Preface

Alberto Mantovani

Published online: 16 April 2010
© Springer Science+Business Media, LLC 2010

Chronic non-resolving inflammation contributes to cancer development as originally formulated in the nineteenth century [1, 2]. This hypothesis has now been supported by experimental and clinical studies and lead to a now a generally accepted paradigm. Even in tumors which are not epidemiologically linked to inflammation, an inflammatory component is an essential element of the tumor micro environment and is also present in tumors which are not epidemiologically linked to inflammation. A leukocyte infiltrate and soluble inflammatory mediators such as cytokines, and chemokines are elements which contribute to cancer-related inflammation. Conditions predisposing to cancer (e.g., ulcerative colitis for colitis-associate cancer) or genetic events which underlie neoplastic transformation orchestrate the build-up of an inflammatory microenvironment. Therefore, an intrinsic pathway driven by oncogenes and an extrinsic pathway driven by chronic inflammatory conditions contribute to cancer-related inflammation which has been suggested to represent the seventh hallmark of cancer [3].

This issue of Cancer Metastasis Reviews is focused on the role of inflammatory cells and mediators in tumor invasion and metastasis. Mediators of inflammation have long been known to increase metastatic dissemination [4, 5]. It is now apparent that inflammatory cells and their mediators contribute to local invasion and dissemination and orchestrate

the construction of a metastatic ecological niche at distant anatomical sites. Moreover, inflammatory mediators are key elements in the involvement of specific organs and tissues such as the bone and nerves by cancer [6]. Hopefully this issue of Cancer Metastasis Reviews, by providing critical reviews of selected aspects of the inflammation-metastasis connection, will represent a useful tool to scholars within and outside the field.

References

1. Balkwill, F., & Mantovani, A. (2001). Inflammation and cancer: back to Virchow? *Lancet*, 357, 539–545.
2. Coussens, L. M., & Werb, Z. (2002). Inflammation and cancer. *Nature*, 420, 860–867.
3. Mantovani, A., Allavena, P., Sica, A., & Balkwill, F. (2008). Cancer-related inflammation. *Nature*, 454, 436–444.
4. Balkwill, F. (2009). Tumour necrosis factor and cancer. *Nature Reviews. Cancer*, 9, 361–371.
5. Giavazzi, R., Garofalo, A., Bani, M. R., Abbate, M., Ghezzi, P., & Boraschi, D. (1990). Interleukin 1-induced augmentation of experimental metastases from a human melanoma in nude mice. *Cancer Research*, 50, 4771–4775.
6. Marchesi, F., Piemonti, L., Mantovani, A., & Allavena, P. (2010). Molecular mechanisms of perineural invasion, a forgotten pathway of dissemination and metastasis. *Cytokine & Growth Factor Reviews*, 21, 77–82.

A. Mantovani (✉)
Istituto Clinico Humanitas, IRCCS and University of Milan,
Milan, Italy
e-mail: alberto.mantovani@humanitasresearch.it