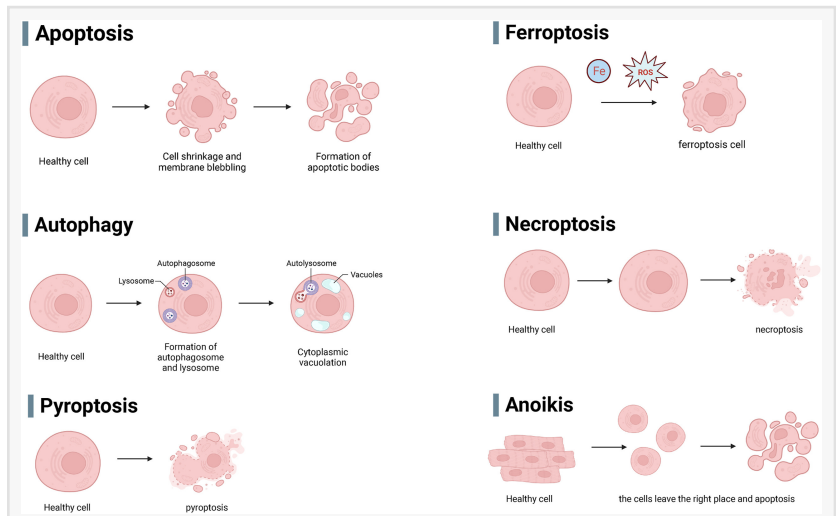


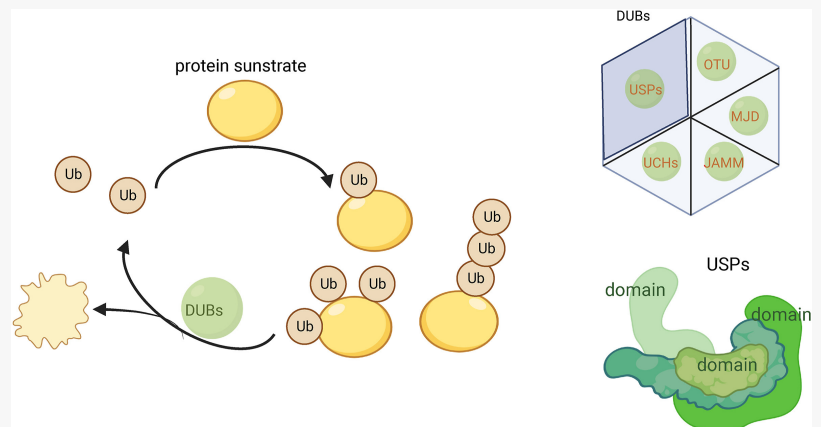
New Avenues in Breast Cancer Treatment: The Role of Ubiquitin-Specific Proteases in Programmed Cell Death

SHANNON, CLARE, IRELAND, March 10, 2025 /EINPresswire.com/ -- This review highlights the critical role of ubiquitin-specific proteases (USPs) in regulating programmed cell death (PCD) in breast cancer (BC). As the most prevalent malignant tumor among women, BC remains a significant global health challenge. Despite advances in therapy, resistance to treatment and metastasis continue to pose major obstacles. This article presents a comprehensive analysis of how ubiquitination and deubiquitination processes, governed by USPs, influence various cell death pathways crucial to cancer progression and treatment response.

The intricate ubiquitin-proteasome system (UPS) plays a vital role in maintaining cellular homeostasis, determining the fate of apoptotic, autophagic, necroptotic, ferroptotic, and pyroptotic pathways. By selectively modulating proteins involved in these pathways, USPs contribute to either promoting or inhibiting cancer cell survival. The review explores the dual role of PCD in BC, where certain forms of cell death act as a defense mechanism, while others enhance tumor growth and resistance to therapy.



Different types of programmed cell death. This picture summarizes the different pathways of programmed cell death (Note: ROS: reactive oxygen species).



Overview of the process of ubiquitination and the structure of ubiquitin specific protease.

Apoptosis, the most studied form of PCD, is closely linked to tumor suppression. However, BC

cells often develop resistance to apoptotic signals, enabling uncontrolled proliferation. USPs such as USP22 and USP7 regulate key proteins like c-Myc and p53, influencing apoptosis in BC. Similarly, autophagy, a mechanism for cellular self-digestion, has a paradoxical role in BC, either inhibiting or promoting tumor survival. The review discusses how USP8 and USP13 influence autophagy by regulating Beclin1 and p62/SQSTM1, potentially affecting therapeutic outcomes.

Emerging non-apoptotic PCD pathways such as ferroptosis and pyroptosis have garnered attention for their

potential in cancer therapy. Ferroptosis, a form of iron-dependent cell death, has been identified as a promising target in triple-negative breast cancer (TNBC). The review highlights the role of USP7 and USP35 in ferroptosis regulation, underscoring the therapeutic possibilities of manipulating iron metabolism and oxidative stress. Pyroptosis, an inflammatory cell death pathway, is also examined, with a focus on USP48 and gasdermin E (GSDME), which contribute to immune responses and tumor suppression.

Beyond their influence on PCD, USPs play a role in tumor metastasis and drug resistance, presenting new challenges and opportunities for targeted therapy. The review emphasizes the need for further investigation into the crosstalk between USPs and PCD mechanisms, particularly in necroptosis and anoikis, which are less understood but critical in BC progression.

By unveiling the complex interactions between ubiquitination, cell death pathways, and cancer biology, this review lays the foundation for novel therapeutic strategies.

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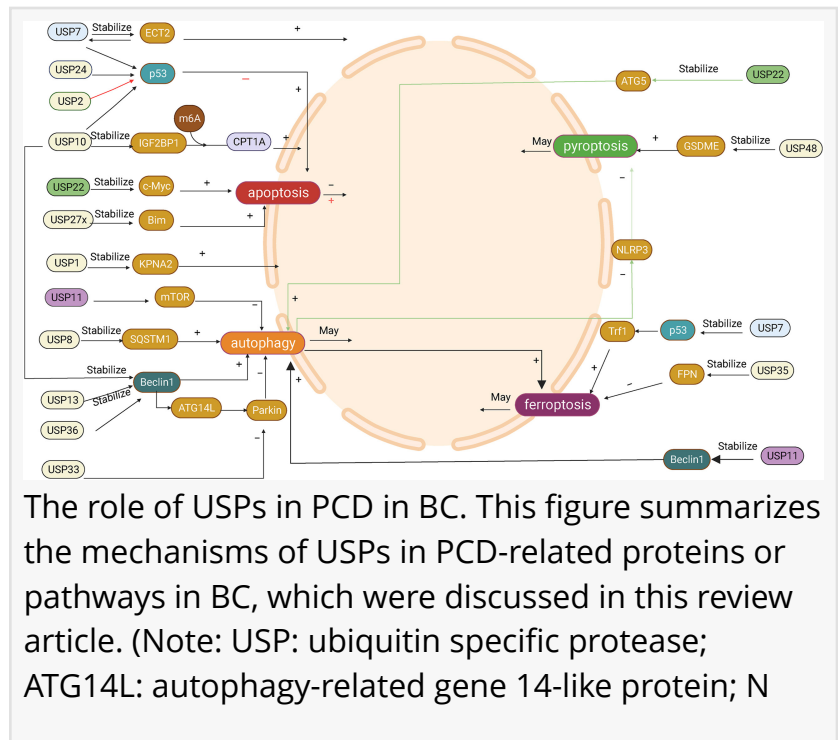
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