SCIENTIFIC EVIDENCE REVIEW

5-Amino-1MQ

NNMT Biology, Compound Chemistry, Preclinical Studies, Evidence Limitations and Research Outlook

A structured, plain-language review of available scientific data

Prepared for Vanguard Compound Research

Evidence search updated 7 September 2026

IMPORTANT RESEARCH CONTEXT

This is a narrative review of laboratory, cellular and animal research. It is not a clinical guideline, medical advice, a treatment recommendation, or evidence of an established human use. No completed controlled human trial of 5-Amino-1MQ was identified in the review search.

Publication Notice and Editorial Standard

This paper was prepared as an educational review for the Vanguard Compound Research website. Its purpose is to organize and explain published information about 5-Amino-1MQ and the nicotinamide N-methyltransferase (NNMT) pathway in a form that is useful to both technically trained readers and non-specialists. The review deliberately separates direct compound evidence from genetic target-validation studies and from work involving other NNMT inhibitors.

It is best described as a structured narrative evidence review, not a systematic review or meta-analysis. Database and registry searches can miss unpublished studies, newly indexed records, conference abstracts, patents, non-English publications, and negative findings that were never reported. Numerical values are retained only where they help readers understand an experiment; they are not converted into personal-use instructions or human-equivalent doses.

REGULATORY STATUS

No Health Canada market authorization for 5-Amino-1MQ as a therapeutic product was identified in this review. Human safety, efficacy, appropriate dosage, product quality standards and clinical use have not been established. The overall context of any webpage matters: educational wording does not neutralize a page that otherwise promotes personal use or sale as a health product.

How to Read This Paper

- Start with the Executive Summary and “5-Amino-1MQ in Simple Terms” for a non-technical explanation.
- Use the chemistry and mechanism sections to understand what NNMT does and why inhibition may change several metabolic pools at once.
- Read the study profiles for design, results and interpretation boundaries rather than relying on a headline outcome.
- Use the evidence limitations, safety gaps and FAQ before drawing conclusions about human relevance.
- Use the website publication package at the end for page title, metadata, headings and structured-data guidance.

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1. Executive Summary

5-Amino-1MQ—also written 5A1MQ, 5-AMQ or 5-amino-1-methylquinolinium—is a synthetic quinolinium-based small molecule studied as an inhibitor of NNMT. It is not a peptide. NNMT catalyzes transfer of a methyl group from S-adenosylmethionine (SAM) to nicotinamide, producing 1-methylnicotinamide (1-MNA) and S-adenosylhomocysteine (SAH). Because nicotinamide is connected to NAD+ salvage and SAM is a major methyl donor, NNMT sits at an intersection of redox, energy and methylation biology.

The compound-specific literature supports several narrow conclusions. First, 5-Amino-1MQ inhibits NNMT in biochemical and cellular systems and can reduce formation of 1-MNA. Second, short-duration mouse studies report changes in body weight, adipose mass, adipocyte size, glucose handling, liver lipid accumulation and selected muscle outcomes. Third, a rat bioanalytical study demonstrated measurable systemic exposure after oral and intravenous administration and reported approximately 38.4% oral bioavailability in that animal model. None of those findings establishes safety or effectiveness in humans.

The strongest evidence is therefore mechanistic and preclinical. The literature is scientifically coherent enough to justify continued investigation, but it is not clinically complete. No established human dose, exposure target, adverse-effect profile, interaction profile, reproductive-risk assessment, long-term toxicology package or controlled human efficacy result was identified. Many prominent studies also arise from a relatively concentrated investigator and commercial-development network, so independent replication is especially important.

BOTTOM LINE

5-Amino-1MQ is a legitimate experimental NNMT inhibitor and research probe. Available evidence does not establish it as a therapy, supplement, weight-loss aid, exercise substitute, muscle-building product, anti-ageing intervention or treatment for metabolic, liver, cardiovascular, kidney or cancer-related disease in humans.

Key Conclusions at a Glance

Field	Research description
Compound class	Synthetic, permanently charged quinolinium small molecule; not a peptide.
Primary target	Nicotinamide N-methyltransferase (NNMT).
Best-supported action	Biochemical and cellular inhibition of NNMT with reduced 1-MNA formation.
Evidence base	Medicinal chemistry, cultured cells, obese and aged mice, and rat pharmacokinetics.
Human evidence	No completed controlled human trial identified in this review.
Main uncertainty	Translation of animal exposure, efficacy and safety to people.
Appropriate description	Preclinical NNMT inhibitor used to investigate metabolism and tissue biology.
Descriptions to avoid	Proven fat burner, NAD+ therapy, metabolic cure, exercise mimetic, muscle builder or clinically validated anti-ageing agent.

2. Review Scope and Method

The central review question was: What does the available published evidence show about 5-Amino-1MQ itself, and what conclusions remain unsupported? Searches emphasized peer-reviewed primary research indexed in PubMed, full-text articles available through PubMed Central or publishers, study records, and authoritative chemical and regulatory sources. The search was updated through 7 September 2026.

Evidence was organized into four groups:

- Direct compound evidence: experiments explicitly identifying 5-Amino-1MQ, 5-amino-1-methylquinolinium, 5-AMQ or 5A1MQ.
- Foundational target evidence: genetic or biochemical work establishing NNMT as a biologically relevant target without administering 5-Amino-1MQ.
- Class-supporting evidence: studies of other NNMT inhibitors that may inform the target but cannot be attributed automatically to 5-Amino-1MQ.
- Contextual evidence: reviews, chemical databases and regulatory guidance used to interpret—not replace—primary experiments.

