy ⇝ necrotic core and instability[17]
Hypertension	Adequate flux preserves NO, limits stiffness	Impaired flux ⇝ ROS↑, NO↓, pro inflammatory signaling, raised resistance [18,19]
Diabetes	Early adaptive clearance of glycated/damaged cargo[19]	Chronic glucolipotoxicity ⇝ fusion block, autophagosome build up, vascular complications[19,20]
Ischemia–reperfusion	Ischemic phase: Survival support via recycling	Reperfusion: Excessive/autonomous activation may promote cell loss[21,22]
Aging	Maintains proteostasis and organelle quality[23]	Declining autophagy ⇝ SASP, inflammaging, stiffness, atherogenesis[23,24]

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The Dual Role of Autophagy in Vascular Pathologies

Despite the protective effect of basal autophagy, disease contexts show a complex, stage-dependent dualism (Table II).

i) Atherosclerosis

Endothelial autophagy inhibits lipid deposition and monocyte adhesion in early atherogenesis by lowering oxidative stress, limiting apoptosis, and maintaining barrier integrity.[1,9,16] Plaque formation in vivo is accelerated by endothelium-specific deletion of Atg genes.[16] However, hypoxia and persistent inflammation in advanced plaques can either inhibit autophagic flux or on the other hand, cause maladaptive overactivation in macrophages and smooth muscle cells, which encourages necrotic-core instability and expansion.[17]

ii) Hypertension

Vascular stiffening and endothelial dysfunction are key components of hypertension. Peripheral resistance is increased when autophagic activity is reduced because that increases ROS, decreases NO bioavailability and encourages pro-inflammatory signaling.[18,19] In preclinical models, endothelium-dependent dilatation is improved by restoring flux using AMPK-linked strategies (e.g. metformin, resveratrol, spermidine) although tissue specificity is still a significant issue.[18]

iii) Diabetic vasculopathy

Insulin resistance and hyperglycemia cause endothelium to experience glucolipotoxic stress. Glycated proteins and damagedorganelles are eliminated by an initial adaptive increase in autophagy, however, autophagosome–lysosome fusion is impaired by chronic metabolic stress, leading to autophagosome accumulation without degradation. In diabetic retinopathy, nephropathy and peripheral arterial disease, the ensuing ROS and inflammatory signaling hasten endothelial damage.[19,20]

iv) Ischemia–reperfusion injury

Autophagy aids in survival during ischemia by removing damage and recycling substrates. However, abrupt ROS surges and ongoing autophagy during reperfusion may cause cells to lose their function, exacerbating tissue damage.[21,22] I/R outcomes are largely determined by the timing and strength of autophagic responses.

v) Vascular aging and senescence

Autophagy efficiency is known to diminish with age, which is one of the key factors of vascular aging.[23] This inability to get rid of cellular debris leads to the endothelial cells to be in the state of senescence. These senescent cells stop dividing and secrete a cocktail of pro-inflammatory molecules that are involved in the chronic, low-grade inflammation of ageing (Aged-related inflammations or inflammaging). This process directly causes an increase inarterial stiffness/age-related vascular diseases.[23,24]

Table II Therapeutic modulation of endothelial autophagy: Opportunities and cautions

Class/agent	Primary target/mechanism	Expected endothelial effect	Key cautions
Rapamycin, everolimus, sirolimus	mTORC1 inhibition ↠ autophagy induction[10]	Reduce oxidative stress, support NO signaling, Atheroprotection (Preclinical)	Systemic effects, dosing/timing critical
Metformin	AMPK activation ↠ increased flux[21]	Improved endothelial function, metabolic benefit	Off target metabolic effects, context dependence
Resveratrol	AMPK activation, sirtuin/ acetyltransferase modulation[24]	Enhanced vasodilation, anti inflammatory profile	Bioavailability, heterogeneous responses
Spermidine	Acetyltransferase inhibition, pro autophagic signaling [24,25]	Vascular protection, longevity markers	Dosing, long term safety in humans
Chloroquine, hydroxychloroquine	Lysosomotropic, block autophagosome–lysosome fusion[9]	Potential benefit when excessive autophagy is harmful (e.g. reperfusion phase)	Narrow window, systemic adverse effects
Lifestyle: Caloric restriction, intermittent fasting	Down• regulate nutrient signaling, activate AMPK[25]	Induce protective autophagy, cardiometabolic gains	Adherence, suitability by comorbidity
Lifestyle: Aerobic exercise	Laminar shear ↠ AMPK/KLF2 ↠ autophagy[6,13]	↑ NO bioavailability, ↓ inflammation	Program design, patient capacity

