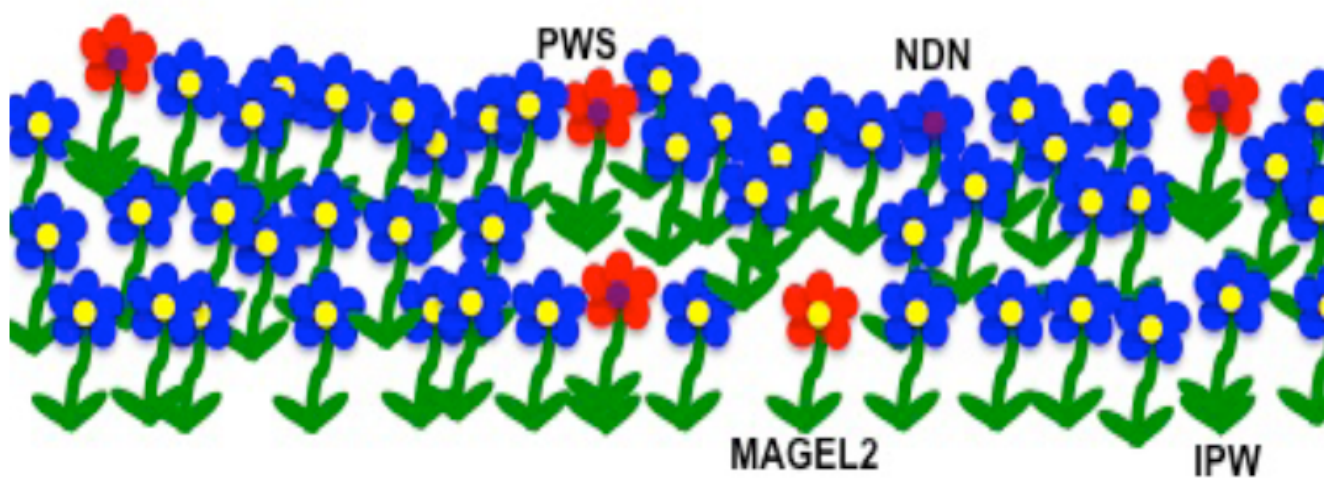
Rachel Wevrick, PhD
Professor of Medical Genetics, University of Alberta

Dr. Rachel Wevrick received her B.Sc. from Queen's University in Kingston, Ontario and her Ph.D. from the University of Toronto. She then completed postdoctoral fellowships in Medical Genetics at the Hospital for Sick Children in Toronto and Stanford University in California. She has been a Professor of Medical Genetics at the University of Alberta since 1996. Her research focuses on the genetic basis of developmental delay and obesity in children. Prader-Willi syndrome is a sporadic chromosomal disorder that causes neonatal hypotonia, developmental delay, childhood-onset obesity with disordered eating, and abnormalities of sleep and respiration. Starting at Stanford University in 1993, Dr. Wevrick co-discovered many of the genes inactivated in Prader-Willi syndrome, including Necdin, MAGEL2, and IPW. The Wevrick research group is currently studying the roles of PWS genes in the normal development of the nervous, muscular, and endocrine systems and investigating the effect of loss gene function in mouse models of Prader-Willi syndrome.

Among all the flowers in a field, the PWS flowers have defining features: purple centres, red petals, and two pairs of leaves. PWS flowers have genes that make them unique. Looking closely at the field, we also see flowers with only one of these features- red petals, because the MAGEL2 gene is missing. The Wevrick research program looks at all the defining features and tries to understand which genes are responsible for the unique nature of PWS.



In the next three years, my laboratory will be focusing more on what the MAGEL2 and Necdin proteins are doing inside of neurons. We know that MAGEL2 is important for shuttling other proteins along the lengths of axons, which are the finger-like projections from brain cells (neurons) that connects these neurons to other neurons and to muscle. We are trying to determine whether we can substitute for the absence of the MAGEL2 or Necdin proteins in neurons, and whether there the other proteins that MAGEL2 and Necdin associate with could be targeted for study to understand more about Prader-Willi syndrome and its symptoms. We are particularly interested in learning more MAGEL2 in autism and developmental delay, and Necdin in nervous system function.