
Recent Advances in Molecular Genetics of the Maghreb and the Middle East Populations: The First Middle East Genetics Association of America (MEGA) Conference

Dahmani M. Fathallah,¹ Koussay Dellagi,¹ Mazin B. Qumsiyeh,² and Ahmad S. Teebi^{3*}

¹Institut Pasteur de Tunis, Tunis, Tunisia

²Duke University, Durham, North Carolina

³McGill University, Montreal, Quebec, Canada

The conference, which was cosponsored by The Pasteur Institute of Tunis was held recently at the Hammamet Resort in Tunisia from December 16–19, 1997. Topics discussed included gene mapping, gene expression and regulation, the molecular etiology of disease with emphasis on immunogenetic and infectious diseases, clinical genetics, clinical and molecular cytogenetics, fetal medicine, and preimplantation diagnosis.

A. Teebi (McGill University, Montreal Quebec, Canada), in his opening lecture, provided an overview of the genetic and malformation syndromes among the population of the Maghreb and the Arabs in general. He pointed to the overall high frequency of autosomal recessive disorders and new syndromes and emphasized the genetic diversity of these populations characteristically having high rates of inbreeding with visible genetic isolates together with large family size.

M. Ben Hamida (Institute of Neurology, Tunis, Tunisia) discussed the clinical molecular genetic aspects of neurological disorders in the Maghreb with emphasis on the autosomal recessive variants.

C. Kozma (Georgetown University, Washington, D.C.) discussed the psychosocial impact of genetic discoveries world-wide with special reference to populations in the area.

S. Deeb (University of Washington, Seattle, Washington), through his presentation on the genetics of familial hyperlipidemia, discussed how the approach involving the refinement of complex phenotypes can lead to identification of component traits and how the use of large families can be very helpful.

V. Der Kaloustian (McGill University, Montreal, Quebec, Canada) reviewed the recent advances in the genetics of skin disease with emphasis on genodermatosis in the Middle Eastern populations.

M. Qumsiyeh (Duke University, Durham, North Carolina) through his presentation on the organization

and the management of a cytogenetic laboratory in the 21st century suggested that the discipline will undergo a revolution as investigators learn how to integrate work with data imported from the Human Genome Project.

F. Tekaïa (Institut Pasteur, Paris, France) discussed the impact of DNA computing in the management of genetic data.

C. Petit and S. Abdelhak (Institut Pasteur, Paris, France) spoke on human hereditary deafness and reported the isolation of 3 genes, including *KAL* in the X-linked Kallmann syndrome, *MYO7A* underlying Usher 1B syndrome, and the isolated form of deafness, *DFNB2* and *EYA1*, responsible for branchio-oto-renal syndrome (the human homologue of the drosophila *eyes absent* gene).

J. Chelly (Inserm, Paris, France) reported on three other identified genes involved in the X-linked syndromic and mental retardation (MR). One of those genes encoded a protein involved in vesicular transport found defective in two families with X-linked MR, the second located on Xq12 encodes a protein belonging to the rho family of GTPase-activating proteins, and the third encodes a protein required for neuronal migration and is involved in X-linked subcortical laminar heteropia and lissencephaly syndrome.

D. Fathallah (Institut Pasteur, Tunis, Tunisia) reported on the molecular basis of leukocyte adhesion deficiency, an inherited immune deficiency, in a group of Maghrebian patients.

E. Skamene (McGill University, Montreal, Quebec, Canada) highlighted how genetic analysis of mouse models of infectious diseases had augmented the identification of candidate genes implicated in the susceptibility to human infectious diseases. The *Nramp 1* (*Bcg/Ity/Lsh*) gene was identified by positional cloning in a mouse model of resistance to mycobacterium infection.

K. Dellagi (Institut Pasteur, Tunis, Tunisia) reported on the complex interplay between genes and environment in human Leishmaniasis. A weak association with major histocompatibility complex (MHC) Class II (DRB) and Class III (TNF α and HSP70) poly-

*Correspondence to: Dr. Ahmad S. Teebi, Division of Medical Genetics, Montreal Children's Hospital, 2300 Tupper Street, Montreal, Quebec, Canada H3H 1P3.

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morphism in Tunisian children who developed visceral Leishmaniasis was observed.

C. Peacock (University of Cambridge, Cambridge, United Kingdom) reported similar results in Brazilian patients. They found no evidence for linkage or allelic association between *Nramp 1* or gene clusters on 5q23-q22 and susceptibility to visceral Leishmaniasis.

D. Capra (Oklahoma University, Stillwater, Oklahoma) in his talk showed how somatic hypermutation introduces insertions and deletions into immunoglobulin V genes.

R. Geha (Harvard University, Boston, Massachusetts) spoke about the importance of CD40/CD40 ligand interactions in immunoglobulin isotype switching.

A. Arnaout (Harvard University, Boston, Massachusetts) discussed the importance of leukocyte endothelial interactions in mounting an effective immune response.

G. Hauptman (Hôpital Pitié-Salpêtrière, Paris, France) reported on a novel family of major histocompatibility complex genes termed MIC. Of interest was the association of the MICA allele with Behçet disease, a prevalent pathology in North Africa.

M. Ben Khalifa (Mérieux Foundation, Lyon, France), in his lecture on the application of fluorescent in situ hybridization and polymerase chain reaction in preim-

plantation diagnosis, talked about the problems encountered in the diagnosis of chromosomal abnormalities and monogenic disorders and presented some new technical approaches to overcome false mosaicism interpretations and polymerase chain reaction pitfalls.

Among other interesting presentations: the localization of open angle glaucoma gene on 1q23-q25 (A. Belmouden, Paris, France), and giant axonal neuropathy (F. Hentati, Tunis, Tunisia), the identification of the marenostin (pyrin) gene responsible for familial Mediterranean fever (N. Samaoui, Paris, France), genetic homogeneity of osteopetrosis and carbonic anhydrase II deficiency in Arabs (M. Bejaoui, Tunis, Tunisia), the description of bilateral rhinoschisis and cranial defects as a new syndrome in a mother and son (J. Maatouk, Saudi Arabia), the expression of *H-19* gene in urinary bladder carcinoma without prognostic value (B. Abu-Libdeh, Jerusalem, Israel), the report that the *CLN3* gene, defective in the juvenile form of Batten disease, defines a novel antiapoptotic pathway operative in neurodegeneration and mediated via ceramid (R.M. Boustany, Duke University, Durham, North Carolina), and the molecular basis of Bare lymphocytes syndrome in Tunisian patients (R. Barbouche, Tunis, Tunisia). The abstracts were published in *Archive de L'institut Pasteur, Tunis, LXXIV (3/4), 1997.*