Inclusion and Exclusion Principles

Priority was given to papers that clearly described the experimental model, comparator, intervention and measured outcomes. Marketing pages, anecdotal reports, personal-use protocols and retailer claims were not treated as evidence. Patents were not treated as proof of safety or efficacy. Review articles were used to understand the broader NNMT field, but claims about 5-Amino-1MQ were anchored to compound-specific primary studies wherever possible.

The paper does not perform a pooled statistical analysis because the studies vary substantially in species, age, disease model, intervention duration, route, exposure and endpoints. A numerical meta-analysis across such heterogeneous designs would create a false sense of precision.

A NOTE ON NEGATIVE EVIDENCE
Absence of an identified human trial is not proof that no person has ever received the compound. It means the review did not identify a completed controlled human study capable of establishing clinical safety or efficacy.

3. 5-Amino-1MQ in Simple Terms

5-Amino-1MQ is a laboratory research molecule designed to reduce the activity of NNMT. Enzymes are proteins that help chemical reactions occur. NNMT takes nicotinamide—a form of vitamin B3—and attaches a methyl group supplied by SAM. The reaction creates 1-MNA and SAH. Researchers study this reaction because the materials used and produced are connected to several systems that cells use to manage energy, redox chemistry and methylation.

A useful analogy is a controlled junction rather than a simple on/off switch. NNMT directs part of the available nicotinamide and methyl-donor supply into a particular product. Reducing the enzyme can change traffic through nearby pathways. But the biological outcome depends on tissue, health state, age, exposure and duration. It is therefore too simplistic to say that blocking NNMT automatically “raises NAD+,” “turns on metabolism,” or “burns fat.” Those phrases skip the experimental conditions and uncertainty that determine what actually happens.

In laboratory and animal studies, investigators have used 5-Amino-1MQ to ask whether lowering NNMT activity changes adipose tissue, glucose regulation, liver lipid accumulation, muscle regeneration, age-associated muscle function and other tissue-specific endpoints. These are research questions. They are not established human benefits.

THE SIMPLEST ACCURATE DESCRIPTION

5-Amino-1MQ is a small-molecule research inhibitor used to study what happens when NNMT activity is reduced. Current published evidence is predominantly preclinical.

4. Compound Identity and Chemistry

The name "5-Amino-1MQ" is shorthand used across research and commercial sources. The compound is commonly represented as 5-amino-1-methylquinolinium iodide. PubChem lists the iodide salt as 5-amino-1-methylquinolin-1-ium iodide with molecular formula C10H11IN2 and molecular weight 286.11 g/mol. The quinolinium nitrogen carries a permanent positive charge, paired with iodide as the counterion.

Field	Research description
Common names	5-Amino-1MQ; 5A1MQ; 5-AMQ; 5-amino-1-methylquinolinium.
Common salt form	5-amino-1-methylquinolinium iodide.
CAS Registry Number	42464-96-0 is commonly associated with the iodide salt; identity should be verified against batch documentation.
Molecular formula	C10H11IN2 for the iodide salt.
Molecular weight	286.11 g/mol for the iodide salt.
Chemical class	Quinolinium small molecule.
Biological class	NNMT inhibitor.
Peptide status	Not a peptide; contains no amino-acid chain or peptide bonds.
Identity caution	Different salt, hydrate, solvate or free-ion descriptions change formula and formula weight. Analytical documentation should name the exact material tested.

Why Salt Form and Analytical Identity Matter

A purity percentage alone does not fully define a research material. HPLC can describe the relative chromatographic profile under a stated method, while mass spectrometry can support molecular-mass assignment. Neither technique, by itself, establishes sterility, endotoxin concentration, residual solvents, elemental impurities, water content, counterion content or suitability for a biological application. A scientifically complete specification should state the exact chemical form, analytical method, reference standard, batch number, acceptance criteria and tests actually performed.

This distinction also affects concentration calculations. A mass of the iodide salt is not chemically identical to the same mass of a hypothetical counterion-free cation or another salt. Researchers should use the exact molecular form reported on the certificate and protocol rather than assuming that every material sold under the shorthand name is interchangeable.

5. NNMT Biology and Proposed Mechanism

NNMT is a cytosolic methyltransferase expressed at different levels across tissues. It transfers a methyl group from SAM to nicotinamide, generating 1-MNA and SAH. The reaction links three important biochemical themes: nicotinamide availability, cellular methyl-donor balance and formation of a bioactive methylated metabolite. Its consequences are therefore context-dependent rather than uniformly beneficial or harmful.

