



Mediators of Inflammation

Special Issue on **A Disintegrin and Metalloproteinases as Key Regulators of Cancer and Inflammation**

CALL FOR PAPERS

Many cytokines, growth factors, and their receptors exist as both membrane type I and II integral membrane proteins, as well as soluble forms. However, many times, biological processes, such as cancer and inflammation, are driven by overproduction of the soluble form of these proteins. The posttranscriptional release of type I and II membrane proteins from the cell surface is a process known as ectodomain shedding and is carried out by enzymes of the a disintegrin and metalloproteinase (ADAM) family.

ADAM family members are important for normal processes in embryogenesis such as neuronal and cardiac development, adipogenesis, and myogenesis. It is their dysregulation that has been linked to cancer, inflammation, and neurological diseases such as Alzheimer's disease. The metalloproteinases are also considered to be biomarkers for many of the same diseases for which they are aberrantly expressed. A better understanding of the roles that ADAM members play in disease processes could help lead to the development of innovative therapeutic agents that target tumorigenesis and inflammatory processes. Furthermore, determination of the levels of different ADAM metalloproteinases in biological fluids during normal and disease states could lead to novel diagnostic tests.

Potential topics include, but are not limited to:

- ▶ Recent discoveries of the role of ADAM family members in tumorigenesis and inflammatory processes
- ▶ Expression of ADAM family members in tissues during disease processes of cancer and inflammation
- ▶ Quantification of activity and protein levels of ADAMs in biological fluids and tissues and their correlation with cancer and inflammation
- ▶ Regulation of ADAM family members at the transcriptional and/or posttranscriptional level
- ▶ Anticancer and anti-inflammatory therapeutic agents that are under development that target ADAMs
- ▶ ADAMs and their role in normal tissue homeostasis

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