La storia dell' Immunogenetica in Italia

Antonio Amoroso
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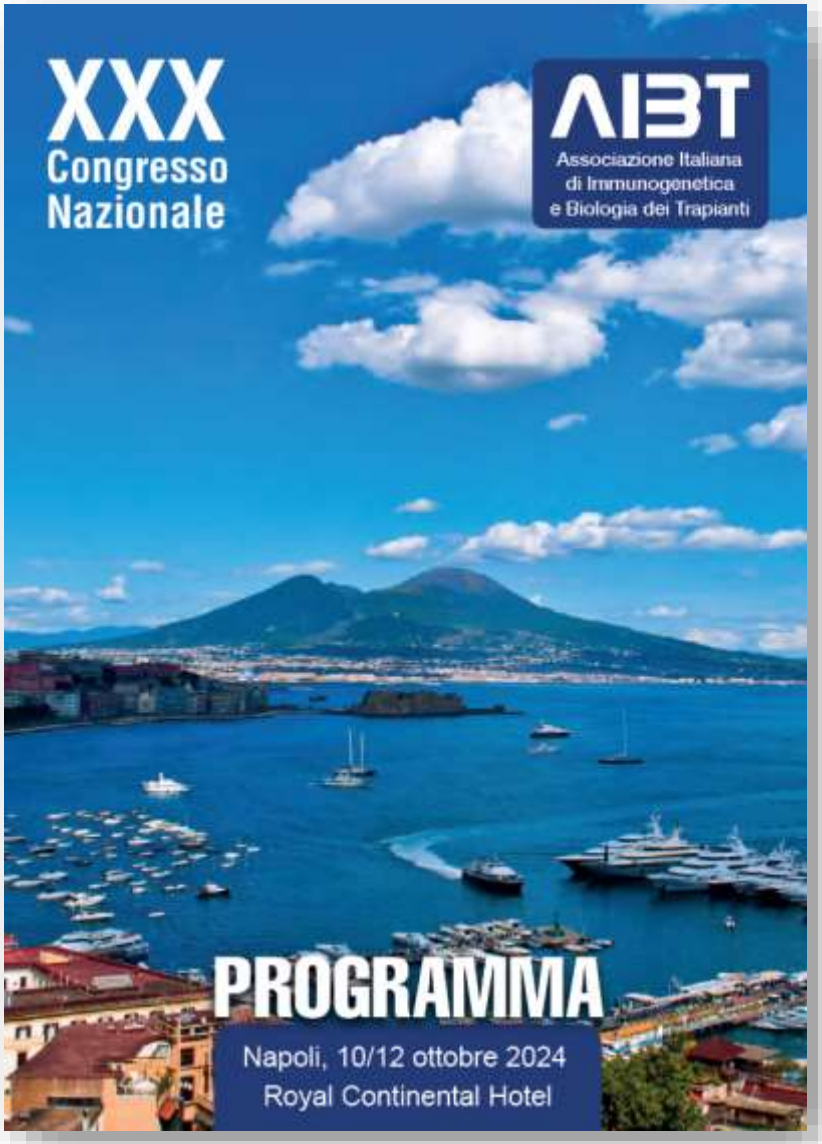
UNIVERSITÀ DI TORINO



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periodo:

1. antico
2. moderno
3. contemporaneo
4. futuro

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Storia antica dell'HLA: I primi 40 anni

Peter A. Gorer

1936, il primo Ag responsabile di rigetto

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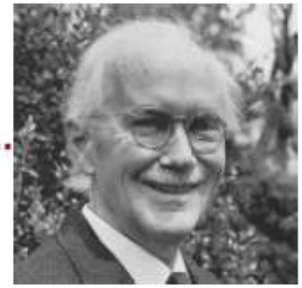

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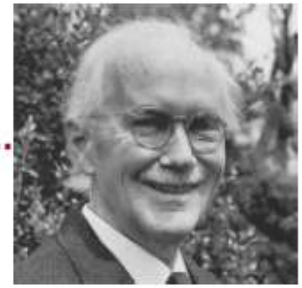

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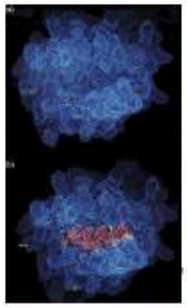

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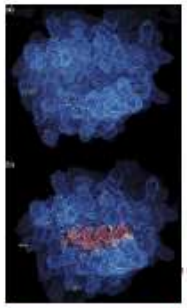

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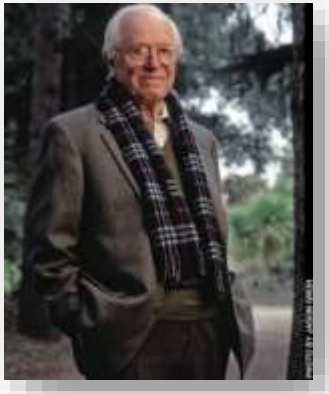
Peter Doherty, Rolf Zinkernagel

Ruggero Ceppellini

Adriano Buzzati-Traverso

Claudio Barigozzi

Giuseppe Montalenti



Luigi Cavalli-Sforza

Pionieri della moderna genetica

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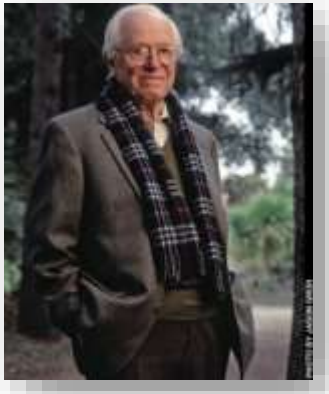
Marco Fraccaro



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Gennarino Sansone

Enrico Gandini

Giuseppe Montalenti

Antonio Cao

Lucio Luzzato

Edoardo Boncinelli

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Ruggero Ceppellini

Angelo Carbonara

Patricia Mőmigliano

Guido Scudeller

Antonio Amoroso

Fabio Malavasi

Sergio Curtoni

Alberto Piazza

Pierluigi Mattiuz

Mario DeMarchi

Nicola Migone

Mariaclara Cuccia

Silvia Deaglio

Roberto Tosi

Vincenzo Miggiano

Pionieri dell'immunogenetica

Massimo Trucco

Giovanni Galfré

Giorgio Trinchieri

Tommaso (Tommy) Meo

Domenico (Meco) Bernoco

Nature Vol. 266 7 April 1977

Antibodies to major histocompatibility antigens produced by hybrid cell lines

FUSION between myeloma cells and spleen cells from immunised donors has been shown to be a successful method of deriving homogeneous anti-SRBC (anti-sheep red blood cell) and anti-TNP antibodies[1,2]. One of the most powerful features of this approach is that, by cloning, one may easily derive cell lines synthesising monoclonal antibodies despite using non-purified immunogens. The multiple components of a heterogeneous population of hybrid cells are resolved by cloning techniques. This feature makes the system a very powerful tool in the study of complex antigenic structures. The established cell lines offer the further advantage of unlimited permanent supply of material, and the possibility of worldwide standardisation.

We report here the derivation of lines of hybrid cells secreting antibody to rat major histocompatibility antigens. The hybrids were made by fusion of a mouse myeloma cell line[3], P3-X63-Ag8, with spleen cells of AO strain rats immunised against DA strain antigens. In previous experiments we used Sendai virus as a fusing agent. In the experiments described here we used polyethylene glycol (PEG). Fusion of animal cells with PEG is becoming a method of choice. Various protocols have been described in detail, usually for cells growing as monolayers[4–7]. We adopted some of

producing antibody against a rat transplantation antigen brings the goal of producing standard permanent supplies of monoclonal antibodies for human tissue typing and other clinical uses one step nearer.

G.W.B. is the holder of a MRC Research Studentship; G.G. was the holder of a Hoffman-La Roche Fellowship.

