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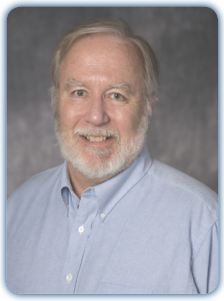

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The obligatory role of endogenous retroviruses in human pregnancy: An overview

The genome of most vertebrates has been transformed over millions of years by in-heritable integration of infectious retroviruses leading to rapid evolutionary changes as the host accommodated and used advantageous retroviral proteins. Approximately 8% of the human genome is of apparent retroviral origin (endogenous retroviruses; ERV). Although most retroviral integration sites are completely or partially inactivated through gene modification, a small number of isolated ERV elements retain transcriptionally active open reading frames so that a variety of Gag, Pol, and Env proteins may be expressed. The placenta is one of few organs that express ERV proteins under physiologic conditions. Specialized cells of the human placenta (villous cytotrophoblast) differentiate into syncytiotrophoblast by exiting the cell cycle, intracellular fusion, and secretion of preg-nancy-related hormones (e.g., chorionic gonadotropin; hCG) concurrent with expression of more than 30 different ERV-encoded proteins and shedding of non-infectious virions from the basal surface of the syncytiotrophoblast. In 1998, we published the first physio-logical role for a placental ERV protein; expression of env of a single copy ERV (ERV3) induced the β subunit of hCG (β-hCG) and diminished cell division in a cAMP/PKA-dependent manner. Later, others described physiologic roles for two other ERV env ele-ments, HERV-W and HERV-FRD, which produce typical ERV Env proteins (syncytin-1, syncytin-2) that mediated the intertrophoblast fusion process. ERV3 Env, however, is highly atypical with a proposed primary sequence incompatible with an effective fusion protein; lacking a functional membrane spanning domain, a relatively hydrophilic fusion peptide, and an atypical immunosuppressive site. As shown recently, ERV3 env encodes a hormonal control site located in the N-terminal p25 truncated molecule from the SU re-gion; completely unique to retroviral env elements. The argument may be put forth that placentation, perhaps throughout all placentate animals, may have evolved through the capture and selective expression of retroviral elements, the physiological function of most of which remain unknown.

Biography

Neal S. Rote completed his Ph.D. at Temple University School of Medicine and postdoc-toral studies at Heidelberg University and UCLA School of Medicine. He is the William Weir, M.D. Professor of Reproductive Biology and Professor of Pathology at Case West-ern Reserve University School of Medicine and Academic Vice Chair and Director of Research in the Department of Obstetrics and Gynecology, University Hospitals Case Medical Center, Cleveland, OH. He has published more than 110 papers in reproductive biology and 75 chapters and books, been NIH-funded for 32 years, and served on many NIH review committees.

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