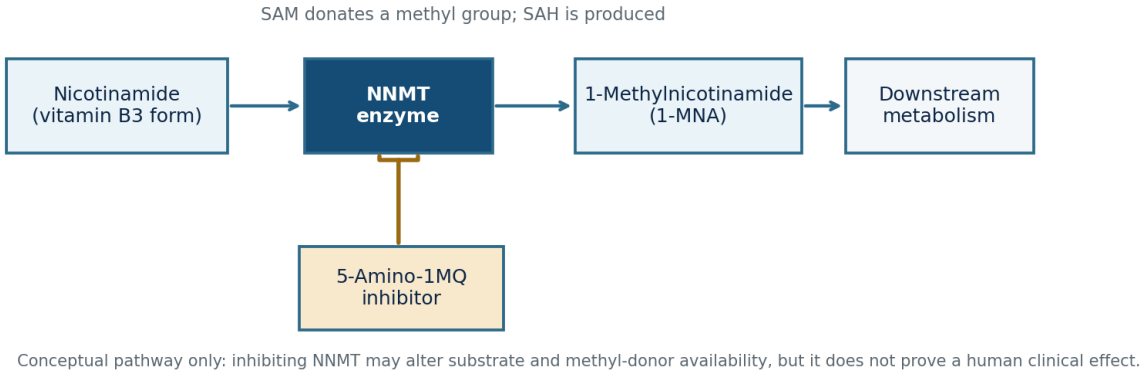


Figure 1. Conceptual NNMT reaction and site of 5-Amino-1MQ inhibition. The figure shows pathway logic, not a clinical outcome.

Nicotinamide and NAD+ Salvage

Nicotinamide can be recycled toward NAD+ through the salvage pathway, beginning with nicotinamide phosphoribosyltransferase (NAMPT). NAD+ is required for redox reactions and also serves as a substrate for enzymes such as sirtuins and PARPs. Diverting nicotinamide through NNMT could influence the amount available to other pathways, but pathway flux is regulated at multiple steps. Measuring one metabolite in one tissue does not prove a whole-body or human “NAD+ boosting” effect.

SAM, SAH and Methylation Potential

SAM donates methyl groups to many reactions, including DNA, RNA, protein, lipid and small-molecule methylation. SAH can inhibit methyltransferases, so the SAM-to-SAH relationship is sometimes used as an indicator of methylation potential. Foundational work has described NNMT as a “methylation sink” in certain settings because high NNMT activity can consume methyl donors and produce a relatively stable methylated metabolite. The magnitude and biological meaning of this effect vary by cell type and disease state.

1-Methylnicotinamide Is Not Merely Waste

Simplified commercial explanations often describe 1-MNA only as an inactive disposal product. That is incomplete. 1-MNA has reported biological actions in vascular, inflammatory and metabolic contexts, and its downstream oxidation products participate in nicotinamide clearance. Reducing 1-MNA could therefore have consequences beyond preserving nicotinamide. A balanced interpretation must consider the lost product as well as the retained substrate.

Selectivity and Off-Target Questions

The 2018 characterization reported selectivity against a limited tested panel and cellular activity consistent with NNMT inhibition. However, broad modern off-target profiling, transporter effects, ion-channel testing, genotoxicity, organ toxicology and long-duration exposure data suitable for human risk assessment were not identified. “Selective” in a specific experimental panel should not be read as “free of off-target effects” under all biological conditions.

6. Evidence Map

The table below separates what each experiment contributes from what it cannot establish. “No direct human inference” does not mean the work lacks value; it means the study answers a preclinical question rather than a clinical one.

Study	Model	Exposure	Main contribution	Human inference
Kraus 2014	Genetic mouse/cell	NNMT knockdown	Established metabolic target rationale; not a compound test.	None directly
Neelakantan 2017	Biochemical	Compound series	Defined structure-activity relationships and research-tool design.	None
Neelakantan 2018	Cells + obese mice	5-Amino-1MQ	Target inhibition and short mouse metabolic outcomes.	No efficacy/safety
Neelakantan 2019	Cells + aged injured mice	NNMT inhibitor	Muscle stem-cell and regeneration endpoints.	No healthy-human claim
Awosemo 2021	Rats	5-AMQ	Validated bioanalysis and animal PK/oral bioavailability.	No human PK
Sampson 2021	Obese mice	5A1MQ + diet	Combined intervention and body-composition endpoints.	Cannot isolate diet translation
Dimet-Wiley 2022	Obese mice	5A1MQ + diet	Exploratory microbiome associations.	No causal human microbiome claim
Babula 2024	Obese mice	5A1MQ	28-day metabolic, liver and tissue-exposure endpoints.	No disease-treatment claim
Dimet-Wiley 2024	Aged mice	5A1MQ ± exercise	Muscle function, histology and multi-omics.	No exercise substitute
Akar 2021	HeLa cells	5MQ	Cell proliferation and apoptosis markers.	No cancer treatment claim
Dong 2025	PAD mice	NNMT inhibitor	Experimental limb-function outcomes in ischemic muscle.	Compound/model limited
Chanvillard 2026	Cells/organoids/ mice	Selective NNMT inhibition	Kidney senescence/fibrosis target evidence.	Not human therapy; inhibitor context

7. Study-by-Study Review

The following profiles are organized by scientific question rather than by marketing theme. Each profile states whether the experiment tested 5-Amino-1MQ directly, supported the NNMT target, or used a broader inhibitor-class approach.

STUDY 01 | FOUNDATIONAL NNMT TARGET EVIDENCE

Genetic reduction of NNMT established the metabolic target rationale

Kraus D, et al. Nature. 2014;508:258–262. doi:10.1038/nature13198.

Design. Researchers used antisense oligonucleotides and cell experiments to reduce NNMT expression in white adipose tissue and liver in models of diet-induced obesity. This study did not administer 5-Amino-1MQ.

