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per unit area of lung in the CP group was only 42% that of the NP or control lungs. At all stages of development studied, whether treated or not, the ultrastructural appearance of the epithelium was normal and no interference with its differentiation could be detected. We conclude that the presence of cleft palate causes pulmonary hypoplasia probably by interfering with the normal fluid exchange between the amniotic sac and the lung. (Barrugan & Cottell, Teratology, in press).

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Spontaneous neural tube defects in the chick embryo at the end of incubation.

The spontaneous malformations associated with the dysraphic state of the chicken CNS are seen when unhatched eggs are opened at the end of normal incubation cycles (Canavese et al., 1979). Encephalo-myelo-dysraphic embryos, still alive on the 21st day of incubation, have been classified as esencephalic and anencephalic merocranial or olocranial (Lemire et al., 1978; Colitti and Canavese, 1989). Material for observation under OM was processed with normal histological procedures; that for SEM was fixed in formalin (10%, phosphate buffer pH7.4) and then dehydrated through a graded series of ethanol, critical-point dried and sputter coated with gold paladium. The authors use comparisons between OM and SEM observations to highlight the seriousness and the open or closed nature of the neural defects in the modification of the structural relationships between the areas involved. SEM observations showed how the forms in which the neural tube had closed had surface structural characteristics similar to those in the controls. However, the forms where the neural tube remains open have surfaces similar to those of the ependima and cerebral ventricles, shown by the widespread presence of microvillae.

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Mouse preimplantation development as revealed by cinemicrography.

Cinemicrographic recording of successive preimplantation developmental events of mouse embryos cultured in vitro was performed, in order to obtain data on the dynamics of early morphogenesis (as a next step, normal data recorded will be used as control in similar, cinemicrographic studies on the development of some blastopathies). Film sequences are presented on development from 2 cell to 8 cell stage, on compaction, on the cavitation process and on „hatching" from the zona pellucida. Comments are made as to some still unexplained aspects as: the rotation of embryos within the zona, the emission of cytoplasmic protrusions through the zona, the pulsatile activity of blastocysts. Based upon present observations the possible mechanism of „hatching" is considered.

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The ectodermal cells of the limb bud: source of the mesodermal compartment

Somites and somatopleura are generally considered to be the sources of the mesodermal compartment of the limb buds. These data are based on experiments with embryos of lower vertebrates, e.g. chick-quail chimaeras. In studies on cultured murine embryos, using wheat germ agglutinin conjugated to gold particles (WG-Au) as a marker for neural crest and ectoderm derived cells, it was noticed that the mesodermal compartment of the limb buds randomly contained WG-Au-labeled cells (Smits-van Prooije et al., Anat. Embryol. 177, 1988). These areas of labeled cells had no spatial connection with either those of neural crest or primitive streak origin. In addition, the somites contained hardly any labeled cells.
Therefore, it was suggested that the mesodermal cells originate from the limb bud ectoderm. On the analogy of the mesoderm formation of the neural crest, the disruption of the basement membrane, visualized with immuno-histochemical techniques, and the occurrence of programmed cell death (apoptosis) was studied. In the surface ectoderm of the limb buds, before and during development, up to stages of the separation of the fingers and toes, apoptosis was observed. Cell death is considered to cause disruption of the basement membrane, resulting in ectodermal cell deposition (Vermeij-Keers and Poelmann, Neth. J. Zool. 30:74-81, 1980). In embryogenesis, the regulatory mechanism of apoptosis has not been elucidated yet. It is probably initiated extracellularly by hormones or growth factors, followed intracellularly by TGFß production. Derailments in apoptosis in connection with cell deposition result in congenital malformations of the extremities, such as reduction anomalies and duplications.