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Australian Birth Defects Society, Inc. Annual Scientific Meeting Friday December 1, 2000 at the Royal Hospital for Women, Randwick New South Wales, Australia Program and Abstracts

PROGRAM

0830–0900—Registration

Session 1—Chair: Associate Professor Paul Lancaster

0900–0905—Opening (P. Lancaster)
0905–0910—Tribute to Assoc. Prof. David Walsh (M. Smith)
0910–1010—Tony Lipson Lecture: Reproductive risk evaluation from clinical and public health perspectives (E. Robert)
1010–1040—Experiments preceding fetal surgery (D. Challis)
1040–1100—Morning tea

Session 2—Chair: Associate Professor Paul Lancaster

1100–1200—M.J. Edwards Annual Lecture: How to reconcile safe fetal development and maternal needs (A. Pastuszak)
1200–1230—Policy for prevention of birth defects by periconceptional folic acid in New Zealand (B. Borman)
1230–1330—Lunch

Session 3—Chair: Dr. Debbie Kennedy

1330–1350—Norman McAlister Gregg and congenital rubella (P. Lancaster)
1350–1410—Increasing temporal trend in gastroschisis and its association with young maternal age in Australia (P. Lancaster)
1410–1430—ICSI, IVF and major birth defects: a cohort study (C. Bower)
1430–1445—The aging gamete, sexual behavior and congenital anomalies (I. Pollard)

Session 4—Posters Chair: Dr. Debbie Kennedy

Thalidomide revisited (Prof Janet McCredie)
Changes in the heart and aorta in experimentally induced fetal alcohol syndrome (J. Chaudhuri)
Do herbal remedies have an adverse effect on pregnancy outcome in the rat ? (M. Yao)
1500–1530—Afternoon tea

Session 5—Chair: Dr. Helen Ritchie

1530–1545—Human germ cells do not have to migrate in embryonic development (B. Freeman)
1545–1600—Human “branchial” whatevers: perpetuating Haeckel’s forgeries (B. Freeman)
1600–1615—Fetal abnormalities associated with high-dose tranylcypromine in two consecutive pregnancies (D. Kennedy)
1615–1630—Maternal age-specific rates of Down syndrome in Australia (T. Hurst)
1630–1650—Male mediated developmental toxicity and the herbicide formulation Tordon 75D (D. Oakes)
1650–1800—Annual General Meeting, Australian Birth Defects Society

were no externally visible malformations. Conclusion: At the doses selected, the herbs hawthorn and golden seal had no adverse effect on reproductive outcome.

FREEMAN, B. School of Anatomy, UNSW, Sydney, NSW 2052. Human germ cells do not have to migrate in embryonic development

Much is written about the independent migration of cells during normal embryonic development: indeed, cell migration has become a dogma of modern embryology teaching and research. However, for any putative migration, few authors give a frame of reference for the movement or a speed; both are necessary if we are to have faith in the dogma. Since it is now known (i) that sclerotomal cells do not migrate to the vicinity of the notochord, (ii) that cranial neural crest cells do not migrate to form mesectoderm, and (iii) that vagal neural crest cells need not migrate to reach the rectal submucosa, it was decided to test the evidence for the migration of human germ cells. A re-examination of Witschi's (1948) paper in the context of the global growth movements of the embryo suggests that the displacement of germ cells can be explained without recourse to the *deus ex machina* of independent cell migration. There appears to be a difference between the behaviour of germ cells in glass dishes and their behaviour *in vivo*. The study of human embryos forces us to re-examine evidence for the active migration of germ cells in other species, such as the mouse where it is possible that growth movements have been ignored in the interpretation.

FREEMAN, B. School of Anatomy, UNSW, Sydney, NSW 2052. Human "branchial" whatevers: perpetuating Haeckel's forgeries.