Main findings. NNMT expression was higher in white adipose tissue and liver of obese and diabetic mice. Reducing NNMT protected against diet-induced weight gain and was associated with increased energy expenditure, altered adipose SAM and NAD+ pools, histone-methylation changes, increased polyamine flux and higher adipocyte oxygen consumption.

Interpretation boundary. Genetic knockdown differs from reversible small-molecule inhibition in timing, tissue distribution, degree of suppression and potential compensatory responses. The paper validates a pathway hypothesis; it does not establish a compound's safety or a human weight-loss effect.

STUDY 02 | DIRECT 5-AMINO-1MQ EVIDENCE

Medicinal chemistry defined a cell-permeable inhibitor series

Neelakantan H, et al. J Med Chem. 2017;60:5015–5028.

Design. A medicinal-chemistry program synthesized and tested quinolinium analogues to relate chemical structure to NNMT inhibition, cell permeability and biological activity.

Main findings. The work identified 5-Amino-1MQ as a practical small-molecule probe with low-micromolar biochemical potency and cellular activity. It provided the chemistry foundation for later mouse studies.

Interpretation boundary. Structure-activity work establishes tool properties under defined assays. Potency values depend on assay conditions and do not predict a safe or effective human exposure. A compound can be useful experimentally without being clinically suitable.

STUDY 03 | DIRECT 5-AMINO-1MQ EVIDENCE

5-Amino-1MQ inhibited NNMT and changed short-term obesity-model endpoints

Neelakantan H, et al. Biochem Pharmacol. 2018;147:141–152. PMID 29155147. doi:10.1016/j.bcp.2017.11.033.

Design. Investigators characterized enzyme inhibition, cellular entry and effects on intracellular 1-MNA, then repeatedly administered the compound subcutaneously for 11 days to diet-induced obese mice.

Main findings. The reported biochemical IC50 was approximately 1.2 μM. In differentiated adipocytes, 5-Amino-1MQ reduced intracellular 1-MNA in a concentration-dependent manner. Treated obese mice had lower body weight, white-adipose mass and adipocyte size than vehicle controls. Food intake was not significantly reduced during the study.

Interpretation boundary. The exposure was short, the model was murine and the route was experimental. The study did not establish long-term toxicity, durability after discontinuation, a human dose, or a clinically meaningful weight-management outcome. "Food intake unchanged" does not by itself prove a specific energy-expenditure mechanism.

STUDY 04 | DIRECT 5-AMINO-1MQ EVIDENCE

NNMT inhibition was tested in aged muscle after controlled injury

Neelakantan H, et al. Biochem Pharmacol. 2019;163:481–492. PMID 30753815.

Design. Very old mice underwent a barium-chloride muscle injury, a standardized model used to study impaired regeneration. Cultured muscle stem cells and in vivo inhibitor exposure were used to examine activation, fusion, fibre repair and contractile function.

Main findings. The inhibitor increased markers of muscle stem-cell activation and fusion. At the assessed point after injury, the authors reported larger regenerated myofibres and improved peak torque relative to controls.

Interpretation boundary. A chemical-injury model in aged mice is not equivalent to training recovery, ordinary muscle soreness, human sarcopenia or muscle growth in healthy people. The study supports mechanistic investigation of regeneration, not a personal-use or performance claim.

STUDY 05 | DIRECT 5-AMINO-1MQ EVIDENCE

Rat pharmacokinetics demonstrated measurable oral exposure

Awosemo O, et al. J Pharm Biomed Anal. 2021;204:114255. PMID 34304009. doi:10.1016/j.jpba.2021.114255.

Design. Researchers developed and validated an LC-MS/MS assay for quantifying 5-AMQ in rat plasma and urine, then applied it to intravenous and oral pharmacokinetic experiments.

Main findings. The assay was linear over 10–2500 ng/mL and met stated precision and accuracy criteria. In rats, the reported terminal half-life averaged 3.80 ± 1.10 hours after intravenous exposure and 6.90 ± 1.20 hours after oral exposure; reported oral bioavailability was 38.4%.

Interpretation boundary. These are rat pharmacokinetic values from a specific formulation and design. They do not establish human oral bioavailability, half-life, dose frequency or safety. Pharmacokinetic exposure is not evidence of therapeutic benefit.

STUDY 06 | DIRECT 5-AMINO-1MQ EVIDENCE

NNMT inhibition was combined with a lower-calorie diet in obese mice

Sampson CM, et al. Sci Rep. 2021;11:5637. PMID 33707534. doi:10.1038/s41598-021-85051-6.

Design. Diet-induced obese mice were switched from a Western-style diet to a leaner, reduced-calorie diet and received either inhibitor or control treatment. The design examined whether NNMT inhibition changed the response to dietary transition.

Main findings. The combined intervention accelerated reductions in body weight and fat mass, increased the lean-mass-to-body-weight ratio, and reduced liver and epididymal fat-pad weights relative to diet-only controls. Multiple measures moved toward those of lean comparison animals.

Interpretation boundary. The intervention intentionally combined diet change and inhibitor exposure. The full outcome cannot be attributed to 5-Amino-1MQ alone, and the mouse diet transition cannot be converted into a human diet protocol or expected magnitude of weight change.

