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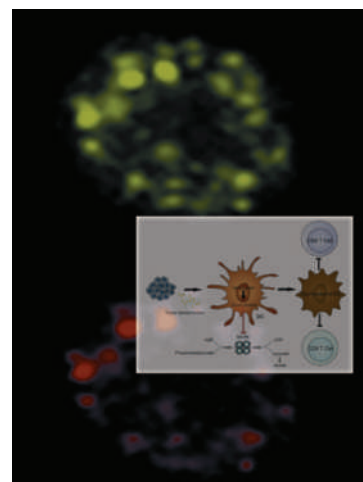
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ABOUT THE COVER

Tumor microenvironment conditions, such as hypoxia, have emerged as important components contributing to dendritic cell dysfunction. However, little is known about the molecular mechanism(s) behind this dysfunction. Previous studies by Zhang and colleagues show that tumor-derived factors may upregulate SOCS3 expression. Interestingly, immunoprecipitation combined with MALDI-TOF/TOF-MS/MS and pull-down analysis reveals the interaction of SOCS3 with M2-PK, as examined by confocal fluorescence microscopy. Importantly, M2-PK activity can be downregulated by SOCS3. Since M2-PK plays a critical role in producing ATP through glycolysis, this may represent a novel mechanism for dendritic cell dysfunction in the tumor microenvironment. For details, see the article by Zhang and colleagues on page 89 of this issue.



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