Haeckel's biogenetic "law" (essentially, that ontogeny is a condensed recapitulation of phylogeny) is based on forged illustrations, or scientific fraud, as Haeckel himself admitted in a letter to the *Berliner Volkszeitung* on 29 December 1908. By 1973, accurate 3-D reconstructions of a series of young human embryos at the University of Göttingen had again proven that Haeckel's idée fixe is completely false, and that the developing human embryo does not pass though a gill-bearing stage. It is impossible to rescue the biogenetic law by subtle rewording, or by suggesting that it is only partially true. Nevertheless, textbooks in Anatomy, Embryology, and Molecular Biology still continue to describe structures in the human head and neck using the adjective *branchial* (i.e., of gills), and to reproduce the forged illustrations. Popularizations of human development perpetuate the myth for the general public. It will be argued that, if our comprehension of early human development is to make any progress, then we need to avoid defective terminology based on fraud. The arches that develop at the cranial end of the human embryo are simple biomechanical flexion folds—such folds occur throughout life at all flexion sites in the body. Since the identification of the "sixth arch" in the human is misconstrued, all attempts to search for derivatives of a "missing" fifth arch are futile. Similarly, claims for the existence of atavisms in the human embryo are false. Nowadays, it is instructive to re-examine how Haeckel dealt with his detractors, including Wilhelm His (senior).

KENNEDY DS[1], EVANS N[1], WANG I[1], and WEBSTER WS[2]. [1]Royal Prince Alfred Hospital and [2]The University of Sydney. Sydney, Australia. Fetal abnormalities associated with high-dose tranylcypromine in two consecutive pregnancies.

We present two cases of multiple fetal abnormalities associated with high-dose tranylcypromine therapy. A 41 year old woman with a long history of severe endogenous depression was treated with tranylcypromine 100mg, pimozide 1mg and diazepam 5-10mg daily. Her first pregnancy resulted in the stillbirth at 31 weeks gestation of a macerated female fetus. Post mortem examination revealed hypertelorism as well as a large atrio-ventricular septal defect, single coronary ostium and right pulmonary isomerism. There were multiple placental infarcts which were considered significant factors in the fetal demise. Following the fetal death she was hospitalised with severe depression and continued on high-dose tranylcypromine (up to 150mg daily). She then presented with secondary amenorrhoea and a positive pregnancy test and an ultrasound scan showed a 19 week fetus with a head described as "lemon-shaped". A fetal echocardiogram at 26 weeks showed an atrioventricular septal defect. Amniocentesis was performed and revealed a normal female karyotype. A C-section was performed at 38 weeks because of poor fetal growth and breech presentation. The baby was born in poor condition and was intubated and ventilated and transferred to the Neonatal Intensive Care Unit where she remained for 4 weeks. She was noted to have several dysmorphic features including hypertelorism, low-set overfolded ears, cleft palate, micrognathia and marked distal phalangeal hypoplasia. Head ultrasound showed agenesis of the corpus callosum and cardiac ultrasound confirmed the antenatal cardiac findings. We believe that these babies represent cases of fetopathy related to high-dose tranylcypromine, a MAO inhibitor known to result in reduced uterine and placental blood flow.

HURST, T., and P.A.L. LANCASTER. AIHW National Perinatal Statistics Unit, University of New South Wales and Sydney Children's Hospital, NSW, 2052. Maternal age-specific rates of Down syndrome in Australia

We analysed data on Down syndrome notified to the AIHW National Perinatal Statistics Unit by State and Territory perinatal data groups and birth defects registers. Among 1.8 million births between 1991 and 1997, there were 2,313 infants with Down syndrome and another 870 terminations of pregnancy after prenatal diagnosis of Down syndrome. Incidence rates for all maternal age groups were 12.9 per 10,000 births for births, 4.9 per 10,000 births for terminations, and 17.8 per 10,000 for births and terminations combined. The incidence of Down syndrome among births generally increased with each advancing single year of maternal age, with a more than thirtyfold increase from 4.0 per 10,000 births at 16 years to 124.8 per 10,000 at 44 years. The five-year age-specific rates for births, and combined births and terminations in brackets, were: less than 20 years—5.8 per 10,000 births (5.9 per 10,000 births); 20–24—6.3(6.7); 25–29—6.7(7.6); 30–34—13.2(14.9); 35–39—24.6(43.2); 40–44—69.0 (145.2); and 45 and over—255.0 (387.2). The age-specific rate at 39 years for births and terminations (85.3 per 10,000 births) was almost 3 times higher than the rate at 35 years (29.3 per 10,000 births) and, for women in their 40s, the rate at 44 years (264.3 per 10,000 births) was more than double that at 40 years (121.5 per 10,000 births). Maternal age-specific rates by single year showed considerable differences within the conventional 5-year age groups, particularly for older women. With increasing use of newer screening techniques for detecting Down syndrome, more younger women need accurate information about their risks. Earlier detection and termination of pregnancy also have important implications for the completeness of ascertainment of all cases among terminations and births and for accurate estimates of risk.