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Received 14 January; accepted 9 February 1977

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Serafino Zappacosta

Valerio Misefari

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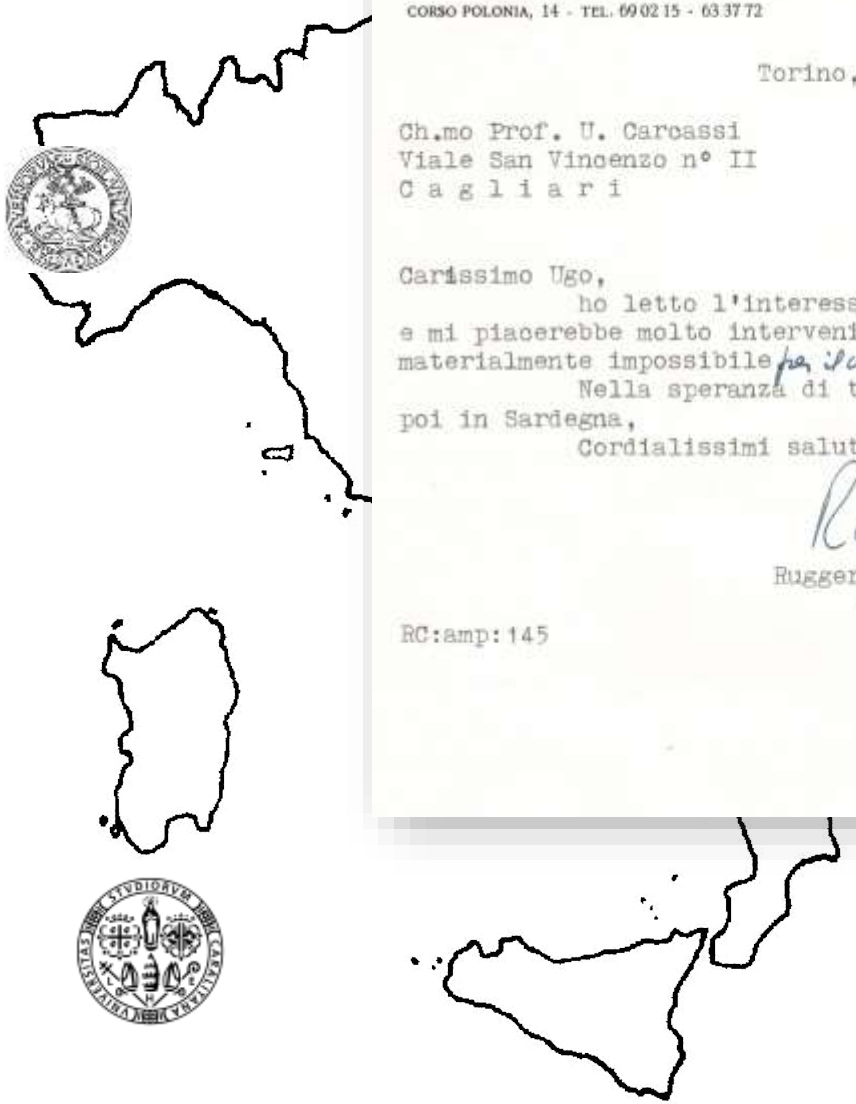
Ugo Carcassi



ISTITUTO DI GENETICA MEDICA
DELL'UNIVERSITÀ DI TORINO
CORSO POLONIA, 14 - TEL. 69 02 15 - 63 37 72

Torino, 12 Marzo 1965

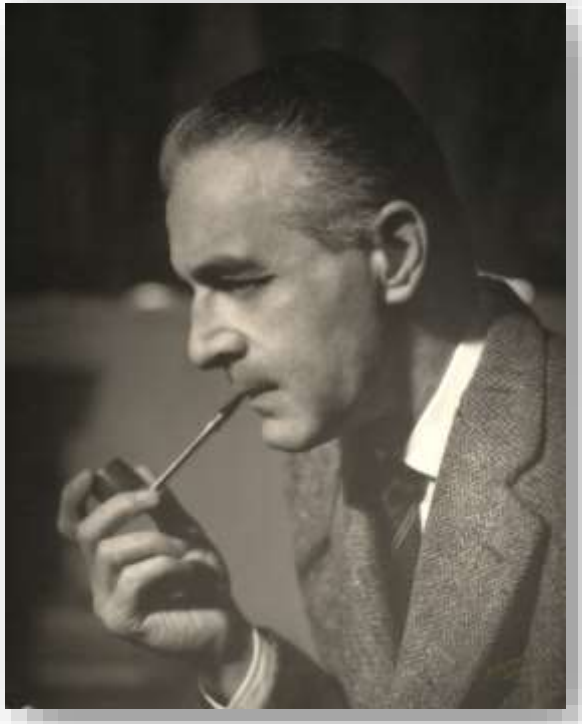
Ch.mo Prof. U. Carcassi
Viale San Vincenzo n° II
C a g l i a r i

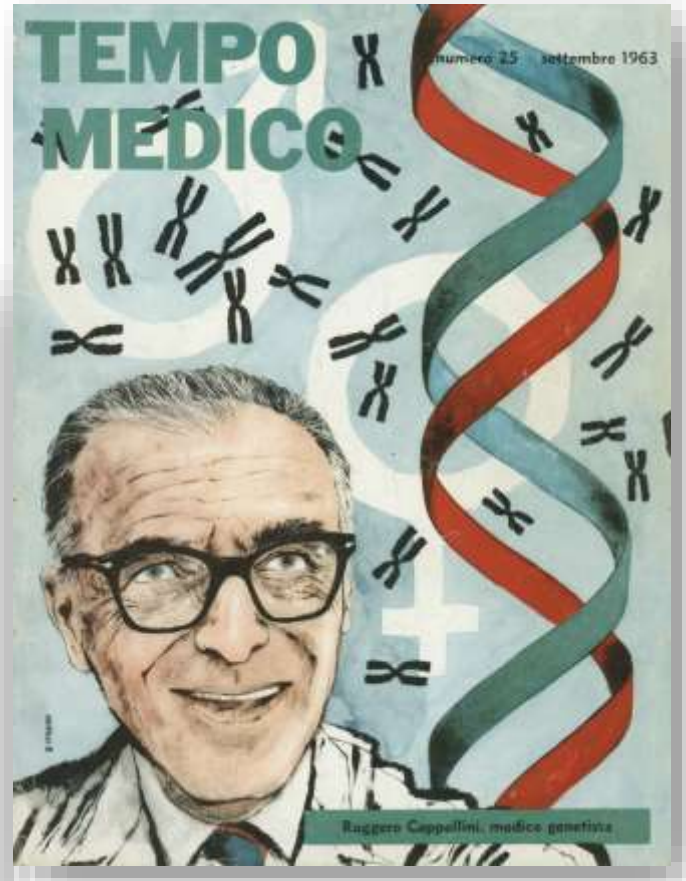
Carissimo Ugo,
ho letto l'interessante programma e mi piacerebbe molto intervenire ma mi è materialmente impossibile per il continuo accumularsi di impegni dopo la mia guarigione.
Nella speranza di tornare prima o poi in Sardegna,
Cordialissimi saluti,

