Analyses of Oligodontia Phenotypes and Genetic Etiologies

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Table S1.List of articles reviewed in this study with brief summaries of their specific findings.**List of Articles Analyzed in this Study.**

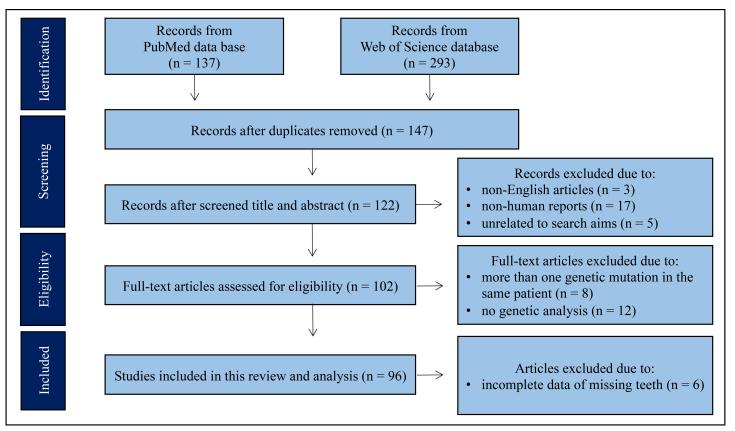


Figure S1. Flow chart of the article selection and review process.

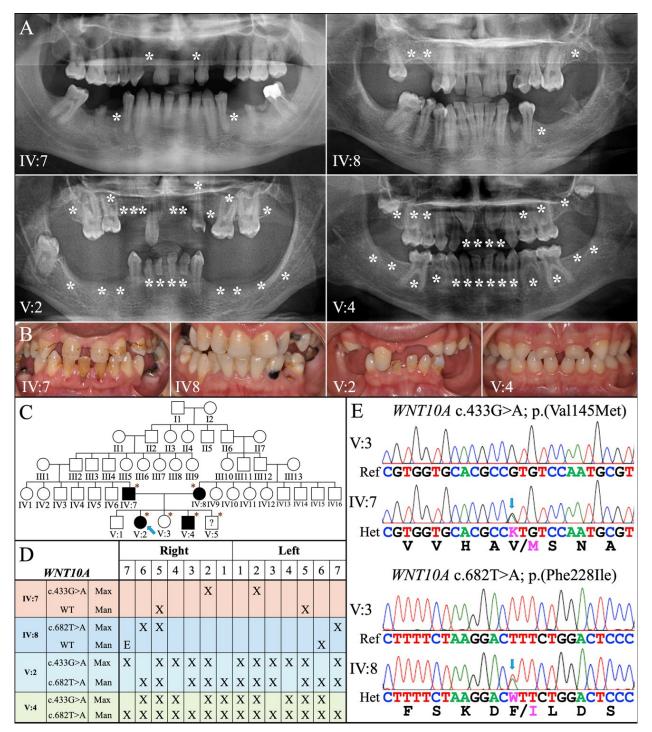


Figure S2. Oligodontia Family 2 from Turkey with Compound Heterozygous *WNT10A* defects c.433G>A, p.(Val145Met) and c.682T>A, p.(Phe228Ile). *A*: Panoramic radiographs with sites of tooth agenesis marked by stars. When the radiographs were taken, subjects IV:7 and IV:8 were adults and subjects were V:2 (16y) and V:4 (8y). *B*: Oral photographs of subjects. *C*: Pedigree of Family 2. Asterisks mark the 6 subjects who participated in the study. Their genotypes are IV:7 +/-, IV:8 +/-, V:2 -/-, V:3 +/+, V:4 -/-, and V:5 +/-. Affection statuses of non-participants areunknown. *D*: Summary chart of missing teeth (X, agenesis; E, extracted). The heterozygous parents (IV:7 and IV:8) were both missing 4 teeth. Offspring with biallelic *WNT10A* defects (V:2 and V:4) exhibited agenesis of 22 and 24 teeth, respectively and were included in the oligodontia analyses. Subject V:5 (3y) was heterozygous for the p.(Val145Met) variation only and was too young to determine affection status. Subjects V:2 and V:4 were included in the oligodontia analysis. *E: WNT10A* chromatograms showing that the father (IV:7) and mother (IV:8) were heterozygous (Het) for the *WNT10A* variations c.433G>A, p.(Val145Met) and c.682T>A, p.(Phe228Ile), respectively. Unaffected subject V:3 was homozygous for the *WNT10A* reference sequence (Ref). K = A or G; W = A or T. Sequences altered by mutation are in magenta. The NCBI reference sequence designations for these variants are NG_012179.1: g.14508G>A; NG_012179.1(WNT10A_v001): c.682T>A, p.(Phe228Ile).

