

RESEARCH MONOGRAPH . COMPOUND PROFILE

Quercetin

A widely distributed flavonoid with weaker senolytic activity than fisetin, often combined with dasatinib in the canonical D+Q senolytic regimen.

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ABSTRACT

Quercetin (3,3',4',5,7-pentahydroxyflavone; CAS 117-39-5; molecular formula C₁₅H₁₀O₇; molecular weight 302.24) is a widely distributed flavonoid present in onions, apples, capers, and many other plants. The compound has multiple mechanisms of pharmacological interest: mast cell stabilization (anti-allergic effects), direct antioxidant activity, anti-inflammatory effects through NF-κB inhibition, and senolytic activity (in combination with the kinase inhibitor dasatinib in the canonical D+Q regimen developed by Kirkland and Niedernhofer). The senolytic activity of quercetin alone is modest; the dasatinib combination is substantially more effective. Bioavailability is poor as the free aglycone but better as the rutinose glycoside; plasma quercetin concentrations after oral dosing are typically less than 1 percent of the dose. Doses range from 500 mg to 2 grams per day; the senolytic D+Q regimen uses pulsatile dosing of 1000 to 1500 mg quercetin with 100 mg dasatinib for 2 to 3 days monthly.

1. Compound Identification

Quercetin is described as flavonoid senolytic / mast cell stabilizer. A widely distributed flavonoid with weaker senolytic activity than fisetin, often combined with dasatinib in the canonical D+Q senolytic regimen.

1.1 Class summary

Flavonoid senolytic / mast cell stabilizer

1.2 Subtitle

A widely distributed flavonoid with weaker senolytic activity than fisetin, often combined with dasatinib in the canonical D+Q senolytic regimen.

2. Abstract

Quercetin (3,3',4',5,7-pentahydroxyflavone; CAS 117-39-5; molecular formula C₁₅H₁₀O₇; molecular weight 302.24) is a widely distributed flavonoid present in onions, apples, capers, and many other plants. The compound has multiple mechanisms of pharmacological interest: mast cell stabilization (anti-allergic effects), direct antioxidant activity, anti-inflammatory effects through NF-κB inhibition, and senolytic activity (in combination with the kinase inhibitor dasatinib in the canonical D+Q regimen developed by Kirkland and Niedernhofer). The senolytic activity of quercetin alone is modest; the dasatinib combination is substantially more effective. Bioavailability is poor as the free aglycone but better as the rutinose glycoside; plasma quercetin concentrations after oral dosing are typically less than 1 percent of the dose. Doses range from 500 mg to 2 grams per day; the senolytic D+Q regimen uses pulsatile dosing of 1000 to 1500 mg quercetin with 100 mg dasatinib for 2 to 3 days monthly.

3. Mechanism of Action

Mast cell stabilizer, anti-inflammatory, weak senolytic. The dasatinib + quercetin (D+Q) combination is the established senolytic regimen.

The mechanistic profile described above represents the consensus position from peer-reviewed literature where available; for compounds with sparse formal characterization, this section reflects vendor-grade reports and structurally-related class pharmacology and should be interpreted with appropriate caution.

4. Pharmacokinetics and Routes of Administration

Pharmacokinetic data are summarized within the abstract for compounds with formal Phase 1 records. Compounds without published human pharmacokinetic studies are noted as such; investigators should treat the absence of data as a research limitation.

5. Reported Research Dose Ranges

500 to 2000 mg per day; 1000-1500 mg pulsatile in D+Q senolytic protocols.

Doses are scaled from clinical use where the compound is approved as a medicine, from Phase 2/3 trials where development advanced, or from rodent dose-response data scaled by allometric assumptions where the compound has no human clinical record. Investigators should treat dose ranges from research-chemical-grade vendors as starting points for their own pharmacokinetic and tolerability characterization rather than validated dosing recommendations.

6. Sourcing and Quality Verification

Investigators should obtain analytical confirmation of identity (mass spectrometry; for peptides, additionally amino acid analysis or N-terminal sequencing) and purity (HPLC area-percent, ideally greater than 95 percent) on every lot. Vendor certificates of analysis vary in quality; third-party analytical confirmation through independent laboratories (e.g., Janoshik Analytical, Anabolic Lab) is the gold standard for research-grade peptides and small molecules.

For compounds with established pharmaceutical-grade marketing in any jurisdiction, sourcing from the marketed product (where legal and accessible) provides the highest quality assurance.

7. Reconstitution and Handling

Peptides should be reconstituted in bacteriostatic water or sterile water for injection in the volume appropriate to the desired concentration; lyophilized peptides should be allowed to come to room temperature before reconstitution to minimize precipitation. Reconstituted peptides should be stored refrigerated and used within the timeframe specified by the manufacturer (typically 14 to 30 days for bacteriostatic water reconstitutions, shorter for sterile water).

Small molecules: storage conditions vary by compound; consult the certificate of analysis for the specific product. Most are stable at room temperature in dry, dark conditions; oxidation-sensitive compounds (e.g., catecholamine precursors) require cool storage and protection from light.

8. Stack Interaction Considerations

Investigators combining this compound with other research substances should consider receptor-level interactions (additive effects at shared targets), enzyme-level interactions (CYP-mediated drug-drug interactions where applicable), and pharmacodynamic interactions (additive sedation, additive serotonergic activity, additive hemodynamic effects).

The stack interaction section in published monographs of well-characterized compounds is necessarily incomplete for any specific combination; investigators should treat novel combinations with elevated caution and titrate gradually.

9. References

1. Zhu Y, et al. The Achilles' heel of senescent cells: from transcriptome to senolytic drugs. Aging Cell 2015.

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