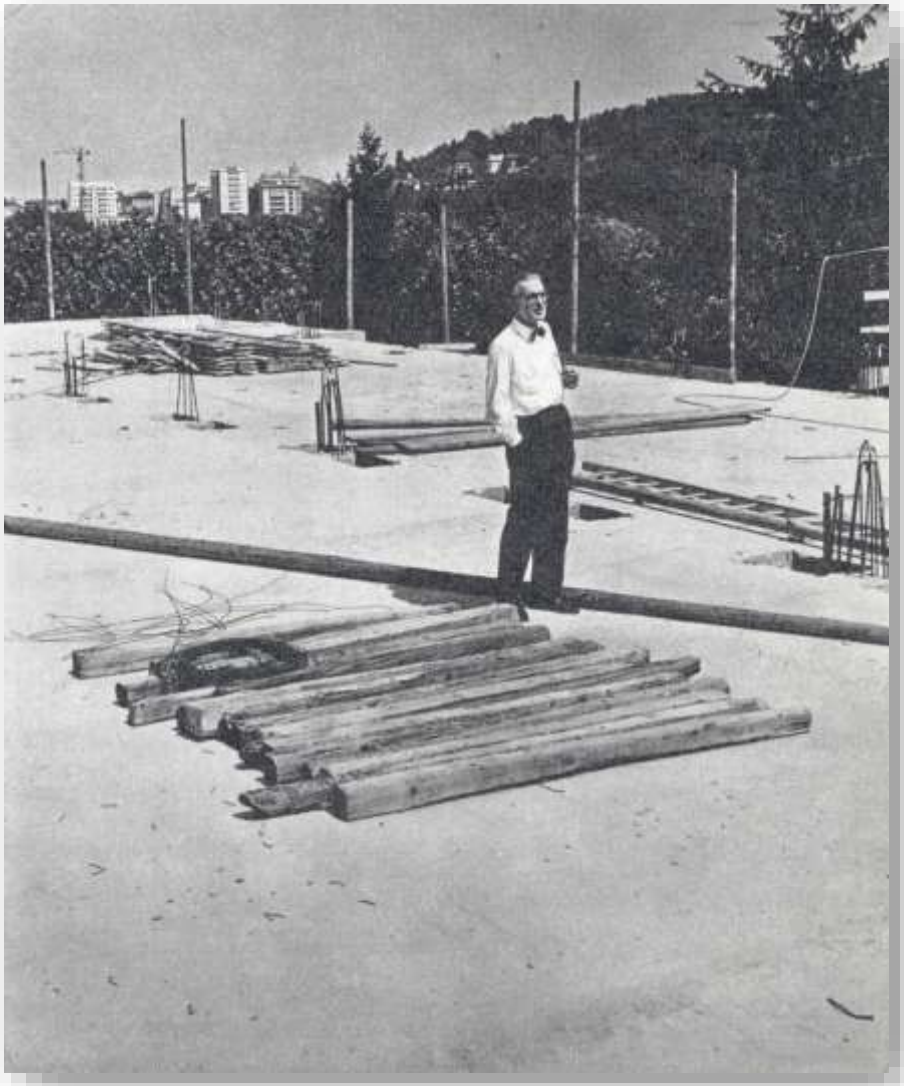
Ruggero Ceppellini

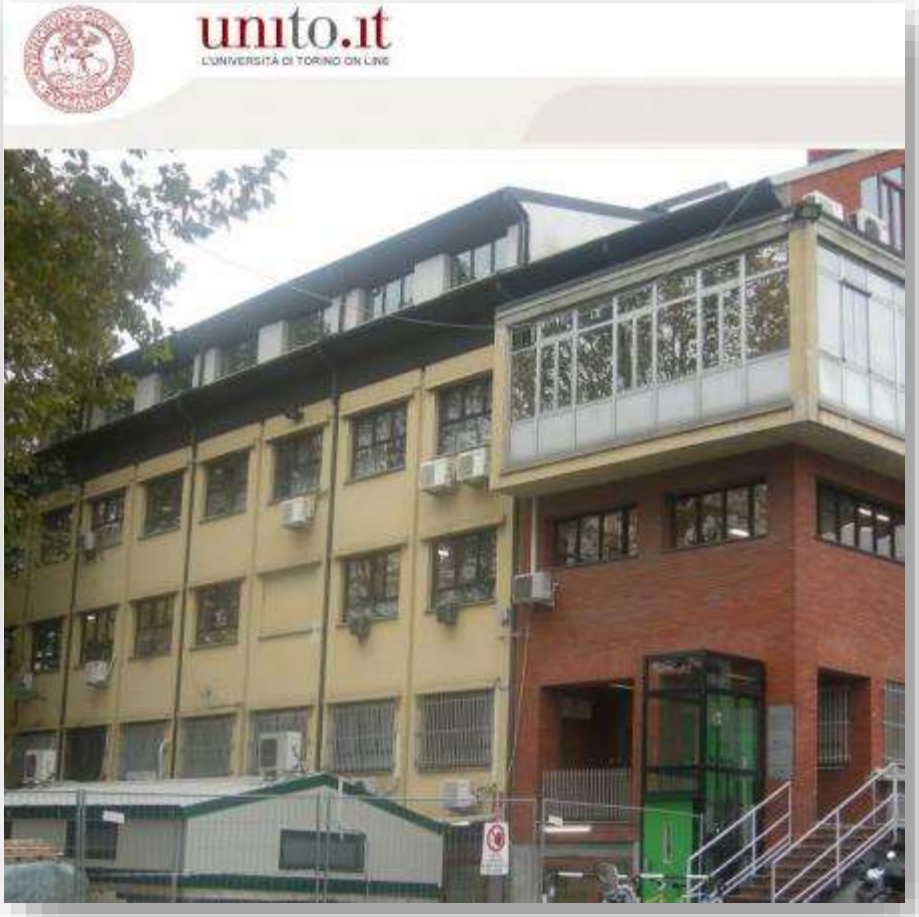
RC:amp:145

Pionieri dell'immunogenetica




TEMPO MEDICO
numero 25 settembre 1963
Ruggero Cappellini, medico genetista

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L'UNIVERSITÀ DI TORINO ON LINE

La sua storia nell'istocompatibilità

Third Histocompatibility Workshop: 1967
The Third Histocompatibility Testing Workshop was organized by R. Ceppellini and was held in Torino, Italy in June of 1967 and included 110 participants

La sua storia nell'istocompatibilità

Third Histocompatibility Workshop: 1967
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venerdì 31/05/2024 12:28

WB Walter Bodmer <walter.bodmer@hertford.ox.ac.uk>

Re: sad news

A antonio.amoroso@unito.it

L'utente ha risposto al messaggio in data 17/06/2024 20:08.

That is good a will look forward to getting your draft.

I remember that photo at the 3rd HLA workshop in Torino. Ruggero Ceppellini made a point of me being a mathematician sitting at a microscope!

Best wishes
Walter

Bull World Health Organ. 1968; 39(3): 483–486.

NOMENCLATURE FOR FACTORS OF THE HL-A SYSTEM*

At the Third Conference on Histocompatibility Testing, held at Saint-Vincent, Italy, in July 1967, the participants agreed to appoint, by postal vote, a Nomenclature Committee drawn from among specialists in tissue typing, immunology and human genetics; that Committee was asked to propose a nomenclature for leucocyte antigens, which, it was hoped, might prove internationally acceptable. The Secretariat of the World Health Organization was asked to facilitate and assist in the work of the Committee. The following is the text of the memorandum drawn up by the Committee.[1]

NEW HL-A NOMENCLATURE AND PREVIOUS DESIGNATIONS [a]

New HL-A nomenclature [b]	Amos	Batchelor	Ceppellini	Dausset	Kissmeyer-Nielsen	Payne/Bodmer	van Rood	Shulman	Terasaki	Walford
HL-A1	19	1	To-8	11	LA1	LA1	LA1	—	1	Lc-1
HL-A2, or HL-AMac	1	5	To-9	1 or Mac	LA2	LA2	8a	PlGrLy^{B1}	2	Lc-2
HL-A3	4	—	To-10	12	LA3	LA3	LA3	Hill	8	Lc-3
HL-A4										
HL-A5	45	25	To-5	5	—	—	Da5	—	6	—
HL-A6										
HL-A7	2	—	To-20	10	—	4d	7c	—	5	Lc-8
HL-A8	41	2	To-7	8	—	7d	7d	—	11	Lc-7

[a] A dash (—) indicates that no symbol has been allocated within the nomenclature concerned.

[b] HL-A4 will be reserved for one of the higher frequency 4[a] factors, and HL-A6 for 4[b]. Before assigning these specificities, an exchange of serum among collaborating laboratories will be necessary.

without altering the size or reproductive capacity of the population.

The same argument applies to genes influencing success in competition for mates.

It is more difficult to analyse the case in which a number of genes influence the probability of surviving some mortality factor which is not density dependent in its operation and in which intraspecific competition is not involved. Consider, for example, resistance to some physical factor, such as winter low temperatures (this probably depends in most cases on intraspecific competition for food and cover, but I shall ignore this). In such a case there is some plausibility in the argument that if $A\,B\,C\,\ldots\,N$ is the optimal genotype for survival, the selective advantages for each of the n loci being appreciable, then an initial population of $a\,b\,c\,\ldots\,n$ individuals could not have survived at all. But this argument applies only to an environment which is uniform in space. Given a spatially variable environment, it is possible that the initial $a\,b\,c\,\ldots\,n$ population could survive in the warmer part of the region, and that each gene substitution $a \rightarrow A$, $b \rightarrow B$, and so on, was favoured by selection because it increased the geographical range in which individuals could survive.

This argument from the variability of the environment refers not only to resistance to physical factors, but to non-density dependent mortality factors in general, in so far as they vary spatially.

The Rate of Evolution

The assumptions that underlie the "cost of natural selection" argument—that fitnesses are multiplicative and that the fitness of a population can be deduced from the relative fitnesses of its component genotypes—are true only in rather exceptional circumstances. Hence the conclusion that it will typically take 300 generations per gene substitution is unjustified; the rate of evolution could be greater than this by one or more orders of magnitude. Kimura's conclusion that a large proportion of amino-acid substitutions are selectively neutral and have occurred by drift, although it may be true, does not follow necessarily from the cost of selection argument.

This article is not the first to query Haldane's conclusions. His assumptions were queried by Van Valen[5], who argued that in the evolution of "general adaptations", there is "no necessary low limit on the number of genes which can be selected for simultaneously". I have never been able to follow his argument, but I do not think it is the same as that presented here.

Essentially the same argument that I have used has, however, been put forward before[1-4] in a slightly different context. It had been argued by Lewontin and Hubby[10], using the cost of selection approach, that the number of loci at which populations are heterozygous is probably too great to be explained by heterosis. This conclusion has been queried[1-3] on the grounds that fitnesses need not be multiplicative, and that selection may have a threshold character. I have tried here to bring out the importance of this argument for the rate of evolution, and to consider in more detail the circumstances in which one or other set of assumptions might be true.