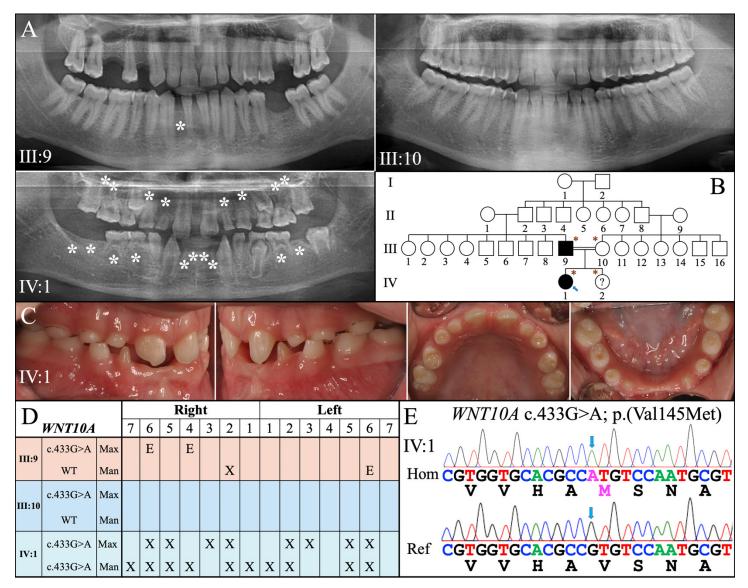


Figure S3. Oligodontia Family 3 from Turkey with Homozygous *WNT10A* defect: c.433G>A, p.(Val145Met). *A*: Panoramic radiographs with sites of tooth agenesis marked by stars. When the radiographs were taken, the parents (III:9 and III:10) were adults and the offspring (IV:1 and IV:2) were ages 10 and 4, respectively. *B*: Pedigree of Family 3. Asterisks mark the 4 subjects who participated in the study. Their genotypes are III:9 +/-, III:10 +/-, IV:1 -/-, and IV:2 +/-. Affection statuses of non-participants are unknown. *C*: Oral photographs of proband (IV:1) showing misshapened teeth and attrition of retained primary teeth. *D*: Summary chart of missing teeth (X, agenesis; E, extracted). The proband exhibited agenesis of 18 permanent teeth and was included in the oligodontia data analysis. Subject IV:2 was heterozygous for the *WNT10A* defect (not shown) and too young to determine affection status. *E*: The *WNT10A* chromatogram shows that the proband (IV:I) was homozygous for the *WNT10A* defect. The NCBI reference sequence designation for this *WNT10A* variant is NG 012179.1: g.14508G>A; NG 012179.1 (WNT10A v001): c.433G>A, p.(Val145Met).

