

RESEARCH MONOGRAPH . COMPOUND PROFILE

Dasatinib

*A multi-target tyrosine kinase inhibitor
approved for chronic myeloid leukemia;
repurposed in senolytic combination
therapy with quercetin (D+Q).*

DOCUMENT	KDC-MN-297
ISSUED	2026-05-09
REVISION	1.0
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COMPOUND CLASS	Tyrosine kinase inhibitor / senolytic

ABSTRACT

Dasatinib (BMS-354825; CAS 302962-49-8; molecular formula $C_{22}H_{26}ClN_7O_2S$; molecular weight 488.01) is a multi-target tyrosine kinase inhibitor developed at Bristol-Myers Squibb and approved by the FDA in 2006 under the trade name Sprycel for chronic myeloid leukemia and Philadelphia chromosome-positive ALL. The senolytic application emerged from work by Kirkland and Tchkonja at Mayo Clinic (2015 onward) demonstrating that the combination of dasatinib (targeting senescent cells of mesenchymal origin) and quercetin (targeting senescent cells of epithelial origin) selectively eliminates senescent cells while sparing healthy cells. The D+Q regimen has been advanced into multiple clinical trials in idiopathic pulmonary fibrosis, diabetic kidney disease, Alzheimer disease, and other age-related conditions, with intermittent dosing (e.g., 100 mg daily for 3 days monthly) hypothesized to clear accumulated senescent cells without continuous chemotherapy exposure. Plasma half-life is approximately 5 hours; metabolism is via CYP3A4. Used as the canonical senolytic small molecule with clinical translation.

1. Compound Identification

Dasatinib is described as tyrosine kinase inhibitor / senolytic. A multi-target tyrosine kinase inhibitor approved for chronic myeloid leukemia; repurposed in senolytic combination therapy with quercetin (D+Q).

1.1 Class summary

Tyrosine kinase inhibitor / senolytic

1.2 Subtitle

A multi-target tyrosine kinase inhibitor approved for chronic myeloid leukemia; repurposed in senolytic combination therapy with quercetin (D+Q).

2. Abstract

Dasatinib (BMS-354825; CAS 302962-49-8; molecular formula $C_{22}H_{26}ClN_7O_2S$; molecular weight 488.01) is a multi-target tyrosine kinase inhibitor developed at Bristol-Myers Squibb and approved by the FDA in 2006 under the trade name Sprycel for chronic myeloid leukemia and Philadelphia chromosome-positive ALL. The senolytic application emerged from work by Kirkland and Tchkonja at Mayo Clinic (2015 onward) demonstrating that the combination of dasatinib (targeting senescent cells of mesenchymal origin) and quercetin (targeting senescent cells of epithelial origin) selectively eliminates senescent cells while sparing healthy cells. The D+Q regimen has been advanced into multiple clinical trials in idiopathic pulmonary fibrosis, diabetic kidney disease, Alzheimer disease, and other age-related conditions, with intermittent dosing (e.g., 100 mg daily for 3 days monthly) hypothesized to clear accumulated senescent cells without continuous chemotherapy exposure. Plasma half-life is approximately 5 hours; metabolism is via CYP3A4. Used as the canonical senolytic small molecule with clinical translation.

3. Mechanism of Action

Multi-target tyrosine kinase inhibitor (BCR-ABL, SRC family, KIT, PDGFR, EPHA2). Senolytic activity via SRC-mediated apoptosis pathways in senescent cells of mesenchymal origin.

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

Clinical CML 100 mg per oral administration daily. Senolytic 100 mg daily for 3 days monthly (with quercetin 1000 mg daily for 3 days).

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.
2. Hickson LJ, et al. Senolytics decrease senescent cells in humans: preliminary report from a clinical trial of dasatinib plus quercetin in individuals with diabetic kidney disease. *EBioMedicine* 2019.
3. Justice JN, et al. Senolytics in idiopathic pulmonary fibrosis: results from a first-in-human, open-label, pilot study. *EBioMedicine* 2019.

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