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Summary

LOSS OF ATM DRIVES NUTRIENT STRESS-INDUCED METABOLIC REPROGRAMMING SUPPORTING PANCREATIC CANCER AGGRESSIVENESS

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Pancreas

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ATM deficiency in pancreatic ductal adenocarcinoma drives metabolic reprogramming that enables adaptation to nutrient scarcity and promotes tumor cell survival and aggressiveness.

KEY POINTS

- Loss of ATM facilitates PDAC cell adaptation to nutrient scarcity by evading metabolic stress-induced apoptosis and correlates with increased ATP production
- ATM-deficient PDAC cells show higher vulnerability to inhibitors targeting oxidative phosphorylation, glycolysis, and pentose phosphate pathway, and rely on acetate metabolism channeled into fatty acid synthesis and TCA cycle under metabolic stress
- Loss of ATM promotes maintenance of aggressive PDAC features under nutrient starvation including mesenchymal phenotype and DNA damage accumulation
- In human PDAC, ATM expression negatively correlates with enzymes related to oxidative phosphorylation, glycolysis, and acetate metabolism
- The findings suggest genotype-specific metabolic vulnerabilities in homologous recombination deficient PDAC may be exploitable through targeted therapeutic interference strategies

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