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Modulation of Endothelial Autophagy for Therapy

The dual nature of autophagy among all these diseases means that it should be approached with caution at all times, although the link between autophagy and vascular disease makes it a very attractive target of innovation.

i) Pharmacological agents

One of the major classes of inducers of autophagy is that of the mTORC1 pathway, and one of the archetypal inducers of the pathway is rapamycin. Another strategy is to turn on AMPKwhich is done by a common diabetes drug known as metformin. Also, the interest has been extended to natural compounds like spermidineand resveratrol, that induce autophagy and have shown vascular protective effects in animal models.[24,25] In situationswhere autophagy is thought to be too destructive, it can be blocked using an autophagy inhibitor like chloroquine. These drugs block the final step of autophagy, but their use is complicated by the potential toxicity and lack of specificity.[9]

ii) Lifestyle interventions

Intermittent fasting and calorie restriction activate AMPK and down-regulate nutrient signaling, which promotes protective autophagy with positive cardiometabolic effects.[25] Through the AMPK–KLF2 pathways, aerobic exercise produces prolonged laminar shear stress, which stimulates endothelial autophagy, increasing NO bioavailability and reducing inflammation.[6,13]

iii) Challenges and future trends

The following are major obstacles: (a) benefit versus harm from autophagy depends on context and stage (b) there are no endothelium-specific delivery platforms for systemic agents (c) there are no validated, non-invasive biomarkers of autophagic flux in patients. Precision therapy may be made possible by advancements in multi-omics biomarkers, temporal control of pathway modulation, and targeted delivery (Such as nanoparticles).[5,9,24,25]

LIMITATIONS

Bias cannot be ruled out because this is a narrative, mechanism-focused review conducted by a thorough search and is not a formal systematic review. Preclinical models are the source of many findings, and human disease may have different endothelial-specific effects. Direct translation is limited by the lack of clinically validated, non-invasive flux biomarkers.

CLINICAL OUTLOOK

Structured aerobic exercise, optimizing cardiometabolic risk (With metformin when necessary) and evaluating calorie restriction in carefully chosen cohorts are the near-term strategies that are most likely to influence future practice. The creation of blood-based flux biomarkers to direct patient selection and dosage, as well as endothelium-targeted delivery systems for autophagy modulators, are medium-term priorities.

CONCLUSION

One of the fundamental pillars of endothelial homeostasis is autophagy. Basal autophagy prepares the endothelium to endure ongoing mechanical and metabolic stress by maintaining NO signaling, mitochondrial integrity, redox balance and barrier function. Atherosclerosis, hypertension and vascular damage associated with diabetes are all influenced by dysregulated autophagy, either too little or too much. While pharmacological modulation shows promise, it requires endothelium-focused specificity, appropriate timing and dependable clinical monitoring. Lifestyle strategies already offer safe ways to support protective autophagy. Autophagy modulation may develop into useful treatments for the world's most common vascular diseases as targeted delivery and flux assessment advance.

RECOMMENDATIONS

- **Create endothelium-specific treatments:** To reduce systemic effects, design delivery vehicles (such as nanoparticles) that concentrate autophagy modulators in endothelial cells.
- **Create non-invasive biomarkers:** Find and validate imaging or blood-based biomarkers that measure autophagic flux for treatment monitoring, diagnosis, and stratification.
- **Describe the therapeutic windows:** Using thorough preclinical and early-phase trials, ascertain the timing, dosage, and direction (Induction vs. inhibition) of autophagy modulation by disease type and stage.
- **Test combination strategies:** Evaluate how well low-dose pharmaceuticals and lifestyle changes (Like organized exercise) work together to optimize benefits while lowering risks.
- **Prioritize high-quality clinical trials:** Transform promising preclinical findings into meticulously designed human trials to ascertain standards for patient selection, safety, and efficacy.

DISCLOSURE

All of the authors declared no competing interests.

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