

RESEARCH MONOGRAPH . COMPOUND PROFILE

Bimagrumab

A humanized monoclonal antibody against activin receptor type II; investigated for sarcopenia, sIBM, and obesity-related muscle preservation.

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COMPOUND CLASS	Anti-activin receptor type II monoclonal antibody

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ABSTRACT

Bimagrumab (BYM338; humanized IgG1 monoclonal antibody; molecular weight approximately 145 kDa) is a humanized monoclonal antibody against activin receptor type II (ActRII), developed at MorphoSys and Novartis. The compound binds ActRIIA and ActRIIB receptors on the cell surface, blocking access by myostatin, activin A, and GDF-11; the receptor-level blockade is mechanistically distinct from ligand traps (ACE-031) and from selective myostatin antibodies (stamulumab). Phase 2 trials in sporadic inclusion body myositis (RESILIENT) and sarcopenia demonstrated muscle mass increases. The compound generated substantial interest in 2020 after a phase 2 trial in obesity with type 2 diabetes (BIM-T2D) showed approximately 21 percent reduction in fat mass with concurrent muscle preservation, an unusual profile that has subsequently driven obesity-related development. Used as a research probe for receptor-level myostatin pathway inhibition.

1. Compound Identification

Bimagrumab is described as anti-activin receptor type ii monoclonal antibody. A humanized monoclonal antibody against activin receptor type II; investigated for sarcopenia, sIBM, and obesity-related muscle preservation.

1.1 Class summary

Anti-activin receptor type II monoclonal antibody

1.2 Subtitle

A humanized monoclonal antibody against activin receptor type II; investigated for sarcopenia, sIBM, and obesity-related muscle preservation.

2. Abstract

Bimagrumab (BYM338; humanized IgG1 monoclonal antibody; molecular weight approximately 145 kDa) is a humanized monoclonal antibody against activin receptor type II (ActRII), developed at MorphoSys and Novartis. The compound binds ActRIIA and ActRIIB receptors on the cell surface, blocking access by myostatin, activin A, and GDF-11; the receptor-level blockade is mechanistically distinct from ligand traps (ACE-031) and from selective myostatin antibodies (stamulumab). Phase 2 trials in sporadic inclusion body myositis (RESILIENT) and sarcopenia demonstrated muscle mass increases. The compound generated substantial interest in 2020 after a phase 2 trial in obesity with type 2 diabetes (BIM-T2D) showed approximately 21 percent reduction in fat mass with concurrent muscle preservation, an unusual profile that has subsequently driven obesity-related development. Used as a research probe for receptor-level myostatin pathway inhibition.

3. Mechanism of Action

Monoclonal antibody against activin receptor type II (ActRIIA/B); blocks receptor at cell surface from binding myostatin, activin A, GDF-11.

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

Trial doses 3 to 30 mg/kg intravenous every 4 weeks; subcutaneous 700 to 2100 mg every 4 weeks.

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. Heymsfield SB, et al. Effect of bimagrumab vs placebo on body fat mass among adults with type 2 diabetes and obesity. JAMA Netw Open 2021.
2. Amato AA, et al. Treatment of sporadic inclusion body myositis with bimagrumab. Neurology 2014.
3. Rooks D, et al. Bimagrumab vs optimized standard of care for treatment of sarcopenia. JAMA Netw Open 2017.

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