Expression of angiogenic and inflammation markers in murine schistosomiasis mansoni

Dtemaei A.1, Fernandes R.1,2, Soares R.2,3, Alves H.4,5, Richter J.6, Botelho M.C.2,4

1 Ciências Químicas e Biomoléculas, Escola Superior de Tecnologia da Saúde do Porto, Instituto Politécnico do Porto, Portugal, 2 I3S, Instituto de Investigação e Inovação da Universidade do Porto, Portugal, 3 Department of Biochemistry, Faculty of Medicine, University of Porto, Porto, Portugal, 4 INSA, National Institute of Health Dr. Ricardo Jorge, Department of Health Promotion and Chronic Diseases, Porto, Portugal, 5 Fundação Professor Ernesto Morais, Porto, Portugal, 6 Institute of Tropical Medicine and International Health, Charité – Universitätsmedizin Berlin, Germany

AIM
To study angiogenesis in the livers of mice infected with *S. mansoni*.

BACKGROUND
• Schistosomiasis is a neglected tropical disease, endemic in 76 countries, that afflicts more than 240 million people. Symmers’ portal fibrosis (also called periportal fibrosis) is a characteristic hepatic disease described in schistosomiasis.
• Although estimates are not available, Schistosomiasis must still be considered to be the most frequent cause of liver fibrosis worldwide.
• Angiogenesis, the formation of new blood vessels from pre-existing ones, is recognized as a key event in a basic change occurring during repair by granulation tissue. This process seems to precede fibrosis formation in most types of chronic liver disease (Fig. 2).

METHODOLOGICAL STRATEGY
By immunohistochemical staining using IL-6 as inflammation marker, Von Willebrand (CD31) as endothelial marker, Microvessel Density (MVD) as angiogenesis marker and LYVE-1 as lymphangiogenesis marker in the livers of normal control mice and *S. mansoni* infected mice.

RESULTS
*S. mansoni* infection increases inflammation (IL-6) in liver

![Non-infected Mice vs Infected Mice](image1)

*S. mansoni* infection increases MVD in liver

![Non-infected Mice vs Infected Mice](image2)

*S. mansoni* infection increases expression LYVE-1

![Non-infected Mice vs Infected Mice](image3)

CONCLUSIONS
• *S. mansoni* infection increases inflammation, angiogenesis and lymphangiogenesis in the liver.
• Thus, blocking blocking IL-6 and/or lymph/angiogenesis may represent the appropriate therapeutic target for the treatment of schistosomal liver fibrosis.