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6 Van Valen, L., *Amer. Nat.*, **97**, 185 (1963).
7 King, J. L., *Genetics*, **55**, 483 (1967).
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9 Ford, A. S., Reed, T. E., and Bodmer, W. F., *Genetics*, **55**, 499 (1967).
10 Lewontin, R. C., and Hubby, J. L., *Genetics*, **54**, 595 (1966).

Genetics of the Human HL-A Transplantation System

by

F. KISSMEYER-NIELSEN
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Blood Bank and Blood-Grouping Laboratory, Municipal Hospital, Aarhus, and Institute of Human Genetics, University of Copenhagen

Genetic and statistical analyses indicate that the HL-A system contains two intimately related chromosome regions containing at least seven and eight alleles, respectively. The complex antibodies which these regions give rise to consist of a mixture of smaller components.

There has recently been rapid progress in the mapping of the HL-A system which seems to be important for human transplantation in the same way as the H2 locus in mice, the Ag-B locus in rats, and the B locus in chickens.

The greatest step forward was made at the Torino workshop on histocompatibility testing in June 1967. The data collected there[1] agreed with the idea that the genetic information determining the most important transplantation antigens is located in closely linked genetic elements on one pair of autosomal chromosomes constituting the HL-A system.

The transplantation antigens belonging to the HL-A system are present on leucocytes and platelets and can be determined by means of agglutinating and cytotoxic antibodies active against leucocytes, and complement-fixing antibodies active against platelets.

Leucocyte and platelet antigens which are independent of the HL-A system are known. The corresponding genetic elements are not linked with the HL-A system. These systems do not seem to be involved in transplantation. It is apparent from previous results[2-4] and from those of the Torino workshop[1] that the most important genetic information of the HL-A locus seems to be located in two separate units most frequently called the LA and 4 series. It is not known whether they are two separate loci or two subunits of one locus. If there are two separate loci they must be very close to each other, for no recombinants have been found in fairly comprehensive family studies.

During the Torino workshop, many teams could determine perfectly identical LA antigens with their reagents: LA1, LA2 and LA3 antigens and another antigen, LA4(?), determined by three of the teams with

Kissmeyer-Nielsen et al. (Nature 219:1116, 1968) defined the first series of HLA antigens assigned to the HLA-A and B loci.

Nomenclature for factors of the HLA system*

A Nomenclature Committee composed of geneticists and immunologists, including specialists in tissue typing, has met after each of the Histocompatibility Workshops beginning with the Third Workshop in 1967. The Committee, in part under the auspices of the World Health Organization and the International Union of Immunological Societies (WHO/IUIS), met after the Sixth Workshop in Aarhus in July 1975. The expanding knowledge of the genetics of the major histocompatibility system of man has necessitated a revision of the terminology for the HLA region following the principles established in previous reports. This has been done with as few changes as possible.

New	Previous [b]	New	Previous
HLA-A1	HL-A1	HLA-B5	HL-A5
HLA-A2	HL-A2	HLA-B7	HL-A7
HLA-A3	HL-A3	HLA-B8	HL-A8
HLA-A9	HL-A9	HLA-B12	HL-A12
HLA-A10	HL-A10	HLA-B13	HL-A13
HLA-A11	HL-A11	HLA-B14	W14
HLA-A28	W28	HLA-B18	W18
HLA-A29	W29	HLA-B27	W27
HLA-Aw19	Li [c]	HLA-Bw15	W15
HLA-Aw23	W23	HLA-Bw16	W16
HLA-Aw24	W24	HLA-Bw17	W17
HLA-Aw25	W25	HLA-Bw21	W21
HLA-Aw26	W26	HLA-Bw22	W22
HLA-Aw30	W30	HLA-Bw35	W5
HLA-Aw31	W31	HLA-Bw37	TY
HLA-Aw32	W32	HLA-Bw38	W16.1
HLA-Aw33	W19.6	HLA-Bw39	W16.2
HLA-Aw34	Malay 2	HLA-Bw40	W10
HLA-Aw36	Mo*	HLA-Bw41	Sabell
HLA-Aw43	BK	HLA-Bw42	MWA

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Leucocyte and platelet antigens which are independent of the HL-A system are known. The corresponding genetic elements are not linked with the HL-A system. These systems do not seem to be involved in transplantation. It is apparent from previous results[2-4] and from those of the Torino workshop[1] that the most important genetic information of the HL-A locus seems to be located in two separate units most frequently called the LA and 4 series. It is not known whether they are two separate loci or two subunits of one locus. If there are two separate loci they must be very close to each other, for no recombinants have been found in fairly comprehensive family studies.

During the Torino workshop, many teams could determine perfectly identical LA antigens with their reagents: LA1, LA2 and LA3 antigens and another antigen, LA4(?), determined by three of the teams with

> Tissue Antigens. 1975 Oct;6(4):229-36. doi: 10.1111/j.1399-0039.1975.tb00637.x.

Cell mediated lympholysis in man. The HL-A third locus antigen, AJ (W20) as sensitizing and/or target determinant

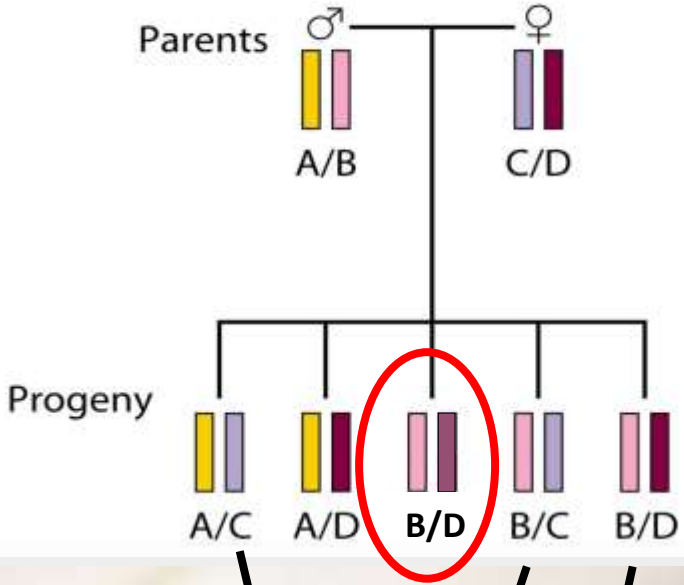
T Kristensen, N Grunnet, F Kissmeyer-Nielsen

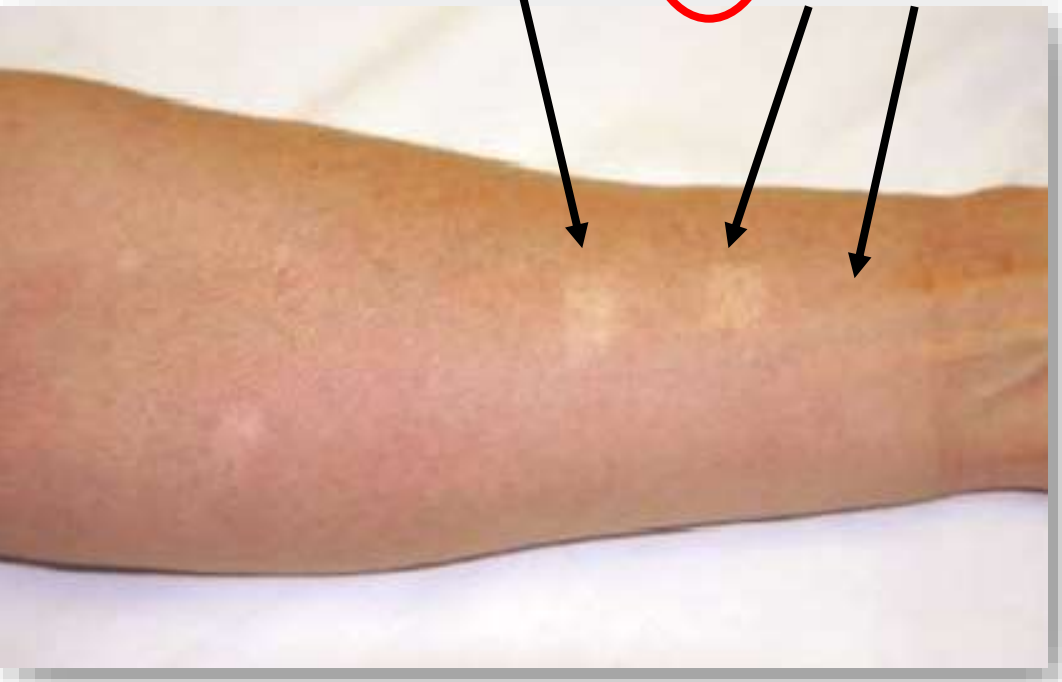
PMID: 53906 DOI: 10.1111/j.1399-0039.1975.tb00637.x

Abstract

The Indirect Cell Mediated Lympholysis (ICML) test has become a reliable in vitro tool in investigations of the immunogenetic background of the cellular immune response. It is well established that the antigens of the LA and FOUR loci of the human Major Histocompatibility Complex (MHC) or antigens governed by closely linked loci exhibiting a marked linkage disequilibrium are of major qualitative and quantitative importance in ICML. This communication is concerned with the importance of the HL-A third series antigen AJ (W20) on lympholysis in ICML. From investigations of 47 combinations where only the AJ (W20) antigen may be attacked, it is concluded that this antigen is, in itself, a poor ICML target determinant, but that AJ (W20) may function as a stronger ICML target determinant in concert with a FOUR antigen, although the actual FOUR antigen cannot be attacked.

