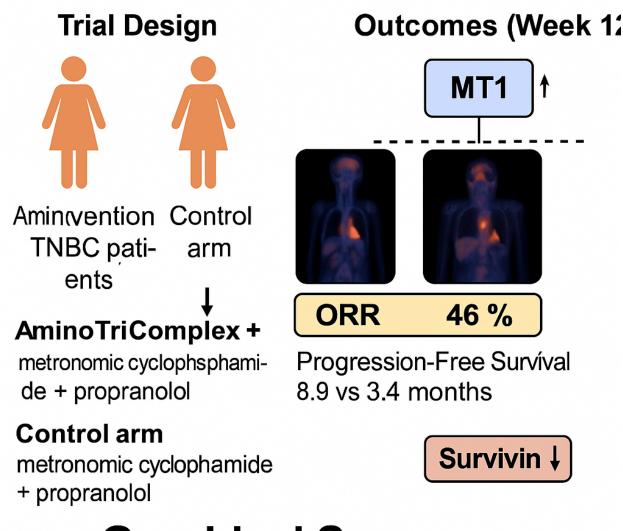
Tavartkiladze A, Simonia G, Reiter RJ, Lou R, Okrostsvaridze N, Kasradze D, et al. Amino Tricomplex Therapy in Advanced Triple-Negative Breast Cancer: A Translational Study Linking Survivin Downregulation, Cystatin C Upregulation, and MT1 Receptor Re-Expression to Clinical Outcomes. J Cancer Immunol. 2025;7(3):128–143.

Supplementary Figure 1 (GS2). Mechanistic Graphical Summary. Integration of the biomarker triad with clinical outcomes.

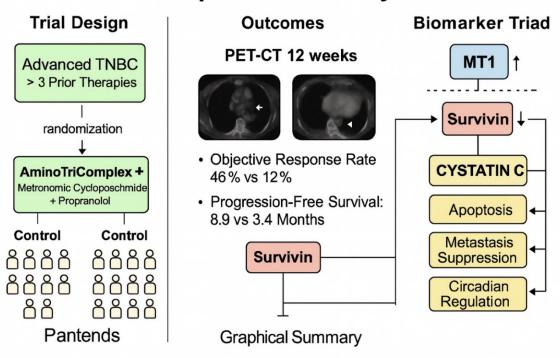
Illustration of trial design, PET-CT outcomes, and biomarker interactions. **MT1 re-expression** suppresses NF-κB/STAT3 signaling, leading to **Survivin downregulation**. Concurrently, circadian regulation enhances **Cystatin C upregulation**, resulting in apoptosis restoration, metastasis suppression, and circadian reactivation. This mechanistic model explains how the **Survivin**↓, **Cystatin C**↑, **MT1**↑ **triad** underlies clinical efficacy of AminoTriComplex.

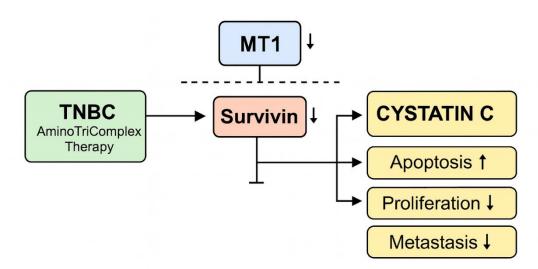


Graphical Summary

Tavartkiladze A, Simonia G, Reiter RJ, Lou R, Okrostsvaridze N, Kasradze D, et al. Amino Tricomplex Therapy in Advanced Triple-Negative Breast Cancer: A Translational Study Linking Survivin Downregulation, Cystatin C Upregulation, and MT1 Receptor Re-Expression to Clinical Outcomes. J Cancer Immunol. 2025;7(3):128–143.

Graphical Summary





Translational Model Between AminoTriComplex and Biomarker Regulation in Advanced TNBC

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Survivin↓ + MT1↑ + Cystatin C↑ into a unified biomarker model figure (a schematic pathway diagram) for part of discussion