STUDY 07 | DIRECT 5-AMINO-1MQ EVIDENCE

Microbiome changes were explored during combined diet and NNMT inhibition

Dimet-Wiley A, et al. Sci Rep. 2022;12:484. PMID 35013352. doi:10.1038/s41598-021-03670-5.

Design. This secondary analysis examined faecal microbial composition in diet-induced obese mice receiving a lower-fat diet with or without 5-Amino-1MQ.

Main findings. The combination was associated with a microbial community pattern distinct from diet alone and from lean controls. The study generated hypotheses about relationships among diet, NNMT inhibition, adiposity and gut ecology.

Interpretation boundary. Microbiome associations do not establish causality. Diet itself strongly changes microbial composition, and mouse microbiomes differ from human microbiomes. The findings do not prove that 5-Amino-1MQ improves gut health or that microbial changes caused the metabolic outcomes.

STUDY 08 | DIRECT 5-AMINO-1MQ EVIDENCE

A 28-day obese-mouse study evaluated metabolism, liver pathology and distribution

Babula JAJ, et al. Diabetes Obes Metab. 2024;26:5272–5282. PMID 39161060. doi:10.1111/dom.15879.

Design. Diet-induced obese mice received once-daily 5A1MQ for 28 days. Investigators measured body composition, oral glucose tolerance, insulin-related outcomes, liver size and histology, circulating markers and compound exposure in metabolically active tissues.

Main findings. Treatment dose-dependently limited additional body-weight and fat-mass gain and improved glucose tolerance, insulin sensitivity and hyperinsulinaemia in the model. Liver weight, triglycerides, steatosis and inflammatory-cell infiltration were reduced. Compound exposure was measured in adipose tissue, skeletal muscle and liver after subcutaneous administration.

Interpretation boundary. The study examined prevention or mitigation of dysfunction in a mouse model, not treatment of human obesity, diabetes or fatty-liver disease. Some authors reported employment by or a founding role in the compound developer, which should be considered alongside the need for independent replication.

STUDY 09 | DIRECT 5-AMINO-1MQ EVIDENCE

5-Amino-1MQ and exercise were compared and combined in aged mice

Dimet-Wiley AL, et al. Sci Rep. 2024;14:15554. PMID 38969654. doi:10.1038/s41598-024-66034-9.

Design. Twenty-two-month-old mice were assigned to sedentary or progressive weighted-wheel-running groups and to daily 5A1MQ or vehicle for eight weeks. Researchers measured grip strength, running, torque, fatigue, muscle size, fibre characteristics, intramyocellular lipid, proteomics and metabolomics.

Main findings. The paper reported approximately 40% greater grip strength in inhibitor-treated sedentary mice, approximately 20% with exercise alone and approximately 60% in the combined group relative to sedentary controls. However, peak plantarflexor torque was not significantly changed. Exercise, rather than inhibitor alone, drove many muscle-mass and fibre-size outcomes. Proteomic and metabolomic responses overlapped only partly.

Interpretation boundary. The result is more nuanced than the phrase “exercise mimetic.” Aged female-mouse grip strength, running behaviour and omics patterns do not establish human strength enhancement, recovery or replacement of exercise. Several endpoints were unchanged, and the sex, age, duration and model limit generalization.

STUDY 10 | DIRECT 5-AMINO-1MQ EVIDENCE

Cell-culture work explored anti-proliferative effects in HeLa cells

Akar S, et al. J Obstet Gynaecol. 2021. PMID 33645410. doi:10.1080/01443615.2020.1854696.

Design. Cultured HeLa cervical cancer cells were exposed to a small-molecule NNMT inhibitor identified as 5MQ, and investigators assessed cell viability and apoptosis-related endpoints.

Main findings. The study reported reduced cell proliferation and changes consistent with apoptosis under the tested culture conditions.

Interpretation boundary. A cancer cell line is not a human treatment model. Concentrations that affect cultured cells may be unachievable or unsafe in vivo, and tumour biology is influenced by absorption, metabolism, immune response and normal-tissue toxicity. This work must not be presented as evidence that 5-Amino-1MQ prevents or treats cancer.

STUDY 11 | EMERGING NNMT INHIBITOR-CLASS EVIDENCE

Experimental peripheral-artery-disease research extended the NNMT hypothesis

Dong G, et al. Physiol Rep. 2025;13:e70615. PMID 41108586. doi:10.14814/phy2.70615.

Design. A mouse model of peripheral artery disease was used to test whether pharmacological NNMT inhibition could influence ischemic limb muscle function and metabolism.

Main findings. The authors reported improved experimental limb-function measures and metabolic changes following NNMT inhibition.

Interpretation boundary. This is disease-model and inhibitor-context evidence. It should not be merged automatically with the obesity studies or described as a proven effect of a particular commercial 5-Amino-1MQ material. It does not establish treatment of human peripheral artery disease.

STUDY 12 | EMERGING NNMT INHIBITOR-CLASS EVIDENCE

Kidney organoid and mouse work examined NNMT, senescence and fibrosis

Chanvillard L, et al. Cell Rep. 2026;45:116823. PMID 41543936. doi:10.1016/j.celrep.2025.116823.

Design. The investigators combined human chronic-kidney-disease transcriptomic and biopsy observations with tubular-cell experiments, kidney organoids, aged mice and diabetic-kidney-disease mouse models. Selective NNMT inhibition was studied in preclinical systems.