Il ruolo del sistema HL-A nei trapianti

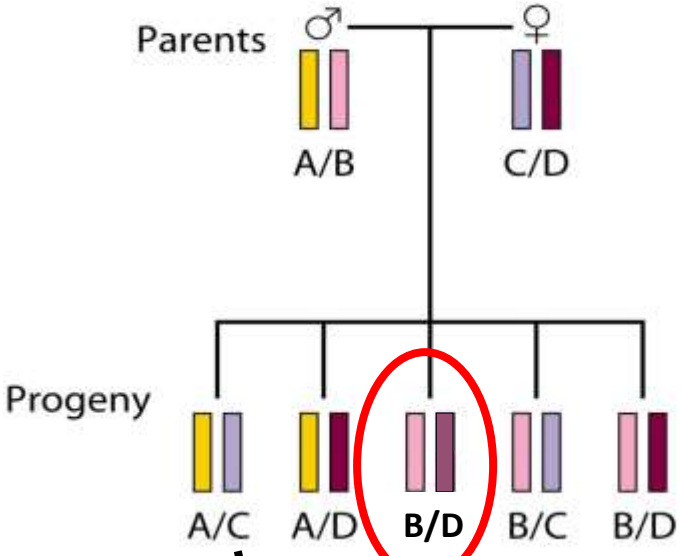


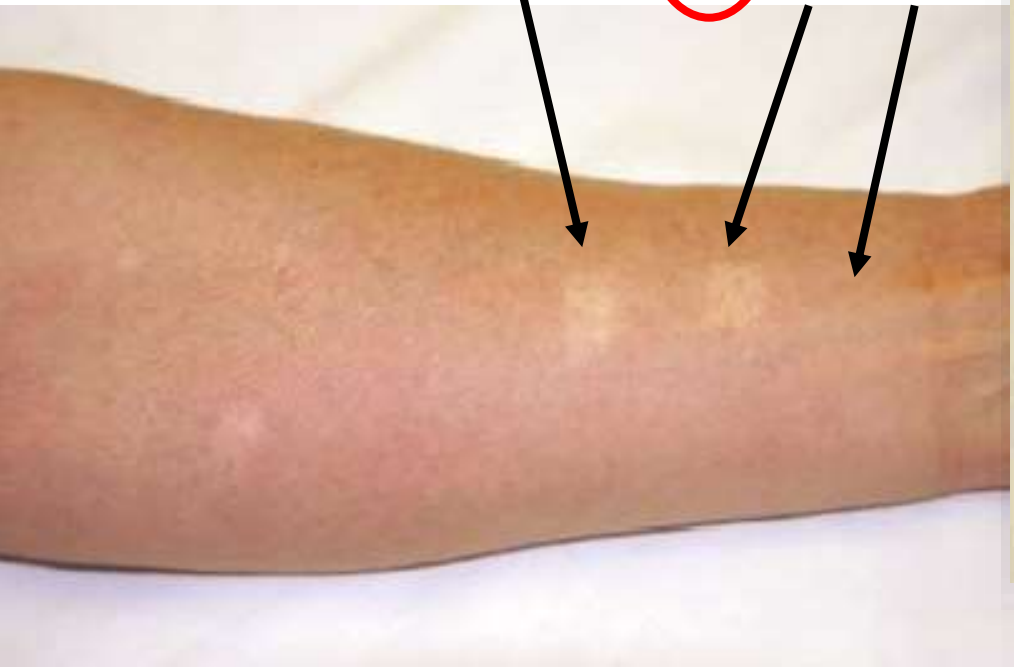


HLA Compatibility

In 1969, Ceppellini et al. (Transplant Proc 1:385, 1969) provided experimental evidence in man that indicated that the survival of skin grafts depended on HLA compatibility between the donor and the recipient.

I trapianti sperimentali erano già cominciati a Torino





Experimental Allotransplantation in Man: I. The Role of the HL-A System in Different Genetic Combinations

By R. Ceppellini, P. L. Mattiuz, G. Scudeller and M. Visetti

There is now a general consensus that test skin grafting in man is a powerful tool for the identification and study of histocompatibility factors which are important in clinical transplantation.[1] This and the subsequent paper[2] summarize the information recently collected by our group.

The proof that some leukocyte antigens, now known to belong to the HL-A system[3] were indeed transplantation antigens, had already been reached with skin grafting experiments in genetically unrelated recipients, preimmunized with white cells.[4,5] By contrast, leukocyte typing allowed a good prediction of skin graft survival only in the case of siblings.[6] In this genetic class of grafts (S:S), a bimodal distribution had been observed with a mean survival time (MST) of 20 days for the long lasting group; it was concluded that this fraction of S:S grafts probably corresponded to compatibility for a predominant histocompatibility system and that the same fraction of S:S transplants in the case of kidney under optimal immunosuppressive treatment, should have a rate of survival approaching that given by MZ twins.[7] Soon it was proved that these S:S pairs have indeed the same HL-A genotype.[8] Similar results were obtained for test skin grafting by Amos,[9] while van Rood[10] with an ingenious *a posteriori* analysis gave indirect but convincing evidence that all kidney grafts between siblings with identical HL-A phenotype (HL-A =) were surviving beyond 18 months from operation; by contrast the majority of HL-A different (HL-A ≠) pairs had disappeared from the sample because of graft rejection and death of the recipient.

Material and Methods: As described previously.[7] All genetically related subjects (sib S, parent P, child C) and the majority of unrelated subjects (U) have been typed for ABO, P and the HL-A determinants of the Torino (TO) series described in ref. 11, with the addition of TO 13, 14 (two new specificities of the LA series) and 20 (=7c).

Results

HL-A Compatibility: The survival time of the 357 grafts studied are individually reported in Table 1, subdivided according to genetic relationship, to HL-A typing and to ABO compatibility. The effect of the ABO system and the possible interplay with HL-A are discussed in the joint paper.[2] Table 2 gives the mean survival times, with the standard deviations and the statistical significance of the observed differences. The data confirm our previous findings[7] that grafts exchanged between siblings have the longest MST, followed in order by grafts between parent and child (P:C) and by grafts between unrelated (U:U). Subdivision on the basis of the HL-A typing, however, has a striking effect on S:S. In fact, the grafts between sibs who are phenotypically (and genotypically) identical for the HL-A system survive eight days longer than grafts between HL-A ≠ sibs. Note that this last group of S:S is now surviving less than P:C; when, however, it is again subdivided on the

From the Departments of Medical Genetics and of Dermatology, University of Torino, Torino, Italy.

Supported by Contracts USPHS PH-43-65-655 and CNR 115. 0282.

