

NAIL-PATELLA SYNDROME

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Medical Dictionary

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Carla Ann Brandon



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The Gale Encyclopedia of Children's Health Kristine M. Krapp, Jeffrey Wilson, 2005 Arranged alphabetically each volume provides in depth coverage of pediatric diseases and disorders along with issues related to physical and cognitive behavioral development Shingles, 2006 **Pemphigus** James N. Parker, Phillip M. Parker, 2004 Pressure Sores, 2004 **A Clinical and Molecular Study of Nail Patella Syndrome** Elizabeth Sweeney, University of Bristol, 2002

The Genetics of the Nail-patella Syndrome James H. Renwick, 1957 *Nail-Patella Syndrome*, Features information on Nail Patella Syndrome a pediatric multisystem disorder characterized by dystrophic nails hypoplastic or absent patellae and renal disease provided as part of the Pediatric Database of the University of Western Ontario Discusses epidemiology pathogenesis clinical features management and investigation Includes links to related sites **Dermatoglyphic Phenotype of Nail-patella Syndrome** Carla Ann Brandon, 2004 **Dictionary of Medical Syndromes** Sergio I. Magalini, Sabina C. Magalini, Giovanni De Francisci, 1990 Covers both eponymous and noneponymous syndromes Definitions include symptoms and signs etiology pathology diagnostic procedures therapy prognosis and a bibliography **Two Families of Nail-patella Syndrome with Novel Splice Site Mutations in the LMX1B Gene** Satoru Shinkuma, 2017 Nail patella syndrome NPS is characterized by dysplasia of the nail and bone abnormalities such as aplastic or hypoplastic patella frequently associated with nephropathy NPS is caused by heterozygous mutations in the LMX1B gene encoding a member of the LIM homeodomain protein family Gene analysis in NPS has so far disclosed that haploinsufficiency of LMX1B leads to the clinical manifestation of NPS In this study we evaluated two families with NPS Direct sequencing analysis revealed Family 1 and Family 2 were heterozygous for the splice site mutations c 140 1G C IVS1 1G C and c 326 1G C IVS2 1G C in LMX1B respectively To identify the mechanism of pathogenesis we performed reverse transcriptase PCR of cDNAs extracted from the dorsal skins of their thumbs which showed normal and shortened PCR products in both samples Intriguingly sequencing of the shortened PCR products revealed the same splice variant with deletion of exon 2 leading to a frameshift and a downstream premature termination codon p Asp49Serfs 18 Our data confirm that NPS is caused by haploinsufficiency of LMX1B

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