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8th *meeting on medicinal biotechnology*

BOOK OF ABSTRACTS

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BOOK OF ABSTRACTS OF THE 8TH MEETING ON MEDICINAL BIOTECHNOLOGY

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Betulinic Acid Modulates Adipocyte Differentiation and Secretome Activity: Downstream Effects on Glioblastoma Cell Migration

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Betulinic acid (BA) has been recognized for its potential to modulate tumor progression and adipocyte function, yet its effects on adipocyte secretome remodeling and the downstream consequences for glioblastoma behavior remain incompletely understood¹⁻⁴. In this study, we investigated the influence of BA on adipocyte secretory activity and its functional impact on glioblastoma cell migration. Direct exposure of U251 glioblastoma cells to BA significantly reduced cell migration, confirming its intrinsic anti-migratory potential. To further explore microenvironmental interactions, adipocyte-conditioned media (ACM) derived from BA-treated adipocytes was evaluated for its effects on glioblastoma behavior. Secretome analysis demonstrated that BA altered matrix metalloproteinase (MMP) expression and reduced gelatinolytic activity, indicating significant modulation of extracellular matrix-related proteolytic pathways. Functional assessment using wound healing assays revealed that ACM from BA-treated adipocytes did not consistently suppress glioblastoma migration. Intermediate BA concentrations were associated with increased migra-

tory behavior relative to basal conditions, although differences compared with control ACM were not statistically significant. Additionally, transwell migration assays did not demonstrate significant differences between treatment conditions, suggesting that BA-induced alterations in adipocyte secretome composition do not uniformly translate into measurable changes in glioblastoma migratory capacity across different experimental models. These findings indicate that modulation of gelatinolytic pathways alone is insufficient to predict glioblastoma cell migration and support the contribution of additional adipocyte-derived signalling mechanisms in regulating tumor behavior. Overall, BA induces substantial remodelling of adipocyte secretory function, producing complex and non-linear downstream effects on glioblastoma cells. This study highlights the importance of tumor micro-environment crosstalk and underscores the need to evaluate multiple regulatory pathways when considering the therapeutic potential of BA and related bioactive compounds.

KEYWORDS: *betulinic acid, obesity, glioblastoma multiforme*

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