Congress venue

Roma Eventi Fontana di Trevi - Piazza della Pilotta - Roma
Situated in the heart of Rome, a short walk from The Quirinale and the Fontana di Trevi (Trevi Fountain), from which it gets its name, the Roma Eventi - Fontana di Trevi Conference Centre is the ideal venue for any event. Situated in the splendid Neoclassical palace chosen by Pope Benedict XV as the location for the Gregorian University and built by Guido Barluzzi in 1930, the Conference Centre extends over 1500 sqm and consists of 15 meeting rooms able to accommodate over 1000 participants.
Versatility, high quality services, international level standards and great professionalism all make the "ROMA EVENTI - FONTANA DI TREVI" the ideal location for Conferences, Seminars and Exhibitions.

How to reach the Venue

http://www.roma-eventi.com/eng/fontana-di-trevi/business-centre-italy.html

FROM 'ROME TERMINI STATION'

Duration of the Journey: 5 mins (public transport) - 20 mins (on foot)
Distance: 2 km.

FROM THE 'LEONARDO DA VINCI"' INTERNATIONAL AIRPORT

Duration of the Journey: 40-45 mins (public transport or private car)
Distance: 28 km.

Provider & Organizing Secretariat

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EWOG-MDS

EWOG-SAA

EWOG 2017

ROMA EVENTI FONTANA DI TREVI
Roma

28•30SEPTEMBER**2017**

FIRST ANNOUNCEMENT

Prof. Franco Locatelli, President of the meeting

MAIN TOPICS

1. Advances in diagnosis of MDS

a. Does morphology still have a role in the diagnosis of MDS?
b. The role of flow-cytometry in the diagnosis of MDS
c. Why I support WHO Classification of childhood MDS
d. Why I think WHO Classification is still imperfect for childhood MDS
e. What the WHO Classification of adult MDS can teach to pediatricians

2. Cytogenetic/Molecular diagnosis and biology of MDS

a. What is new in cytogenetics
b. What NGS can offer in the diagnosis of childhood MDS
c. How I can avoid misdiagnosis of inherited BMF syndrome
d. The role of microenvironment in MDS

3. Myeloid malignancies predisposition syndromes/genes

a. Overview on familial MDS
b. Chromosomal aberrations predicting clonal evolution in Fanconi Anemia
c. Games of clones in congenital neutropenia
d. GATA-2 and more in childhood MDS….

4. Aplastic Anemia

a. Somatic mutation in SAA
b. Aplastic anemia and clonal evolution: risk factors
c. Apoptosis and stem cells
d. EPN and aplastic anemia: friend or foe?
e. Telomeropathies

5. Biology of JMML

a. RASopathies
b. The genomic landscape of JMML
c. Epigenetic subclasses in JMML: the development
d. Epigenetic subclasses in JMML: clinical implications
e. microRNA and JMML
f. PDX model in JMML: a strategy for improving both comprehension of pathophysiology and treatment?

6. Treatment

a. 5-Azacitidine for treatment of patients with MDS and JMML
b. How I prevent and treat infections in MDS
c. Eltrombopag for low-risk MDS and SAA
d. Do we have to tailor HSCT strategy on the basis of genetic lesions in JMML?
e. How I Treat MDS and AML in Fanconi Anemia
f. How I diagnose and treat therapy-related AML
g. CAR T Cell Immunotherapy for Myeloid Malignancies