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Determination of Glucocorticoids with Intercellular Microbes in Metastatic Cancer Cells

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INTRODUCTION

Metastatic spread of cancer is achieved by hematogenous spread of circulating tumour cells (CTCs). In general, however, the temporal dynamics that determine the formation of metastatic CTCs are poorly characterized and it is postulated that CTCs are continuously expelled from the growing tumour or as a result of mechanical shock. Here we observe a striking and unexpected pattern of CTC generation dynamics in both breast cancer patients and mouse models, highlighting that the most spontaneous her CTC invasion events occur during sleep. Moreover, we show that his CTCs in quiescent phase are highly susceptible to metastasis, whereas CTCs generated in active phase lack the ability to metastasize. Mechanistically, single-cell RNA-seq analysis of CTCs reveals marked up regulation of mitotic genes only during quiescence in both patient and mouse models, enabling metastatic potential. Systemically, key circadian hormones such as melatonin, testosterone and glucocorticoids determine the dynamics of CTC generation, resulting in insulin directly but time-dependently promoting tumour cell proliferation *in vivo*. Therefore, the spontaneous occurrence of highly metastatic-prone CTCs is concentrated in the dormant phase of affected individuals rather than occurring continuously, providing a new rationale for the timed question and treatment of metastatic cancers.

DESCRIPTION

Tumour-resident intracellular microbiota are novel tumor components that have been reported in various cancer types with unknown biological functions. Here, we investigated the functional importance of these intratumoral bacteria primarily using the mouse spontaneous breast tumour model MMTV-PyMT. We found that depletion of intra tumoral bacteria significantly reduced lung metastases without affecting primary tumour growth. During metastatic colonization, intratumoral bacteria carried by circulating tumour cells promoted host cell survival by increasing resistance to fluid shear stress through reorganization of the actin cytoskeleton. Furthermore, we showed that intratumoral administration of selected bacterial strains isolated from the resident tumour microbiota promoted metastasis in two mouse tumour models with significantly different metastatic potentials. Our results suggest that the resident tumour microbiota plays an important role in promoting cancer metastasis despite its low biomass. Cancer metastasis requires the transient activation of cellular programs, which enable dissemination and dissemination to distant organs. Genetic, transcriptional and translational heterogeneity contribute to this dynamic process. Metabolic heterogeneity has also been observed, but its role in cancer progression is poorly understood. Here, we find that loss of phosphoglycerate dehydrogenase (PHGDH) enhances metastatic spread. In particular, we find that heterogeneous or low PHGDH expression in primary tumours of breast cancer patients is associated with reduced metastasis-free survival.

CONCLUSION

Mechanistically, Phgdh interacts with the glycolytic enzyme phosphofructokinase, and loss of this interaction activates the hexosamine-sialic acid pathway, providing a precursor for protein glycosylation. The result is aberrant protein glycosylation, including increased sialylation of integrin αvβ, which enhances cell migration and invasion. Inhibition of sialylation weakens the metastatic potential of Phgdhlow cancer cells. In conclusion, PHGDH catalytic activity supports cancer cell proliferation, whereas low PHGDH protein expression non-catalytically enhances cancer spread and metastasis. Therefore, the presence of PHDGH heterogeneity in primary tumours can be taken as a sign of tumour aggressiveness.

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