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Discovery of anticancer candidate targeting peroxiredoxin 2-deficient Triple-Negative Breast Cancer for personalized treatment

VasThera Co., Ltd.

VASTHERA

ONCOLOGY | **Candidate**

Product Type	Small molecule
Indication	Triple-Negative Breast Cancer (TNBC)
Target	Peroxiredoxin 2 (PRDX2)
MoA(Mechanism of Action)	Under chronic H_2O_2 stress, EGFR becomes hyperactivated due to persistent oxidative modifications. Peroxiredoxin 2 (PRDX2) is the key redox enzyme that controls oxidative modifications of EGFR. PRDX2, however, is epigenetically silenced in TNBC. VTA is a mechanism-driven compound that mimics catalytic activity of PRDX2. Upon treatment, VTA compensates for the silenced PRDX2 and inhibits hyperactivation of EGFR by blocking oxidative modifications, suppressing growth and metastasis of TNBC.
Competitiveness	Competitor: Cetuximab, Trodelvy® - Antibody agents such as cetuximab are approved only for colorectal and head-and-neck cancers, and no EGFR-pathway biologics or biosimilars are currently available for TNBC. - VTA uniquely suppresses redox-driven EGFR hyperactivation and downstream metastatic signaling, representing a first-in-class mechanism distinct from conventional EGFR TKIs or antibodies.
Development Stage	Candidate
Route of Administration	Oral administration