Positions are available for both **wet-lab** and **computational postdocs** and **PhD students** to study transcriptional mechanisms controlling **normal and pathological inflammation**.

Tissue responses to microbial and endogenous danger signals involve the inducible expression of hundreds of inflammatory genes. How enhancers and promoters controlling inflammatory gene expression are coordinately bound by lineage-determining and stimulus-activated TFs has been extensively characterized. However, we still have a very incomplete knowledge of the necessary next step in the process, namely how distinct combinations of DNA-bound TFs regulate recruitment and function of the co-regulators and machineries that control the inducible expression of inflammatory genes.

In the context of a five-year EC-funded project starting at Humanitas University in Milan in November 2016, we will integrate **genomics** and **computational approaches** with **genetic screens**, **biochemistry**, and **mouse genetics** in order to obtain a detailed mechanistic understanding of the information flow linking genomic regulatory elements to inflammatory gene expression.

Highly motivated scientist with a strong interest in transcriptional regulation and epigenetics are encouraged to apply.

CV, list of publications and contact information for referees should be sent to gioacchino.natoli@ieo.eu

**Recent publications from the lab**

- Molecular control of macrophage activation and priming (C.K. Glass and G. Natoli) *Nature Immunology* 17, 26-33 (2016)
- A dual cis-regulatory code links IRF8 to constitutive and inducible gene expression in macrophages (A. Mancino ... G. Natoli) *Genes & Development* 29, 394-408 (2015).
- The histone methyltransferase Mll4 controls macrophage function through glycosylphosphatidylinositol anchor synthesis (L. Austenaa ... G. Natoli) *Immunity* 36, 572-585 (2012).