Investigation into the cellular function of the Opitz Syndrome gene, *MID1* and its homologue, *MID2*

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Human congenital disorders impose a large impact not only on the affected individuals and their immediate families but also on communities, often inflicting great healthcare burdens. This thesis concentrates on one congenital disorder, Opitz Syndrome, which is a genetic disorder caused by mutations in *MID1*. Opitz Syndrome (OS) patients present an array of clinical features including some of the more commonly found congenital structural anomalies, such as cleft lip and palate and hypospadias. The information gained from an enhanced understanding of the important cellular and molecular processes and pathways involved in Opitz Syndrome will subsequently aid in the elucidation of the basis of the individual clinical features. Only through an increased understanding of the underlying mechanisms of these congenital malformations can advances be made in prevention, diagnosis and ultimately treatment of them.