Da parte di ricercatori ital
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sutale", con il seguente

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b) svolgere attività colla
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c) promuovere l'aggiorn
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Torino, 22 novembre 1975

Umberto Fagiolo

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Pierluigi Mattiuz

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Cristiana Mazzilli

Olavio Baricordi

Armando Gabrielli

Cesare Gambelunghe

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Primo Congresso Nazionale dell'Associazione Italiana di Genetica Medica
Genova 3-4-Marzo 1978

ANNUAL REVIEWS

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ANNUAL REVIEW OF IMMUNOLOGY Volume 30, 2012

Review Article

The Basel Institute for Immunology

Fritz Melchers[1]

View Affiliations

Vol. 30:23-38 (Volume publication date April 2012) | https://doi.org/10.1146/annurev-immunol-020711-074912

First published as a Review in Advance on November 30, 2011

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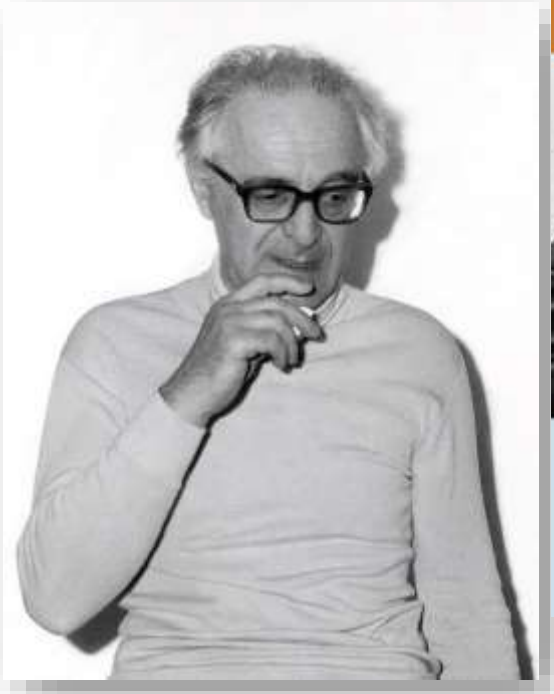
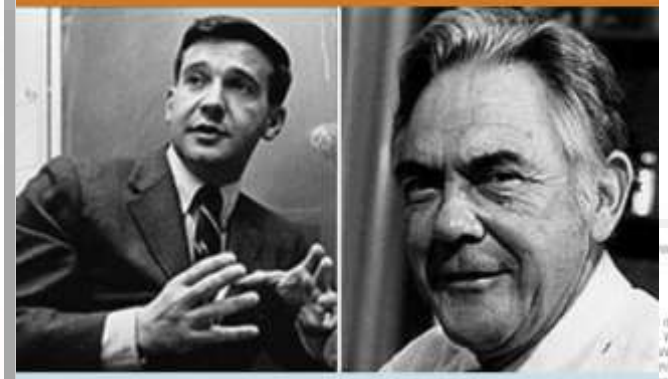
At the Centennial Exhibition of the Nobel Prize, the Nobel Foundation called it one of the ten cradles of creativity (1). The journal *Nature* likened its ideals to those of the French revolution—Liberté, Egalité, Fraternité—and called it a paradise (2) devoted to the science of immune systems: the Basel Institute for Immunology (BII). Founded by Roche in 1968, inaugurated in 1971, and closed in 2000, it was home to almost 450 scientific members, over 1,000 scientific visitors, and nearly 100 scientific advisors from more than 30 countries who worked in complete academic freedom and without commercial motives on over 3,500 projects, publishing more than 3,200 scientific papers, almost all of them on the structure and functions of immune systems of different species. This review contains a first collection of historical facts and dates that describe the background of the exceptionally successful performance and the strong scientific impact of the institute on the field of immunology.

Tommy Meo



Mixed Lymphocyte Culture (MLC) Reaction



In 1967, Fritz Bach and Bernard Amos reported that HL-A genes control the Mixed Lymphocyte Culture (MLC) reaction.

Ruggero Ceppellini (1917–1988)

from Fritz H. Bach

Ruggero Ceppellini, brilliantly creative geneticist and outstanding academician, died on 5 June 1988 having enjoyed an extraordinary scientific career spanning 36 years. Born in Milano, Italy on 19 January 1917, Ceppellini began his career as a practicing physician. In 1951 Professor Claudio Barigozzi offered Ceppellini a post (as assistant professor without salary) after reading some of his papers. Ceppellini established his formal grounding in genetics during this period, and spoke with inordinate enthusiasm about the influence of Luca Cavalli-Sforza ("a remarkable intellect and constant friend") as a teacher throughout his career.

From 1954 to 1958, Ceppellini studied at Columbia University. He had nothing other than praise and respect for both Dobzhansky and Dunn ("a great man"). During his time at Columbia, Ceppellini met Henry Kunkel ("Kunkel was great; he had imagination, originality, and the ability to see things differently from others").

After his return to Italy from Columbia, Ceppellini's career blossomed. His insights into genetic systems illuminated many areas. He undertook a study of genetic factors that played a role in resistance to *Plasmodium falciparum*. He noted that glucose-6-phosphate dehydrogenase deficiency and thalassemia were more frequent in areas endemic for malaria, and demonstrated the protective influence of heterozygosity for these genes against malaria. In 1962, he became director of a CNR (National Research Council) sponsored center for the study of immunogenetics and histocompatibility. From 1962–1978

Ruggero Ceppellini (1917–1988)

he was Professor of Medical Genetics at the University of Torino and Director of the Istituto di Genetica Medica. In 1978 he became Professor of Clinical Immunology at the University of Milano, and in 1983 Professor of Human Genetics at that university. He held these latter positions until his death. He was appointed one of the first senior members of the Basel Institute of Immunology and became a permanent member of that institute.

Through Cavalli-Sforza, Ceppellini was exposed to the teachings of R.A. Fisher. Ceppellini liked to recall finding a printing error in a formula published by Fisher (which he rederived to ensure that he understood it) and his nervousness about writing to the master. Fisher not only sent thanks to Ceppellini (it turned out that the formula initially published by Fisher was correct but had been reproduced incorrectly), but later, when they met, complimented the young scientist for noticing an error that had been missed by all, even through the many translations of that work. This need to understand in detail any facet of his work accompanied Ceppellini for the rest of his career; with full understanding came imaginative application, critical interpretation and penetrating insights.

The presentations at the first International Congress of Genetics in Bellagio in 1953 intensified Ceppellini's fascination with human genetics and with Fisher's formulation of a series of closely linked genes functioning as a supergene. If one can point to a single focus pervading his subsequent research, it was Ceppellini's enchantment with the fundamental genetics and biological import of inter-related, closely linked genes – a critical concept that he brought into focus for all of us. He summarized this concept of a group of functionally related, genetically linked loci in his introduction of the term 'haplotype', about which he once joked that if he had a penny for each time that the term was used, he would be a very rich man. It was at

Storia moderna dell'HLA: I miei primi 40 anni

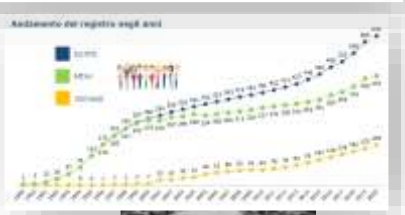
Torino, 9 novembre 1979

Storia moderna dell'HLA: i **miei** primi 40 anni

2016 NGS typing: la nuova frontiera

2005 SBT typing: esplodono nuovi alleli

2017 trapianti: mai così tanti in Italia



2000 incrementano i donatori del registro

1999 Legge 91 sui trapianti e nasce il CNT

1987 HLA cristallizzato

1995 IHWS a S. Malo: DNA typing

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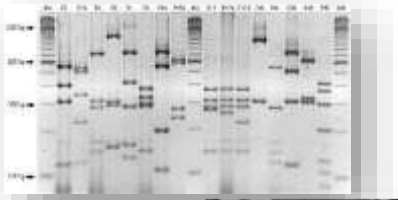
1987 Nasce AIBT



1985 Nasce l'EFI

1985 inizia l'epoca della biologia molecolare e HLA

1981 primo trapianto di rene a Torino

1980 Comincio a lavorare al centro di Immnogenetica di Torino

1986 a Londra l'Anthony Nolan Found recluta 100.000 donatori

1984 nel IX IHWS di Monaco si definisce HLA-DQ

1980 Borel scopre la ciclosporina e inizia una nuova era per i trapianti

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Original Investigations Published: December 1981

Volume 12, pages 615–626, (1981) Cite this article

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Patricia Richiardi, Antonio Amoroso, Tiziana Crepaldi, Ruggero Ceppellini & Massimo Trucco

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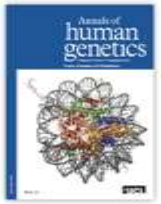

Volume 49, Issue 1
January 1985
Pages 47-63

Genetic and population structure of four Sardinian villages

A. PIAZZA, W. R. MAYR, L. CONTU, A. AMOROSO, I. BORELLI, E. S. CURTONI, C. MARCELLO, A. MORONI, E. OLIVETTI, P. RICHIARDI, R. CEPPELLINI

First published: January 1985 | https://doi.org/10.1111/j.1469-1809.1985.tb01675.x | Citations: 61

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L'AIBT: la nostra comunità

- Il contributo della ricerca italiana
- I nostri congressi
- I nostri presidenti
- L'album dei ricordi

Storia moderna dell'HLA: il contributo italiano

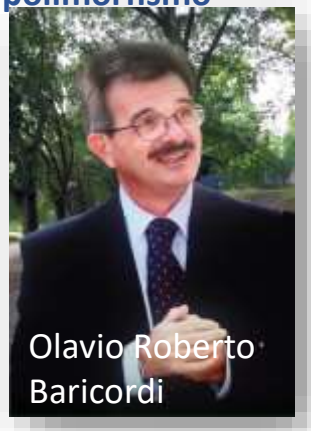

Katharina Fleischhauer


Luca Vago

2009 Perdere l'HLA spiega la ricaduta nei TMO aploidentici

2003 L'incompatibilità HLA-DP può essere permissiva nei TMO

2002 A cosa serve HLA-G e il suo polimorfismo


Olavio Roberto Baricordi


Cristina Mazzilli

1986 HLA-DQ e celiachia

1983 Il polimorfismo HLA a livello molecolare


Roberto Tosi

1978 un nuovo locus HLA: DC, che diventerà DQ


Giovanna Angelini

EUROPEAN FEDERATION for IMMUNOGENETICS
Ceppellini Award

CEPPELLINI AWARD

Each year at the annual EFI meeting a scientist who has made a substantial contribution to the field of Immunogenetics is honoured by the society and invited to present their work in the form of the Ceppellini lecture.

