ATP (adenosine triphosphate) and Cancer

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8517097/
Defueling the cancer: ATP synthase as an emerging target in cancer therapy 2021
Reprogramming of cellular metabolism is a hallmark of cancer. Mitochondrial ATP synthase (MAS) produces most of the ATP that drives the cell. High expression of the MAS-composing proteins is found during cancer and is linked to a poor prognosis in glioblastoma, ovarian cancer, prostate cancer, breast cancer, and clear cell renal cell carcinoma. Cell surface-expressed ATP synthase, translocated from mitochondrion to cell membrane, involves the angiogenesis, tumorigenesis, and metastasis of cancer. ATP synthase has therefore been considered a therapeutic target. We review recent various ATP synthase inhibitors that suppress tumor growth and are being tested for the clinic.

https://pubmed.ncbi.nlm.nih.gov/34121201/
ATP and cancer immunosurveillance 2021
While intracellular adenosine triphosphate (ATP) occupies a key position in the bioenergetic metabolism of all the cellular compartments that form the tumor microenvironment (TME), extracellular ATP operates as a potent signal transducer. The net effects of purinergic signaling on the biology of the TME depend not only on the specific receptors and cell types involved, but also on the activation status of cis- and trans-regulatory circuitries. As an additional layer of complexity, extracellular ATP is rapidly catabolized by ectonucleotidases, culminating in the accumulation of metabolites that mediate distinct biological effects.

https://pubmed.ncbi.nlm.nih.gov/35738682/
ATP and Adenosine Metabolism in Cancer: Exploitation for Therapeutic Gain 2022
Tumors create an adenosine-rich immunosuppressive microenvironment through the increased release of ATP from dying and stressed cells and its ectoenzymatic conversion into adenosine. Therefore, the adenosine pathway becomes an important therapeutic target to improve the effectiveness of immune therapies.

https://pubmed.ncbi.nlm.nih.gov/34722292/
High ATP Production Fuels Cancer Drug Resistance and Metastasis: Implications for Mitochondrial ATP Depletion Therapy 2021
Recently, we presented evidence that high mitochondrial ATP production is a new therapeutic target for cancer treatment. Using ATP as a biomarker, we isolated the "metabolically fittest" cancer cells from the total cell population. Importantly, ATP-high cancer cells were phenotypically the most aggressive, with enhanced stem-like properties, showing multi-drug resistance and an increased capacity for cell migration, invasion and spontaneous metastasis. In support of these observations, ATP-high cells demonstrated the up-regulation of both mitochondrial proteins and other protein biomarkers, specifically associated with stemness and metastasis. Therefore, we propose that the "energetically fittest" cancer cells would be better able to resist the selection pressure provided by i) a hostile micro-environment and/or ii) conventional chemotherapy, allowing them to be *naturally-selected* for survival, based on their high ATP content, ultimately driving tumor recurrence and distant metastasis. In accordance with this energetic hypothesis, ATP-high MDA-MB-231 breast cancer cells showed a dramatic increase in their ability to metastasize in a pre-clinical model *in vivo*. Conversely, metastasis was largely prevented by treatment with an FDA-approved drug (Bedaquiline), which binds to and inhibits the mitochondrial ATP-synthase, leading to ATP depletion. Clinically, these new therapeutic approaches could have important implications for preventing treatment failure and avoiding cancer cell dormancy, by employing ATP-depletion therapy, to target even the fittest cancer cells.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10762532/
Adenosine triphosphate induced cell death: Mechanisms and implications in cancer biology and therapy 2023
Adenosine triphosphate (ATP) induced cell death (AICD) is a critical cellular process that has garnered substantial scientific interest for its profound relevance to cancer biology and to therapeutic interventions.