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50 Years Ago in *The Journal of Pediatrics*

Familial Russell-Silver Syndrome

Fuleihan DS, Der Kaloustian VM, Najjar SM. The Russell–Silver syndrome: report of three siblings. J Pediatr 1971;78:654-7.

In 1971 Fuleihman et al reported 3 siblings (2 male) with clinical features of Russell–Silver syndrome. These features included low birth weight, short stature with craniofacial disproportion, triangular facies, high forehead, café au lait macules, and clinodactyly. Despite the parents' consanguineous relationship, an autosomal-dominant inheritance for Russell–Silver syndrome in this family appeared to be likely, as the mother of the siblings had short stature and facial features consistent with Russell–Silver syndrome.

We know now that Russell–Silver syndrome may be caused by uniparental maternal disomy for chromosome 7 and hypomethylation of the imprinting center (ICR, H19/IGF2:IGBMR) in 11p15.5 regulating the expression of imprinted genes H19 and IGF2.[1] Most cases of Russell–Silver syndrome occur sporadically. What molecular mechanisms would account for a 50% transmission risk for Russell–Silver syndrome to parental offspring? Maternal microdeletions and microduplications involving chromosome 7, chromosome abnormalities including unbalanced translocations involving 11p15.5, maternal microduplications including *CDKN1C*, and paternal microdeletions involving 11p15.5 are associated with Russell–Silver syndrome. CDKN1C is a maternally expressed cyclin-dependent kinase inhibitor that represses cell proliferation. Pathogenic *CDKN1C* variants inherited from female probands, *IGF2* pathogenic variants inherited from male probands, and *PLAG1* and *HMGA2* variants inherited from either parent are associated with the occurrence of Russell–Silver syndrome.[2] Although less likely, 3M syndrome, associated with severe short stature, macrocephaly, triangular facies, and skeletal features, is in the differential diagnosis for the case reported by Fuleihman et al. Inheritance is autosomal recessive, which can be observed in a consanguineous mating in which one partner is clinically affected with 3M syndrome and the other partner is a carrier for 3M syndrome.

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