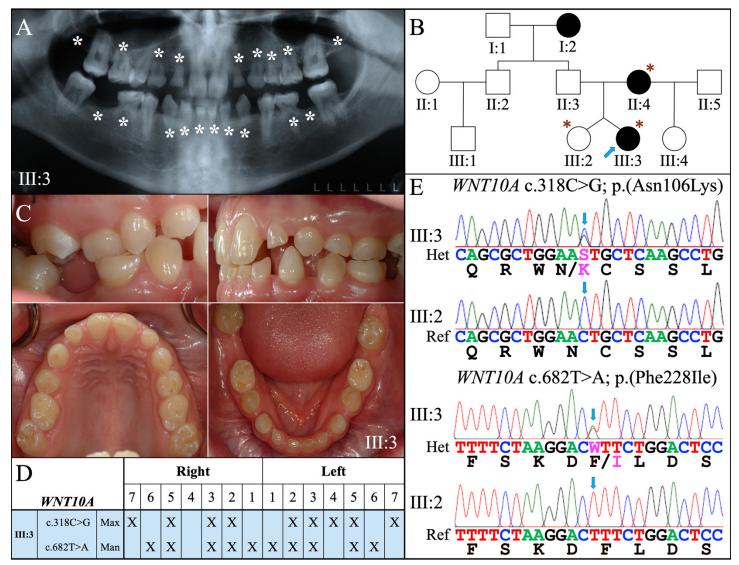


Figure S4. Oligodontia Family 4 with Compound Heterozygous *WNT10A* Defects c.318C>G, p.(Asn106Lys) and c.682T>A, p.(Phe228IIe). *A*: Panoramic radiograph of the proband (III:3) with sites of tooth agenesis marked by stars. No radiographs were available for the affected mother (II:4) who reported to have two maxillary lateral incisors absent. *B*: Pedigree of Family 4. Asterisks mark subjects recruited in this study. Their genotypes are II:4 +/-, III:2 +/+, and III:3 -/-. *C*: Oral photographs of the proband (III:3) who lacked *D*: Summary chart showing the sites of tooth agenesis (X). The proband (III:3) exhibited tooth agenesis of 18 permanent teeth and was included in the data analysis. *E*: The *WNT10A* chromatograms show that the proband (III:3) was a compound heterozygote for the *WNT10A* c.318C>G, p.(Asn106Lys) and c.682T>A, p.(Phe22IIe) defects, whereas her fraternal twin sister shows the reference sequence at both variant sites. Sequences altered by mutation are in magenta. S = G or C; W = A or T. The NCBI reference sequence designations for these *WNT10A* variants are NG_012179.1: g.6833C>G; NG_012179.1 (WNT10A_v001): c.682T>A, p.(Phe228IIe).

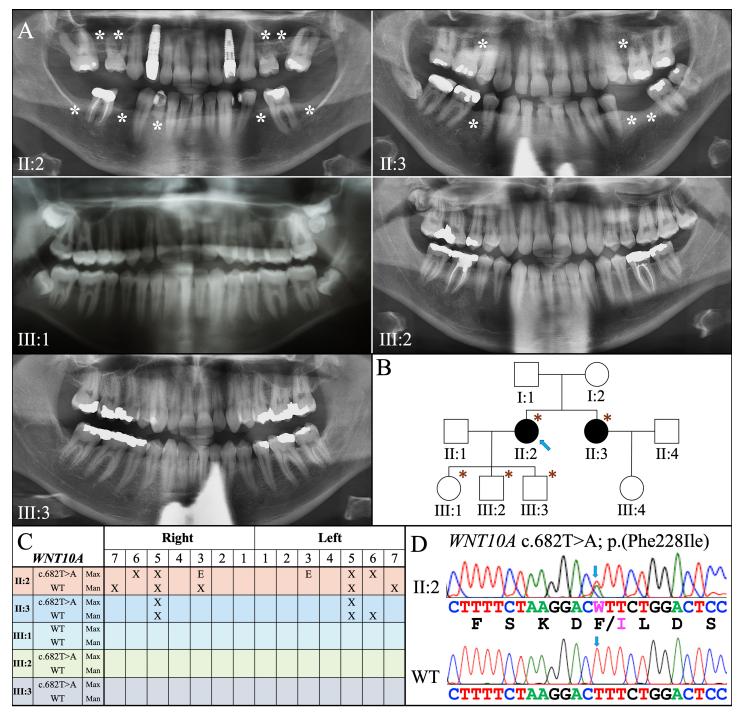


Figure S5. Oligodontia Family 5 with the Heterozygous *WNT10A* defect: c.682T>A, p.(Phe228IIe). *A:* Panoramic radiographs of Family 5 with sites of tooth agenesis marked by stars. *B:* Pedigree of Family 5. Asterisks mark the 5 subjects recruited in this study. Their genotypes are II:2 +/-, II:3 +/-, III:1 +/+, III:2 +/-, and III:3 +/-. *C:* Summary chart of missing teeth (X, agenesis;E, extracted). The proband (II:2) exhibited agenesis of 9 permanent teeth and was included in the oligodontia data analysis. *D:* The *WNT10A* chromatogram shows the proband (II:2) was heterozygous for the c.682T>A, p.(Phe228IIe) defect, as were subjects II:3 and III:3 (not shown). Sequences altered by mutation are in magenta. W = A or T. The NCBI reference sequence designations for this *WNT10A* variant are NG_012179.1: g.14757T>A; NG_012179.1(WNT10A_v001): c.682T>A, p.(Phe228IIe).

