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Cancer Cell Metabolism: A Potential Target for Cancer Therapy

Editors: Kumar, Dhruv (Ed.)

Reveals the molecular mechanism of metabolic regulation in cancer cells

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This book illustrates various aspects of cancer cell metabolism, including metabolic regulation in solid tumours vs. non-solid tumours, the molecular pathways involved in its metabolism, and the role of the tumour microenvironment in the regulation of cancer cell metabolism. It summarizes the complexity of cancer cell metabolism in terms of the switch from anaerobic to aerobic glycolysis and how mitochondrial damage promotes aerobic glycolysis in cancer cells. The reserachive chapters provide the latest information

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de Melto, R.A. (et al.) (Eds.) (2019)
- Tumor Cell Metabolism**
Mazurek, S. (et al.) (Eds.) (2015)
- Cancer Stem in Cancer Ther**
Pathak, S. (et al.)

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Role of c-Met/HGF Axis in Altered Cancer Metabolism

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Vaishali Chandel, Sibi Raj, Ramesh Choudhari,
and Dhruv Kumar

Abstract

c-Met (mesenchymal–epithelial transition factor) is a receptor tyrosine kinase that belongs to the Met family and is majorly expressed on the surfaces of epithelial cells. Hepatocyte growth factor (HGF) is the receptor specific to c-Met. HGF binding to c-Met leads to the initiation of series of cascade mediating wound healing and embryogenesis. However, in cancer cells, mutation in c-Met stimulates various downstream signalling pathways such as PI3K/AKT, Ras/MAPK, and JAK/STAT, causing aberrant c-Met/HGF axis activation and resulting in development and progression through migration, invasion, and metabolic reprogramming in cancer. c-Met/HGF axis modulates glucose metabolism in cancer by altering major enzymes and transporters such as hexokinase, phosphofructokinase, lactate dehydrogenase, and glucose transporters and shifts the reliance of cancer cells on glucose rather than oxidative phosphorylation even in the presence of oxygen (Warburg phenomena). In addition, c-Met/HGF axis modulates and interferes with other pathways such as pentose phosphate pathway, amino acid metabolism, and TCA cycle leading to its aggressive phenotypes. Therefore, understanding the association between c-Met/HGF axis and signalling pathways is critical and clinically important to develop

The original version of this chapter was revised with the correction received from the author. The correction to this chapter can be found at https://doi.org/10.1007/978-981-15-1991-8_12

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