Main findings. NNMT expression was associated with senescence and fibrosis signatures in human tissue observations. Inhibition in cells, organoids and mice produced protective changes in early-stage experimental models.

Interpretation boundary. The human component was observational, not a 5-Amino-1MQ clinical trial. The abstract does not justify attributing all results to the same compound, formulation or exposure used in the

metabolic papers. The evidence supports the target's biological relevance, not a human kidney-disease claim.

8. Integrated Interpretation of the Evidence

What Is Well Supported

- 5-Amino-1MQ is a quinolinium small molecule, not a peptide.
- It can inhibit NNMT in biochemical assays and reduce 1-MNA formation in cells.
- It reaches measurable systemic exposure in rats and tissues in mouse studies under the tested conditions.
- NNMT inhibition can change selected metabolic and tissue endpoints in multiple preclinical models.
- The biological consequences are context-dependent and involve more than a single "NAD+" mechanism.

What Is Plausible but Not Clinically Established

The literature makes it plausible that pharmacological NNMT inhibition could influence adipose metabolism, glucose regulation, liver lipid handling, ageing muscle biology and tissue senescence. Plausibility is not proof. Each proposed application requires its own exposure-response work, toxicology, formulation development and controlled human testing. A positive result in one tissue does not guarantee a favourable effect elsewhere.

What Is Not Supported by the Current Evidence

- A claim that 5-Amino-1MQ is proven to cause fat loss in humans.
- A claim that it treats obesity, diabetes, fatty-liver disease, sarcopenia, peripheral artery disease, kidney disease or cancer.
- A claim that it is a safe oral supplement or that rat oral bioavailability establishes a human regimen.
- A claim that it replaces exercise, builds muscle in healthy adults, or reliably improves recovery.
- A claim that increasing or preserving NAD+ is guaranteed, whole-body, durable or clinically beneficial.
- A claim that research-grade purity or a chromatogram establishes suitability for human use.

Why the Literature May Look Stronger Than It Is

Several papers use confident language because scientific articles often discuss therapeutic potential. That language describes a future research direction, not a completed regulatory or clinical conclusion. Online summaries frequently remove qualifiers such as "in mice," omit unchanged endpoints, collapse diet-plus-compound designs into compound-only claims, and treat animal pharmacokinetics as personal-use guidance. These shortcuts create an evidence mismatch.

The number of papers also overstates independence if several studies share investigators, intellectual property or commercial-development relationships. Repeated findings from the same research network are valuable, but independent laboratories, preregistered designs and transparent publication of null results provide a stronger test of reproducibility.

9. Safety, Toxicology and Evidence Gaps

The most important limitation is not simply that human efficacy is unknown. A clinical development program must characterize exposure and risk before benefit can be weighed responsibly. The literature reviewed here does not provide a complete human-relevant safety package.

Major Unanswered Safety Questions

- Acute and repeated-dose toxicity across relevant species and durations.
- Cardiovascular safety, including blood pressure, rhythm, repolarization and haemodynamic effects.
- Liver and kidney toxicity under repeated systemic exposure.
- Neurologic and behavioural effects, including consequences of tissue distribution not captured in short studies.
- Genotoxicity, carcinogenicity and the implications of altering NNMT in tissues where its role may be protective or context-dependent.
- Fertility, embryo-fetal development, reproductive toxicity and transfer during pregnancy or lactation.
- Interactions with medicines, supplements, alcohol, nicotine, dietary patterns and other compounds affecting NAD+ or methyl-donor metabolism.
- Consequences of chronic reduction of 1-MNA and alteration of SAM/SAH balance.
- Effects of impurities, residual solvents, counterion content, degradation products and non-sterile handling in unregulated materials.

Why Short Animal Studies Cannot Close These Gaps

An 11-day or 28-day mouse study can identify selected signals but cannot exclude uncommon, delayed, reproductive or human-specific adverse effects. Species differences in absorption, transporters, metabolism, renal clearance and enzyme expression can change both exposure and toxicity. Even the rat half-life and oral bioavailability values apply only to that model and experimental formulation.

Quality and Analytical Limitations

Scientific papers test materials characterized for a defined protocol. A commercial vial with the same name is not automatically equivalent. Research interpretation depends on identity, concentration, purity, salt form, stability and handling. HPLC purity is not an all-purpose safety certificate, and LC-MS molecular-mass confirmation does not establish sterility or endotoxin status. Only tests listed on a batch-specific report should be represented as performed.

SAFETY CONCLUSION
The absence of a reported severe event in a small preclinical experiment is not evidence that a material is safe for human consumption. “No safety signal observed” and “safety established” are scientifically different statements.

10. Frequently Asked Questions

Is 5-Amino-1MQ a peptide?

No. It is a synthetic quinolinium small molecule. The “amino” in its chemical name does not make it a peptide.

What does 5-Amino-1MQ inhibit?

It is studied primarily as an inhibitor of nicotinamide N-methyltransferase, or NNMT.

What does NNMT do?

NNMT transfers a methyl group from SAM to nicotinamide, producing 1-MNA and SAH. This links nicotinamide metabolism with methyl-donor balance.

Does 5-Amino-1MQ raise NAD+?

Some preclinical studies report changes in NAD+-related pathways or pools after reducing NNMT. A consistent, whole-body, clinically meaningful increase in human NAD+ has not been established.

Has 5-Amino-1MQ been studied in humans?