The lecture is named in honour of Ruggero Ceppellini (1917-1988) an Italian geneticist who was greatly influential in the HLA field. He was the organiser of the Third International Histocompatibility Workshop held in Torino, Italy in June 1967.

2024	Jamie Rossjohn	2006	Rolf Zinkernagel
2023	Ronald E. Bontrop	2005	Mary Carrington
2022	Peter C. Doherty	2004	John Trowsdale
2021	Jacques Neefjes	2003	Peter Parham
2020	-/-	2002	Marie-Marthe Tongio
2019	Pamela Bjorkman	2001	Dominique Charron
2018	Lorenzo Moretta	2000	Julia G. Bodmer
2017	John Kappler	1999	Peter Morris
2016	Effie Petersdorf	1998	Paul Terasaki
2015	Frans Claas	1997	Eva Klein
2014	Marco Colonna	1996	Hidde Ploegh
2013	James McCluskey	1995	Hugh McDevitt
2012	Gerhard Opelz	1994	Andrew McMichael
2011	Erik Thorsby	1993	Alberto Piazza
2010	Emil Unanue	1992	Fritz Bach
2009	Cees Melief	1991	Jack Strominger
2008	Hans-Georg Rammensee	1990	Walter F. Bodmer
2007	Klas Karre	1988	Jon J. van Rood

2014 Marco Colonna

2018 Lorenzo Moretta

EUROPEAN
Julia Bodme

2016	Han *Unive*
2015	Celi *Unive*
2014	Clen *Oxfor*
2013	Dan *Unive*
2012	Pier
2011	Lloy *Leide*
2010	Con *Hann*
2009	Luca Vago *San Raffaele Scientific Institute, Italy*
2008	Roberta Rizzo *University of Ferrara, Italy*
2007	Blanca Rueda
2006	James Traherne *University of Cambridge, UK*
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Candidates must be an EFI member (or becor
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candidates will be reviewed by all members c
highest priority to scientific merit for establis
candidate will be selected. The JBA winner wi
speaker of the EFI Conference Opening Sessi
and to receive €1.000 in prize money. In addi
lodging for the 3 nights of the EFI Conference

The call for applications will be published clos
Histocompatibility Conference.

2024	Agnes Bonifacius *Medical School Hannover, Germany*
2023	Esteban Arrieta Bolanos *University Hospital Essen, Germany*
2022	Jesse Bruiijnesteijn *Biomedical Primate Research Center, Rotterd*
2021	Cristina Toffalori *IRCCS San Raffaele Scientific Institute, Milano*
2020	-/-
2019	Asbjørn Christophersen *University of Oslo, Norway*
2018	Maxime Rotival *Institut Pasteur, France*
2017	James Lee *Harvard University, USA*

2005 GB Ferrara

2012 Francesca Poli

2013 Maria Edvige Fasano

EFI medal can be proposed to the Executive Committee by any of the EFI committees. Exclusions to candidates for the EFI medal include past EFI Presidents and past Ceppellini lecturers.

A list of all the recipients of this award is given below.

2021 Carlo Carcassi

www.efi-web.org

2021 Luca Mascaretti

2022 Francesca Quintieri

2017	Pascale Perrier
2016	Susan Martin
2015	Anne Cambon-Thomsen
2015	Jean-Marie Tiercy
2014	Susan Corbin
2014	Chryssa Papasteriades
2013	Maria Edvige Fasano
2013	Ciaran Dunne

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2005	GB Ferrara
2005	Zafirys Polymenidis

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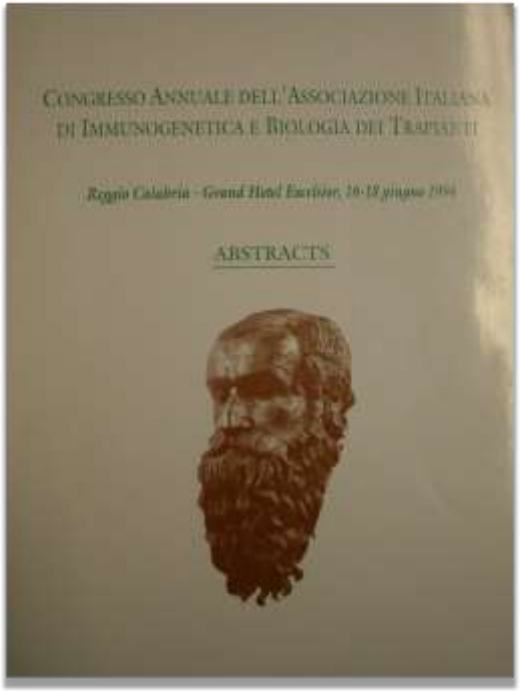
Storia moderna dell'HLA: I nostri congressi



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2004	Reggio Ca
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2006	Pavia
2007	L'Aquila
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2009	Verbania
2010	Firenze EFI
2011	Bologna
2012	Roma
2013	Pisa
2014	Trieste
2015	Matera
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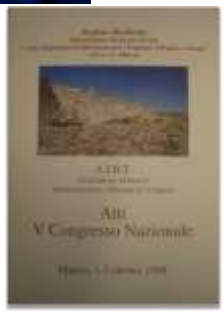
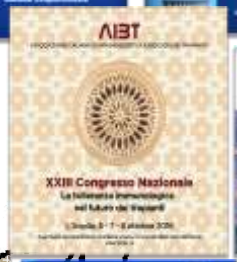
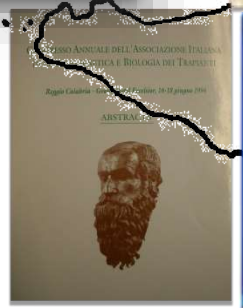
2021	Virtuale
2022	Parma
2023	Roma
	Napoli





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2023	Roma
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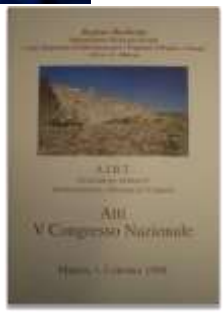


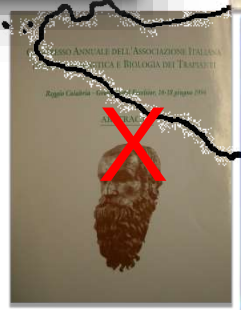


Storia moderna dell'HLA: I nostri presidenti

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Sergio Curtoni

Pier Luigi Mattiuz


Sergio Curtoni


Carlo Carcassi


Valerio Misefari


Valeria Miotti


Franco Papola


Miriam Martinetti

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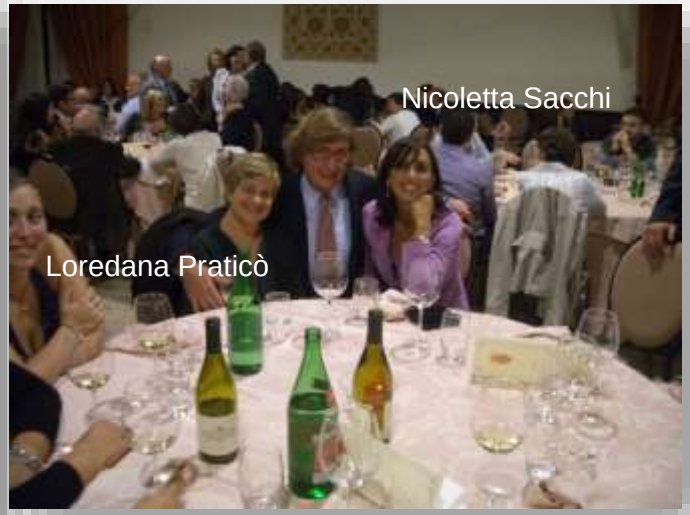

