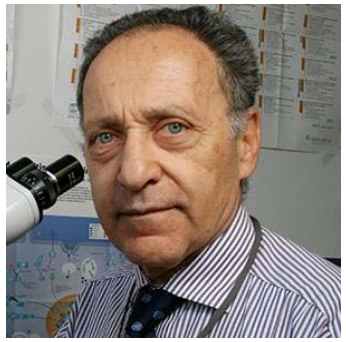
Professor Lucio Luzzatto



Honorary Degrees
Laurea ad Honorem in Pharmacy, University of Urbino, Italy
DSc University of Ibadan, Nigeria
Doctor *honoris causa* in Medicine, University of Patras, Greece

Honours and Awards
Fullbright Senior Fellow
Henry M. Stratton Lecture
William Dameshek Medal
Ham Wasserman Lecture
Medaglia Pio XI
Medaglia Josè Carreras
Membra dell'Accademia Americana delle Arti e Scienze
Membro dell'Accademia dei Lincei

MD graduation	University of Genova, Genova, Italy
Specialty in Hematology	University of Pavia, Pavia, Italy
Lib Doc in Biochemistry	Ministry of Education, Rome, Italy
FRCPath	Royal College of Pathologists, UK
FRCP	Royal College of Physicians, UK
-Lecturer and then Professor of Hematology, Univeristy of Ibadan	Ibadan, Nigeria
-Director, International Insitute of Genetics and Biophysic, NRC	Naples, Italy
-Professor of Hematology, Director of the Hematology dpt Royal Postgraduate Medical School, Consultant hematologist, Hammersmith Hospital	London, UK
-Chairmen, Dept of Human Genetics, and attending Physician Memorial Sloan Kettering Cancer Center	New York, USA
-Scientific Director, National Insitute for Cancer Research	Genova, Italy
-Professor of Hematology, Genova University	Genova, Italy
-Scientific Director, Istituto Toscano Tumori	Florence, Italy
-Professor of Hematology, University of Florence	Florence, Italy
-Professor of Hematology, Muhimbili University	Dar-es-Salam, Tanzania

Genetics and Clinic of G6PD deficiency

First demonstration that G6PD A- is structurally different from G6PD A

Cloning of the G6PD gene

ESCs defective for G6PD have normal pentose synthesis but are sensitive to oxydative stress

G6PD inactivation is lethal early in embryo development

Solving of the 3D structure of tetrameric human G6PD

G6PD deficiency and protection from Malaria

Clinical relationship between G6PD deficiency and sickle cell disease (mouse model of SCA)

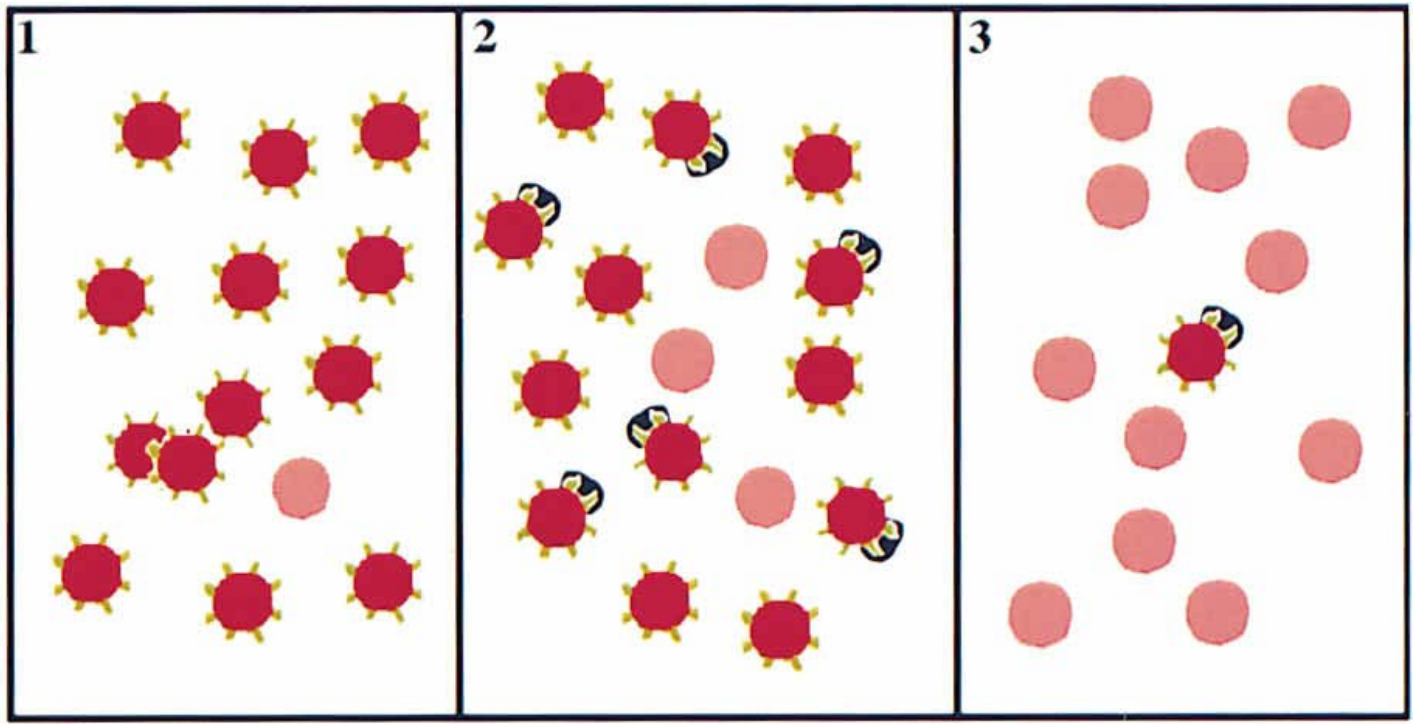
PNH pathogenesis and treatment

Demonstration that PNH is a clonal disorder

Conditional clonal selection

Eculizumab: the first PNH-specific treatment

A model for the pathogenesis of PNH: «the escape theory»



1.A normal bone marrow where an occasional stem cell with a PIG-A mutation exists but does not expand.

2.Normal stem cells are suffering a damage, while cells lacking GPI-linked molecules (PNH cells) escape from damage and may expand.

3.As the result of selection, the majority of stem cells (and their progeny) are PNH cells.

Adapted from: Luzzatto L, Bessler M and Rotoli B. Somatic mutations in paroxysmal nocturnal hemoglobinuria: a blessing in disguise. Cell 1997;88,1-4.