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Abstract

Ninety percent of cancer deaths are the result of metastasis. The development of robust and sensitive technologies that capture metabolic processes in metastasizing cancer cells has hampered understanding of the role of metabolism during metastasis. We discuss the current technologies for studying I metabolism in primary and metastatic cancer cells, as well as metabolic interactions between cancer cells and the tumour microenvironment (TME) at various stages of the metastatic cascade. We discuss the benefts and drawbacks of each method, as well as how these tools and technologies will help us better understand metastasis [1-15]. Studies using cutting-edge metabolomic technologies to investigate the complex metabolic rewiring of di ferent cells have the potential to reveal novel biological processes and therapeutic interventions for human cancers

Metabolism is essential to all cellular functions because it ultimately drives the generation and utilisation of ATP required for cellular activity. Changes in metabolism can lead to a variety of diseases, including cancer. Cancer cells have the ability to activate and suppress various metabolic pathways. Furthermore, metabolic di erences found in cancer cells versus non-cancer cells suggest potential metabolic vulnerabilities that could be therapeutically modulated to slow cancer progression. There has been remarkable progress in understanding the metabolic regulation of cancer cells in recent years, particularly in the context of primary tumour metabolism. However, a thorough understanding of the metabolic regulation of cancer cells during metastasis remains an active area of investigation.

Metastasis is a multifaceted process that includes I uncontrolled proliferation of cancer cells at the primary site, local invasion into surrounding tissue, intravasation into the bloodstream or lymphatics, transportation through circulation, extravasation into distant tissues, and colonisation of secondary organs Because most cancer cells do not survive in these harsh environments, metastasis via this metastatic cascade is a highly ine f cient process. However, some cancer cells undergo metabolic adaptations to maximise survival in these harsh environments.

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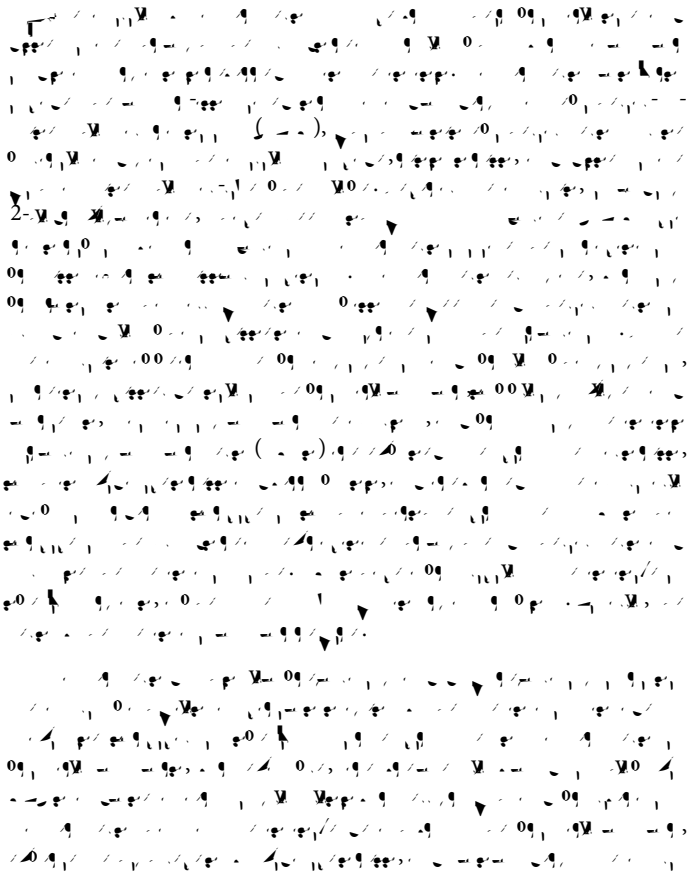

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References

1. Qin J, Li R, Raes J (2010) A human gut microbial gene catalogue established by metagenomic sequencingNature.464: 59-65.
2. Abubucker S, Segata N, Goll J (2012) Metabolic reconstruction for metagenomic data and its application to the human microbiome. PLoS Comput Biol 8.
3. Hosokawa T, Kikuchi Y, Nikoh N (2006) Strict host-symbiont cospeciation and reductive genome evolution in insect gut bacteria. PLoS Biol 4.
4. Canfora EE, Jocken JW, Black EE (2015) Short-chain fatty acids in control of body weight and insulin sensitivity. Nat Rev Endocrinal 11: 577-591.
5. Lynch SV, Pedersen (2016) The human intestinal microbiome in health and disease. N Engl J Med 375: 2369-2379.
6. Araújo A.P.C, Mesak C, Montalvão MF (2019) Anti-cancer drugs in aquatic environment can cause cancer insight about mutagenicity in tadpoles. Sci Total Environ. 650: 2284-2293.
7. Barros S, Coimbra AM, Alves N(2020) Chronic exposure to environmentally relevant levels osimvastatin disrupts zebrafsh brain gene signaling involved in energy metabolism. J Toxic Environ Health A 83: 113-125.
8. Ben I, Zvi S, Kivity, Langevitz P (2019) Hydroxychloroquine from malaria to autoimmunity.Clin Rev Allergy Immunol 42 (2): 145-153.
9. Bergqvist Y, Hed C, Funding L (1985) Determination of chloroquine and its metabolites in urine a feld method based on ion-pair. ExtractionBull World Health Organ 63 (5): 893.
10. Burkina V, Zlabek V, Zamarats G (2015)Efects of pharmaceuticals present in aquatic environment on Phase I metabolism in fsh.Environ Toxicol Pharmacol 40 (2): 430-444.
11. Cook JA, Randinitis EJ, Bramson CR (2006) Lack of a pharmacokinetic interaction between azithromycin and chloroquin. Am J Trop Med Hyg 74 (3): 407.
12. Davis SN, Wu P, Camci ED, Simon JA (2020) Chloroquine kills hair cells in zebrafsh lateral line and murine cochlear cultures implications for ototoxicity .Hear Res 395: 108019.
13. De JAD Leon C (2020) Evaluation of oxidative stress in biological samples using the thiobarbituric acid reactive substances assay. J Vis Exp (159): Article e61122.
14. Dubois M, Gilles MA, Hamilton J (1956) Colorimetric method for determination of sugars and related substances. Anal Chem 28 (3): 350-356.
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