

4-1943

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### Recommended Citation

Oliver, C. P. (1943). A Case Of Mongolism With Some References To The Collection Of Data. *Journal of the Minnesota Academy of Science, Vol. 11 No. 1*, 29-32.

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A CASE OF MONGOLISM WITH SOME REFERENCES  
TO THE COLLECTION OF DATA

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Although the defect has been recognized since the description by Langdon-Down in 1866, mongolian imbecility is not yet well understood. Many theories have been offered to explain mongolism, but most of them do not fit all the facts. Mongolism occurs most often among children of mothers who are approaching the end of the child-bearing period. In many cases, the mongol is reported to be the last child of a family. Some of them are the last child due to the desire of the mother who did not want to take the risk of having a second defective child. In other cases the age of the mongol at the time the data were reported indicates that other pregnancies may follow (1).

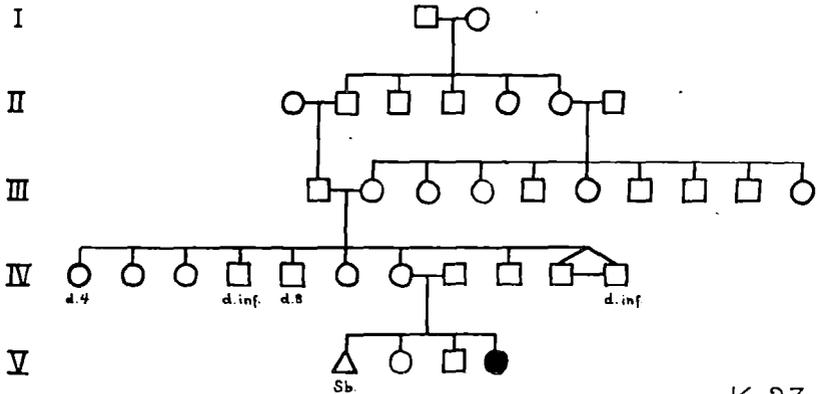
Maternal age is a recognized factor in mongolism. If mothers of mongols are graded by ages, the proportion of mongol children rises steadily from the youngest to the oldest mothers. The chance of producing a mongol child more than doubles for each increase of five years in maternal age (2). Apparently the maternal-age factor is transitory in nature or the children differ in their reaction to the prenatal environment. Some mothers produce normal children after they have produced one or more mongol (1). In every known case of mongolism in fraternal twins, only one has been a mongol. If maternal age were the only factor involved, one should expect twins to be alike in the expression of the trait. Hereditary differences should cause the children to vary in their response to the prenatal environment even though the environment might be an active factor in the development of the trait.

The inference that mongolism is an hereditary trait is supported by the familial cases of mongolism, even though the familial incidence is extremely slight. It is true, however, that the exact method of inheritance has not been established. Genetic interpretations are difficult to make when traits require the interaction of hereditary factors and environment. Mongolism develops as a result of a coexisting prenatal environment in the mother and hereditary factors in the child. The maternal environment may itself be caused by an hereditary combination, a genotype which affects the development and, thus, causes a child with the proper genotype to develop abnormally. Interacting genotypes, somewhat similar, are known in other animals.

Most of the data which have been collected on families with mongolian imbeciles do not allow complete genetic analysis of the anomaly. The data do not distinguish between the hereditary condition of the child and the possible inheritance in the mother. The

family history to be reported also lacks completeness due, as is so often the case, to the reticence of the members to discuss the condition. One can hope that people will eventually lose the feeling of shame and will discuss more freely the evidences of hereditary conditions in members of their family.

### FAMILY HISTORY OF MONGOLIAN IDIOCY



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The records of the family shown in Fig. 1 were collected by the Dight Institute. A mother in generation IV had four pregnancies: a stillborn at age 30, two normal children, and a mongol child at age 37. The mongol is shown by the solid circle. The mother had nine sibs; two died in early childhood; two died in infancy. No data are available regarding the children of the mother's sibs or other relatives. The informant reported that no other mongol occurred in the kinship. The maternal grandmother of the mongol, one of nine sibs, married her first cousin who was a son of her mother's brother. She was 38 years of age when her last children (twins) were born. No data are available about the relatives of the father of the mongol.

