INSTRUCTIONS FOR ABSTRACTS

DEADLINE FOR ABSTRACT SUBMISSION IS SEPTEMBER 9, 2022

In order to facilitate the formatting and printing of the final program, the Organizing Committee strongly encourage you to follow the instructions provided below:

Title : **Times New Roman 14 pts**
Bold Capitals,

Authors : **Times New Roman 12pts**
First and last names in full, with affiliations indicated with superscript numbers,

Affiliations : **Times New Roman 12 pts**
Laboratory/Department, Institution, City, State, Country

Body of the abstract : **Times New Roman 12pts**

Word limit : 400

See below an example of abstract from a previous workshop in the recommended format.

CCN1 IN INTESTINAL MUCOSAL HEALING

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Crohn's disease (CD) and ulcerative colitis (UC), two major subtypes of inflammatory bowel disease (IBD), are chronic inflammatory disorders of poorly defined etiology. Traditional therapies have focused on amelioration of inflammation, although recent studies have indicated that mucosal healing is an important prognostic endpoint. The integrin-binding matricellular protein CCN1 (CYR61) is upregulated in human patients with CD and UC, and is emerging as a key injury response molecule that coordinates multiple aspects of wound healing and tissue repair in colitis. Knockin mice expressing integrin-binding defective CCN1 suffer exacerbated morbidity and mortality in experimental colitis, showing impaired epithelial regeneration, neutrophil persistence, and elevated fibrosis. Furthermore, treatment of wild-type or *Ccn1* mutant mice with purified CCN1 protein significantly accelerates mucosal healing from colitis, suggesting a therapeutic potential of CCN1 for IBD. Here we will discuss the role of CCN1 in mucosal healing and intestinal injury repair following experimental colitis.