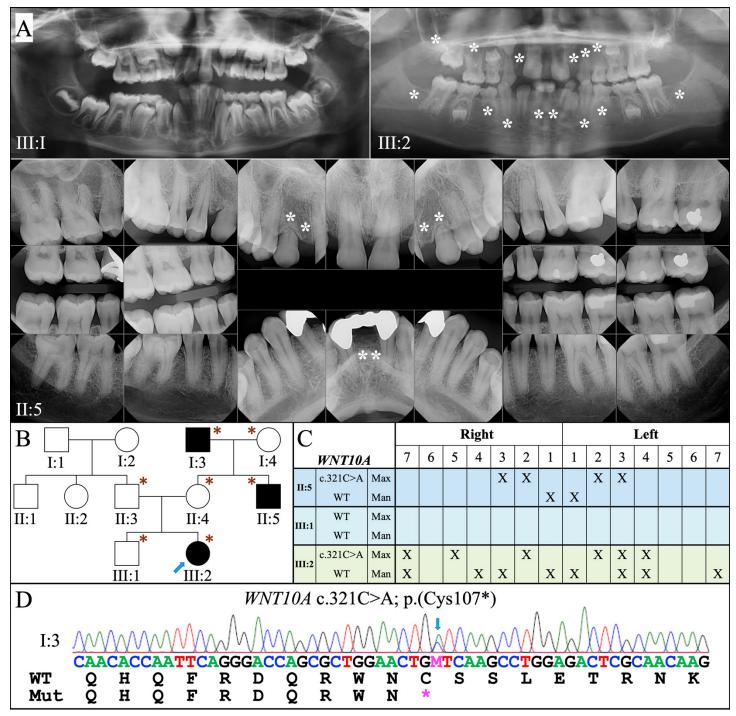


Figure S6. Oligodontia Family 6 with the Heterozygous *WNT10A* defect: c.321C>A, p.(Cys107*). *A*: Panoramic radiographs of Family 6 (III:1, 10 years old; and III:2, 7 years old) and anterior periapical radiographs of subject II:5 (adult) with sites of tooth agenesis marked by stars. *B*: Pedigree of Family 6. Asterisks mark the 7 subjects recruited in this study. Their genotypes are I:3 +/-, I:4 +/+, II:3 +/+, II:4 +/-, II:5 +/-, III:1 +/+ and III:2 +/- *C*: Summary chart showing the sites of tooth agenesis (X). The proband (III:2) lacked 14, and subject II:5 lacked 6 permanent teeth and both were included in the data analysis. *D*: Chromatogram from subject I:3 showing the heterozygous *WNT10A* c.321C>A, p.(Cys107*) defect that was also identified in subjects II:4, II:5, and the proband (III:2), although the number of missing teeth on subjects I:3 and II:4 could not be ascertained . Subjects I:4 and II:3 were wild-type for this *WNT10A* defect (not shown). Sequences altered by mutation are in magenta.M = A or C. The NCBI reference sequence designations for this *WNT10A* variant is NG_012179.1: g.6836C>A; NG_012179.1(WNT10A_v001): c.321C>A; NG_012179.1(WNT10A_i001): p.(Cys107*).