No completed controlled human safety or efficacy trial was identified in this review. Published compound-specific evidence is predominantly cellular and animal research.

Does it cause weight loss?

Short mouse studies reported changes in body weight and adipose tissue. Those results do not establish human weight loss or a safe method of use.

Is it an exercise mimetic?

That phrase overstates the evidence. One aged-mouse study found overlap in some muscle outcomes and molecular signatures, while other endpoints differed or were unchanged. Exercise has broad effects that cannot be reduced to NNMT inhibition.

What did the rat pharmacokinetic study show?

It showed that 5-AMQ could be quantified in rat plasma and urine and produced measurable exposure after oral administration, with reported oral bioavailability of 38.4% in that model. It did not establish a human dose or regimen.

Is 5-Amino-1MQ approved by Health Canada?

No Health Canada market authorization as a therapeutic product was identified in this review. It should not be presented as an approved treatment, preventative or cure.

Does a high HPLC purity result prove safety?

No. HPLC addresses the chromatographic profile under a stated method. It does not by itself establish identity, dose accuracy, sterility, endotoxin, residual solvents, elemental impurities or human suitability.

What is the most accurate one-sentence description?

5-Amino-1MQ is a preclinical small-molecule NNMT inhibitor used to investigate metabolic and tissue-specific biology in laboratory and animal models.

What research is still needed?

Independent replication, standardized exposure-response studies, comprehensive toxicology, human pharmacokinetics and carefully controlled clinical trials would be required before human safety or efficacy conclusions could be made.

11. Canadian Publication and Regulatory Context

Health Canada distinguishes information from advertising by examining the purpose and overall context of a message. Under its guidance, no single disclaimer or factor determines the result. The regulator may consider the provider, sponsor, audience, balance, scientific rigour, visual presentation, product focus, linkages to sales material and whether benefits are emphasized while risks or uncertainties are minimized.

Health Canada's guidance specifically notes that an educational message involving an unauthorized health product may be considered promotional if it does not caution that safety and effectiveness remain under investigation and that market authorization has not been granted. It also warns that brand colours, packaging-like visuals, product-focused layouts and links to related commercial material can contribute to a promotional determination.

Publication Principles for a Lower-Risk Educational Page

- Place the paper in a clearly labelled Research Library or Scientific Education section.
- Keep the evidence-limitations notice visible near the beginning, not only in a footer.
- Use neutral molecular, pathway and study visuals rather than transformation imagery, syringes or implied personal use.
- Do not include dosing, cycling, stacking, reconstitution, administration or self-experimentation instructions.
- Avoid testimonials, before-and-after claims, outcome guarantees and calls to action tied to health benefits.
- Link directly to primary studies and identify the model—cell, rat, mouse, organoid or human—beside each result.
- Disclose the page author, review date, editorial method, commercial relationship and conflicts or funding noted in the source studies.
- Keep product specifications and batch testing factual; do not imply that analytical purity equals clinical safety.
- Have Canadian regulatory counsel or an advertising preclearance agency assess the final page in its complete commercial context.

IMPORTANT
This paper can improve scientific balance, but it cannot guarantee compliance. The final determination depends on the complete website, links, visuals, purchasing pathway, audience and actual business activities.

12. SEO-Ready Website Publication Package

Google does not provide a "compliance" certificate or guarantee ranking. For a health-related topic, the strongest strategy is a helpful, transparent, well-sourced page designed for readers first. Google states that

trust is especially important for topics that may affect health and recommends clear authorship, original value, strong organization, accurate dates and reliable sourcing.

Field	Research description
Suggested URL slug	/research/5-amino-1mq-nnmt-evidence-review/
SEO title	5-Amino-1MQ Research Review: NNMT, Studies & Evidence
Meta description	A plain-language scientific review of 5-Amino-1MQ, NNMT inhibition, preclinical studies, mechanisms, limitations and major research gaps.
H1	5-Amino-1MQ: Scientific Review of NNMT Research and Available Evidence
Primary topic	5-Amino-1MQ research
Related terms	5A1MQ; 5-AMQ; NNMT inhibitor; nicotinamide N-methyltransferase; preclinical research; NNMT studies.
Author display	Vanguard Compound Research Editorial Team — do not invent medical or scientific credentials.
Review date	Published date plus “Last reviewed: 7 September 2026.”
Recommended schema	Article or BlogPosting JSON-LD with Organization author, dates, headline, canonical URL and representative image.
FAQ treatment	Keep the visible FAQ for readers. Do not expect FAQ rich results; eligibility is limited and markup must match visible content.
Canonical	Use one preferred, indexable URL for the full article.
Indexing check	Confirm the page is not blocked by robots.txt, noindex, login or an inaccessible script-only rendering path.

Recommended On-Page Heading Structure

- H1: 5-Amino-1MQ: Scientific Review of NNMT Research and Available Evidence
- H2: What Is 5-Amino-1MQ?
- H2: What Does NNMT Do?
- H2: How Does 5-Amino-1MQ Inhibit NNMT?
- H2: Chemical Identity and Research Specifications
- H2: Evidence from Cell and Animal Studies
- H2: Study-by-Study Review
- H2: What the Evidence Does Not Establish
- H2: Safety and Research Gaps
- H2: Frequently Asked Questions
- H2: References and Review Method

Trust and Quality Signals to Add to the Webpage

- A visible byline and an About/Editorial Policy link explaining who prepared the page and how sources were selected.

