

Cell Defects in Melanocytes Cultured from Patients with Vitiligo

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1. Scientists in our laboratories have completed the isolation, characterization and complete structural identification of an entirely new class of molecules which affect melanocytes and might be useful for treatment of pigmentary disorders. More information will be forthcoming in future updates.
2. We believe that we have evidence for the presence of substances that stimulate pigment cell proliferation. This agent holds significant promise for treating vitiligo by methods other than PUVA.
3. We have identified where the vitiligo gene resides in murine genome. (C. Lamoreaux et al., J. Heredity Dec. 1992). The cloning of this gene labeled the "mi" gene, is proceeding rapidly. Significant portions of the gene have been isolated and sequenced. A second molecular biologist has been hired to speed up the work. This work has been possible through the generosity of Frank and Simone Dodge and the NVFI.
4. A new study on the effects of the vitiligo gene on human skin has been initiated at the University of Cincinnati.
5. A collaborative study to search for other genes is in the planning stages.
6. Documentation that pigment cells have significant effects on healing of wounds (S. Boyce et al JID in press – High Impact Paper).

PAST PROGRESS

The progress throughout the world on understanding the cause and effects of vitiligo has been momentous in the last five years. The highlights of work done just at the University of Cincinnati include:

1. Development of the technology to culture normal or vitiliginous melanocytes from mice and humans. Cells can now be studied extensively.
2. Identification of intrinsic defects within murine and human melanocytes, an observation which gives us important clues to etiology of some types of vitiligo which probably is more than one disease.
3. Identification of some effects of the genes on functions of some skin cells.
4. Location of the vitiligo gene at the "mi" locus in mice.
5. The fortunate identification of the transgenic mouse which allows us to isolate these normal and abnormal genes.
6. The transmission completion of studies on genetics of vitiligo in humans.
7. The completion of studies on clinical manifestations and associations of vitiligo in humans. The results should modify our concepts about the disease vitiligo.
8. The documentation that melanocytes have significant effects on cutaneous functions. These data should help convince funding agencies that vitiligo is important to study and is worth funding, It might also convince insurance companies to pay for care of patients with vitiligo.

FUTURE FIVE YEARS

1. The vitiligo gene will be cloned and its defects identified.
2. Other genes contributing to vitiligo will be identified.
3. New drugs will be in preparation to alter these defects.
4. Agents that might halt the progression of vitiligo might be available.
5. Techniques for identifying carriers of vitiligo genes might be available; they certainly will be under study.

COMMENTARY

In 10 years, the problem of vitiligo could be resolved. To accomplish this, each of the one million or more people in the US needs to contribute to the NVFI to support this research and insure that it can be completed as rapidly as possible.