

isolate the true genetic ground of the phenomenon.

An account is given of basic techniques in time-series analysis, and results obtained in the field of infant mortality in Italy are discussed.

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CHRONOGENETICS OF MUSCULAR DYSTROPHY

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A genealogical study has been carried out in the families of subjects affected by progressive muscular dystrophy.

The following parameters have been taken into account: (1) the age of onset, (2) the clinical characteristics, and (3) the age of the possible exitus.

Consanguineous series, with their genetic correlation, are drawn from this sample of families, and r correlation coefficients are calculated with respect to the above experimental parameters. The r values thus obtained are then compared with those that one would expect on the hypothesis that the temporal traits considered be genetically determined.

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A CHRONOGENETIC APPROACH TO PSORIASIS

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Psoriasis is a genetically conditioned dermatological disease characterized by significantly different sex and age distributions. It could thus be the object of a chronoge-

netic analysis, using the age of onset as a genetically determined and quantitative phenotypic parameter.

A total of 2000 index cases has been considered and their families analyzed with respect to the segregation ratios in the sibships of the subjects and in those of their parents. The observed values of segregation are found to be not significantly different from those expected under the hypothesis of a diallelic monomeric autosomic trait.

It is concluded that the temporal trait, age of onset, must be genetically conditioned and that its chronogenetic variability — determined by the genetic variability of the starting ergons — may be assessed by means of standard techniques for quantitative analysis.

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WILSON'S DISEASE AND MENKES' DISEASE—A CONTRAST IN CHRONOGENETICS

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In discussing the "timing" of hereditary diseases in their book *Cronogenetica: l'Eredità del Tempo Biologico*, Gedda and Brenci point out the limitations of the traditional classification of abnormal genes into lethal and sublethal categories.

The diseases with which this paper is concerned provide a striking confirmation of such limitation, in their clinical and metabolic aspects. Both diseases can be characterized by the disorders of the physiology of copper, an essential trace-element, which they display.

In Wilson's disease the patho-physiology is one of copper-toxicity associated with failure of biliary excretion of copper and its accumulation in various organs.

In Menkes' disease the problem is related to defective gastro-intestinal absorption of copper and the pathologic lesions are for the