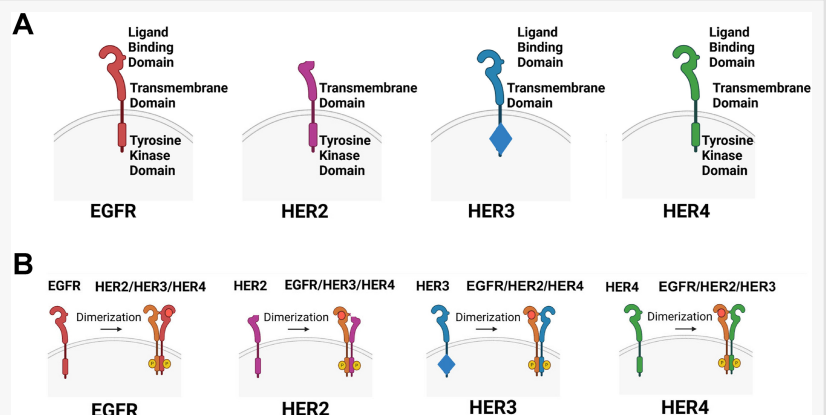


HER3 Re-Emerges as a Pivotal Target in the Fight Against Cancer

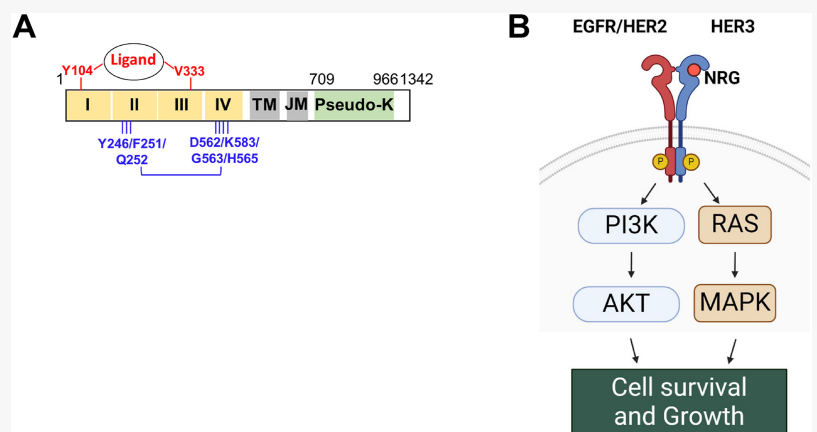
SHANNON, CLARE, IRELAND, May 12, 2025 /EINPresswire.com/ -- A new publication shines a spotlight on HER3, a long-overlooked member of the ErbB receptor family, revealing its critical role in cancer progression and resistance to therapy. Once considered a passive player due to its weak kinase activity, HER3 is now recognized as a major contributor to the survival and spread of various solid tumors, including breast, lung, colorectal, pancreatic, and gynecologic cancers.

HER3 functions through its interaction with other ErbB receptors, particularly HER2, forming potent signaling pairs that drive cell survival, proliferation, and metastasis. These dimerized complexes activate key downstream pathways, such as MAPK and PI3K/Akt, both essential in cancer cell growth and evasion of apoptosis. Overexpression or mutation of HER3 in tumor cells correlates with poor patient outcomes and therapeutic resistance, positioning it as a high-value therapeutic target.

Despite the development of multiple HER3-targeted therapies, most clinical applications have yielded modest results. The limited success is now attributed to the failure to match treatments to patients with active HER3 signaling. Crucially, only a subset of tumors—such as those with NRG1 gene fusions or high HER3 expression—appear to respond well, highlighting the need for predictive biomarkers to guide treatment selection. Without precise patient stratification, many



Schematic illustration of ErbB receptor structures and dimerization patterns. ErbB, ERB-B2 receptor tyrosine kinase.



Schematics of HER3 structure and downstream signaling.

promising therapies fall short of their potential.

The tumor microenvironment plays a decisive role in regulating HER3 activity. Stromal components, particularly fibroblasts and liver endothelial cells, secrete factors that activate HER3 independently of its known ligands, contributing to therapy resistance and disease recurrence. This underscores the importance of considering non-genetic activation mechanisms in future treatment strategies.

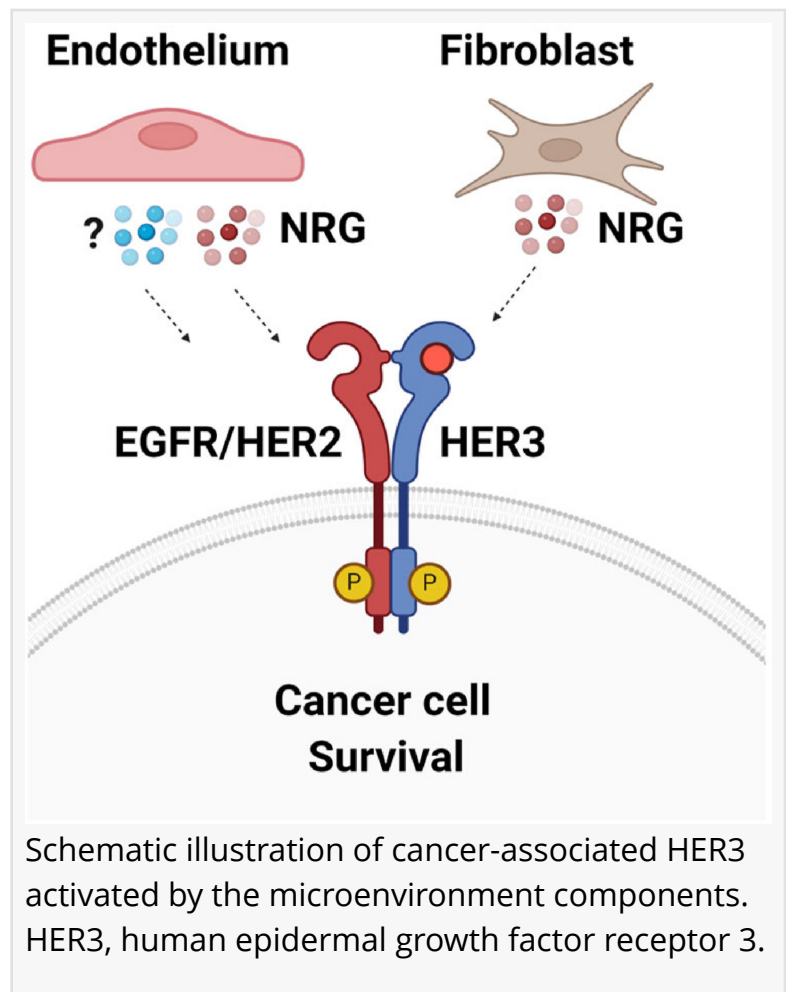
Antibody-drug conjugates (ADCs) have emerged as a powerful approach to overcome resistance. By combining HER3-targeting antibodies with cytotoxic payloads, ADCs selectively eliminate HER3-positive cancer cells while minimizing systemic toxicity. Encouraging early results in HER3-expressing breast and lung cancers demonstrate their potential to reshape HER3-directed therapies.

The review also calls for integrating HER3 expression profiling into clinical trial design and patient care. Accurate detection methods and biomarker-driven therapies could transform HER3 from a failed target to a cornerstone of precision oncology.

With a better understanding of its biology, interaction with the microenvironment, and the emergence of next-generation therapies, HER3 is being redefined as a central player in cancer therapeutics—one that may finally deliver on its long-overdue promise.

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Reference

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