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Venezia 2018


Antonina Piazza
Mimmo Adorno
Paul Terasaki


Valerio Misefari
Ekkehard Albert


Valerio Misefari
Marcelo Fernandez Vina

AIBT Torino 2017


IHW Rio 2008
Edvige Fasano


Efi Praga 2011
Miriam Martinetti


Efi Oslo 2006
Katharina Fleischhauer
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SIMULA
downhill skiing


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AIBT Roma 2023

AIBT Pisa 2013

Brancaleone 2012


Alter Ego
Alter Ego


Biagio
Favoino
Valeria
Miotti

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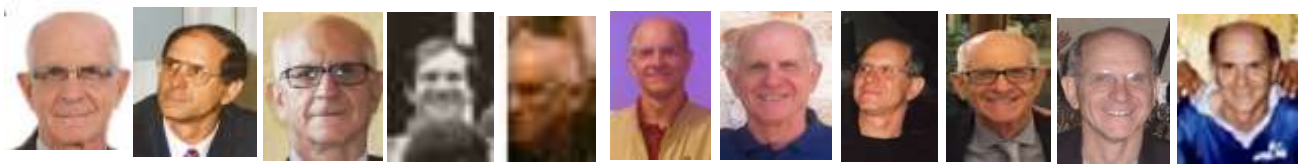
- Valerio Misefari
- Carlo Carcassi
- Valeria Miotti
- Sergio Curtoni
- Antonina Piazza
- Miriam Martinetti

Di chi vi ho mostrato il maggior numero di fotografie?

- Valerio Misefari



- Carlo Carcassi

- Valeria Miotti

- Sergio Curtoni

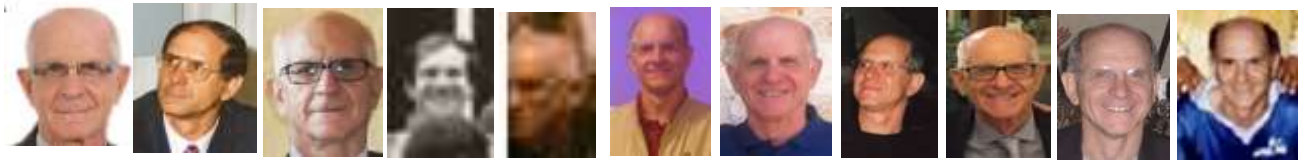
- Antonina Piazza

- Miriam Martinetti

Di chi vi ho mostrato il maggior numero di fotografie?

- Ruggero Ceppellini

- Valerio Misefari



- Carlo Carcassi

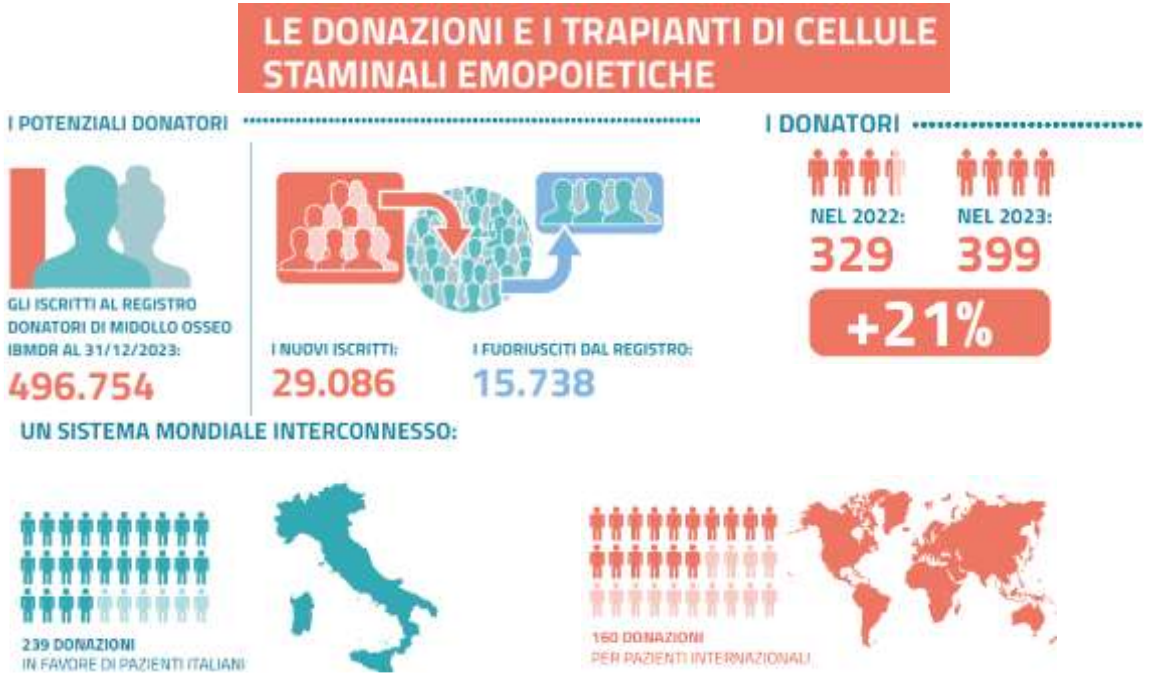
- Valeria Miotti

- Sergio Curtoni

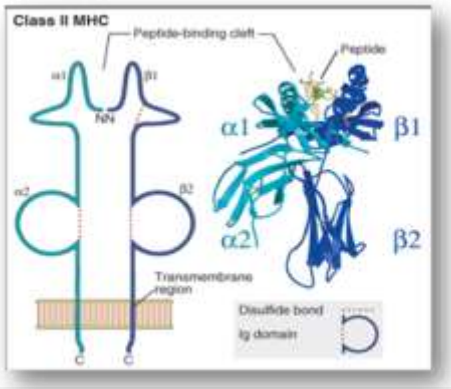
- Antonina Piazza

- Miriam Martinetti

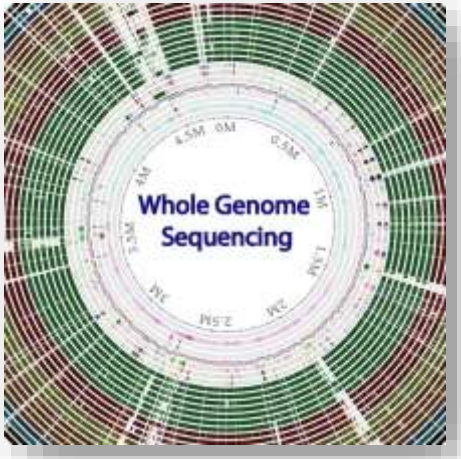
Storia contemporanea dell'HLA: dove siamo?



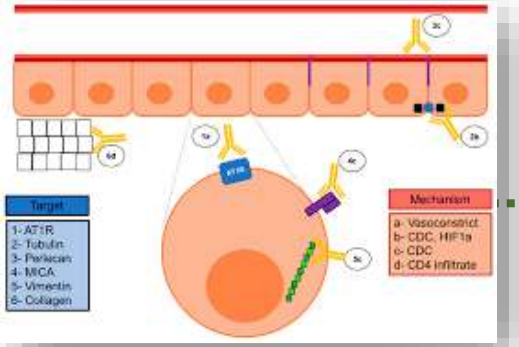
Storia dell'HLA: i **vostri** prossimi anni



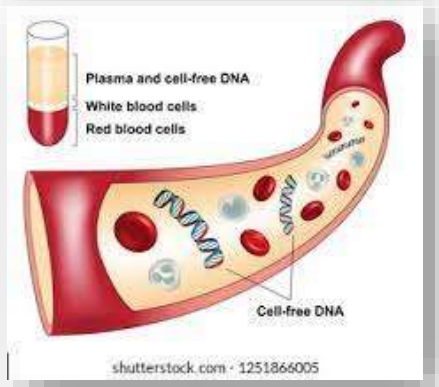
tipizzazione HLA per i vaccini



la tipizzazione HLA dal genoma



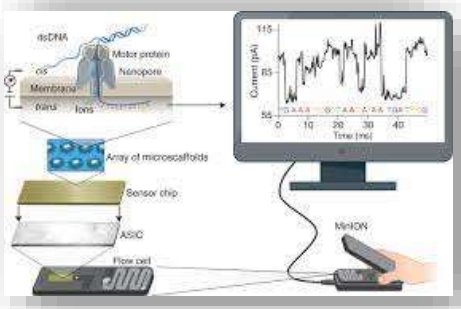
Studio degli anticorpi non-HLA



HLA dal DNA libero circolante

farmacoimmunogenetica

Immunogenomica: Oltre l'HLA



long rank HLA typing single strand

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- Guido Forni
- Franco Papola
- Miriam Martinetti

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