

Special Report: The March of Dimes Clinical Genetics Conference— July 19-22, 1987, Minneapolis, Minnesota

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Growth, Genetics, and Hormones

The theme of this conference was "disorders of the neural crest and craniofacial skeleton." Thus, neural crest development and the possible effects of teratogens upon it were reviewed. Clearly, neural crest cells in the cranial area are more diversified in their

actions than neural crest cells in the trunk.

A number of new experimental techniques permit identification of neural crest cells and observation of their migratory patterns in experimental animals. In the craniofacial area, neural crest cells or ectomesenchymal tissue separate from the neural tube laterally and migrate within the mesoderm to form somitomeres, which are

segmental areas within the cranial region. The neural crest cells provide muscle and bone, as well as endothelial cells for angiogenesis, in the cranial area. In the trunk, neural crest cells play a role primarily in pigmentation and in development of the autonomic nervous system.

Utilization of a Nile blue stain to indicate areas of necrosis in an embryo permits the suggestion

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that there are large areas of cell death that are programmed as part of normal embryological development. These areas that are programmed for cell death seem to be accentuated by teratogens,

appearing earlier and resolving later than they normally would.

It was clear from the presentations that simple neurocristopathies, which are associated with single defects, should be distinguished from complex neurocristopathies, which are characterized by structural anomalies and/or ongoing problems, such as dysplasia and overgrowth.

A number of new craniofacial syndromes were described, but the classification of neural crest disorders is still very difficult and complex. New imaging techniques, such as magnetic resonance imaging and stereoradiophotogrammetry, are improving our abilities to diagnose and describe the natural history of already described conditions.