- A published date, last-reviewed date and short change log when the scientific review is updated.
- Direct links to PubMed, PubMed Central, DOI or the journal—not retailer summaries—as the primary citations.
- A conflict-of-interest note for studies involving the compound developer or related intellectual property.
- A plain-language summary followed by the technical review, so both novice and specialist search intent are served on one canonical page.
- Descriptive image alt text and captions that explain the scientific figure rather than repeat keywords.
- Internal links to an analytical-testing explainer, terminology glossary, editorial policy and research-only statement; avoid benefit-focused product anchors.
- A concise medical-information notice and a separate legal/regulatory review of the finished page.

Suggested Article Structured Data

```
{
  "@context": "https://schema.org",
  "@type": "Article",
  "headline": "5-Amino-1MQ: Scientific Review of NNMT Research and Available Evidence",
  "description": "A plain-language scientific review of 5-Amino-1MQ, NNMT inhibition, preclinical studies,
mechanisms, limitations and major research gaps.",
  "author": {"@type": "Organization", "name": "Vanguard Compound Research"},
  "publisher": {"@type": "Organization", "name": "Vanguard Compound Research"},
  "datePublished": "YYYY-MM-DD",
  "dateModified": "2026-09-07",
  "mainEntityOfPage": "https://YOUR-DOMAIN/research/5-amino-1mq-nnmt-evidence-review/",
  "image": ["https://YOUR-DOMAIN/path/5-amino-1mq-research-graphic.jpg"]
}
```

Replace all placeholders with real canonical URLs and publication dates. The structured data must match visible page content. Validate it with Google's Rich Results Test and Schema Markup Validator before deployment.

Keyword and Writing Guidance

Use the full name and key synonym naturally in the title, introduction, one or two headings, figure alt text and study section. Do not repeat "5-Amino-1MQ" mechanically in every sentence. Google's systems can understand related language such as NNMT inhibitor, 5A1MQ, 5-AMQ, nicotinamide metabolism and preclinical research. Accuracy, source quality and reader satisfaction matter more than keyword density.

Avoid pages that separately target trivial spelling variations or repeat the same article with slightly different titles. Publish one strong canonical review and update it when meaningful new data appear. Keep product pages short and factual; place the complete evidence review in the research library so the scientific discussion is not reduced to promotional copy.

13. References

1. Kraus D, et al. Nicotinamide N-methyltransferase knockdown protects against diet-induced obesity. Nature. 2014;508:258–262. Source

2. Neelakantan H, et al. Structure-Activity Relationship for Small Molecule Inhibitors of Nicotinamide N-Methyltransferase. J Med Chem. 2017;60:5015–5028. Source

3. Neelakantan H, et al. Selective and membrane-permeable small molecule inhibitors of NNMT reverse high-fat-diet-induced obesity in mice. Biochem Pharmacol. 2018;147:141–152. Source

4. Neelakantan H, et al. Small molecule NNMT inhibitor activates senescent muscle stem cells and improves regenerative capacity of aged skeletal muscle. Biochem Pharmacol. 2019;163:481–492. Source

5. Awosemo O, et al. Development and validation of LC-MS/MS assay for 5-amino-1-methyl quinolinium in rat plasma: application to pharmacokinetic and oral bioavailability studies. J Pharm Biomed Anal. 2021;204:114255. Source

6. Sampson CM, et al. Combined NNMT inhibition and reduced-calorie diet normalizes body composition and enhances metabolic benefits in obese mice. Sci Rep. 2021;11:5637. Source

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8. Babula JAJ, et al. Nicotinamide N-methyltransferase inhibition mitigates obesity-related metabolic dysfunction. Diabetes Obes Metab. 2024;26:5272–5282. Source

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11. Dong G, et al. Nicotinamide N-methyltransferase inhibition improves limb function in experimental peripheral artery disease. Physiol Rep. 2025;13:e70615. Source

12. Chanvillard L, et al. NNMT inhibition counteracts tubular senescence and fibrosis in early stages of chronic kidney disease. Cell Rep. 2026;45:116823. Source

13. Ulanovskaya OA, et al. NNMT promotes epigenetic remodeling in cancer by creating a metabolic methylation sink. Nat Chem Biol. 2013;9:300–306. Source

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15. Liu JR, et al. Roles of nicotinamide N-methyltransferase in obesity and type 2 diabetes. Biomedicines. 2021;9:1155. Source

16. PubChem. NNMTi: 5-amino-1-methylquinolin-1-ium iodide. Compound Summary CID 66522933. Source

17. Health Canada. Guidance on distinction between advertising and other activities for health products. Source

18. Google Search Central. Creating helpful, reliable, people-first content. Source

19. Google Search Central. Search Engine Optimization Starter Guide. Source

20. Google Search Central. Article structured data. Source

Editorial Closing Statement

Vanguard Compound Research presents this review to support accurate discussion of experimental science. The review should be updated when material new primary evidence appears. Revisions should preserve the distinction between mechanistic plausibility, animal findings and demonstrated human outcomes.

RESEARCH-USE STATEMENT

For laboratory research information only. Not for human consumption, therapeutic use, veterinary administration or personal experimentation. This paper does not provide dosing, administration, treatment or medical guidance.