Probably due to the recognized influence of maternal age, most histories, as the one here reported, make a more complete survey of the maternal side of the family than of the paternal side. The parents of the mother may transmit the condition which is responsible for the development of the abnormal prenatal environment (in the mother of the mongol) and also the genotype to the child which causes him to react abnormally and, therefore, to become a mongol. The genetic complex of the child, however, may come from the paternal side of the family. Both parents of the child may be respon-

sible for the appearance of the trait and, consequently, both parental lines should be studied.

Data concerning the maternal side should include any evidence of mongolism among her collateral relatives. Her sisters may inherit the genes which cause them to develop an abnormal uterine environment, but they may produce no mongols because their children do not have the abnormal genotype. The data on the family should include any evidence which might be an indication of the hereditary condition in the mother's relatives. It is necessary to know the ages of the mother's sisters and mother when their children were born, including miscarriages and stillbirths. Evidences should be recorded of any latent expression or signs of mongolism among all relatives of the mongol, including his own brothers and sisters (3). The exact significance of such signs is not fully understood, but they may become important for a complete analysis of the genetics involved. Records should indicate whether the mother's parents were related in any degree.

The mongol child may have an hereditary basis which is entirely independent of the genotype of the mother, except that he responds to her abnormal uterine environment. He may receive the gene complex from the father. Some cases have been reported which indicate that the father may transmit the potentiality (4, 3). Therefore, the data about the father's relatives should give complete information about collateral relatives, the ages of his female relatives when they produced their children, and any latent expressions or signs which may indicate a mongolian influence.

It is probable that a dominant gene in the child causes him to respond to the maternal factor and to become a mongol. The low incidence of consanguineous marriages found among parents of mongols, not exceeding that in the general population, suggests that the trait is not recessive. A few familial cases support an interpretation that the trait is dominant. The type of heredity involved in the development of the maternal-age factor has not been studied. Suggestions as to the data about the mother's relatives; which must be collected if the genetic analysis is to be made, have been given. The family history shown in Fig. 1 has some of the essential data. The ages of the maternal grandmother when she produced her children are given, and the relationship of the maternal grandparents is shown. A single case of related parents of the mother of a mongol will not explain or prove that heredity is associated with the maternal-age factor, but it is evidence which can help in the genetic analysis.

Families which have mongols among the members can use that knowledge to their advantage. Most mongols occur after mothers have passed 35 years of age. Members of a family with a mongol who is a close relative should, in order to guard against the production of mongols, have their children when the mother is relatively young.

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## MINNESOTA SPECIES OF ALEURODISCUS

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The genus *Aleurodiscus* is a Basidiomycete of the family THELEPHORACEAE which was formerly included in the genus *Corticium*. Rabenhorst separated it as a distinct genus under which have been described a number of species many of which are doubtful. Most widely distributed in Minnesota is *Aleurodiscus Oakesii* of Cooke. This is a bark inhabitant occurring on Ironwood, Burr Oak, and White Oak, and in lesser degree on other broad-leaved trees. So commonly does it occur on these species that it seems safe to say that its distribution is the same as the distribution of these species of trees.

The fruit bodies are of irregular shape, varying in size from a millimeter to dimensions up to two centimeters. The context is leathery and the margin of each fruit body is fringed by slender hyphae that have a white cottony appearance, particularly when dry. The surface of the fruit body inside this border is smooth and of a light gray to light brown color. The fruit bodies originate as globose masses of hyphae, which gradually develop into minute cup-shaped structures having the appearance of those of *Peziza*. In fact, the species has been described under that genus. The cup form is, however, gradually obliterated as the hymenial surface flattens out. So long as the plant is moist it keeps the flat shape with the white fibrous margin framing it. However, on drying out, it again assumes a cup shape, even to the extent of the edges becoming quite involute. On drying, the hymenial surface whitens and the large protruding basidia as well as the paraphyses appear as powdery white grains under the hand lens, suggesting wheat flour, a characteristic of the genus that suggested to Rabenhorst the name *Aleurodiscus*.

On oak trees, the fruit bodies appear most abundantly on those areas in which the outer rough bark is not too thick. If a part of the outer cork segments have by chance been rubbed away at an earlier time, the area makes a most favorable site for the growth of the fruit bodies. The mycelium makes stromata beneath the cork and finds exit through cracks in the cork layer. These cracks are en-