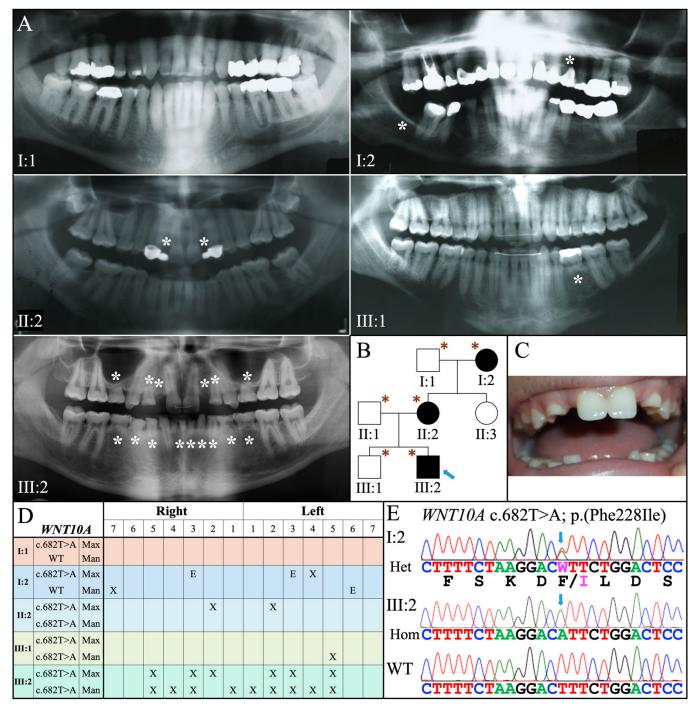


Figure S7. Oligodontia Family 7 with the Homozygous *WNT10A* defect: c.682T>A, p.(Phe228Ile). *A*: Panoramic radiographs of Family 7. A star marks the location of each absent (undeveloped) permanent tooth. *B*: Pedigree of Family 7. Asterisks mark subjects recruited in this study. Their genotypes are I:1 +/-, I:2 +/-, II:1 +/-, II:2 -/-, III:1 -/-, and III:2 -/-. *C*: Oral photo of proband (III:2) showing a lack of contour on the central incisors and attrition of the retained primary teeth. *D*: Summary chart of missing teeth (X, agenesis; E, extracted). Despite the fact that subjects I:1 and II:1 were heterozygous, and subjects I:2, III:1, and III:2 were homozygous for the c.682T>A, p.(Phe228Ile) defect, only the proband (III:2) showed oligodontia agenesis of 15 permanent teeth) and was included in the data analysis. *E*: Chromatograms showing the heterozygous (I:2), homozygous (III:2), and wild-type (II:1) sequences for the c.682T>A, p.(Phe228Ile) variation. Subjects I:1 and III:2 also carried a heterozygous *EDARADD* variation NM_145861.4: c.308C>T; p.(Ser103Phe) (rs114632254), which was previously described as a functional variant. [Salvi, A. et al.Mutation analysis by direct and whole exome sequencing in familial and sporadic tooth agenesis. *Int J Mol Med* **38**, 1338-1348, (2016)]. The NCBI reference sequence designations for this *WNT10A* variant are NG_012179.1: g.14757T>A; NG_012179.1(WNT10A_v001): c.682T>A, p.(Phe228Ile).

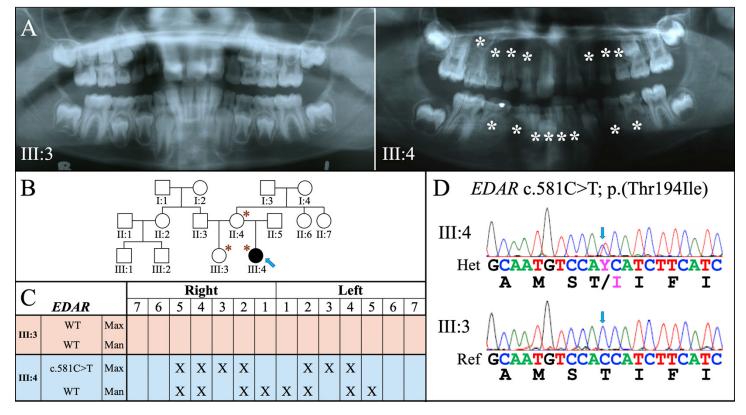


Figure S8. Oligodontia Family 8 with the Heterozygous EDAR defect: c.581C>T, p.(Thr194Ile). A: Radiographs of III:3 and III:4 in Family 8. At the time when the radiographs were taken, subjects III:3and III:4 were age 9 years 7 months and 8 years 2 months, respectively. B: Pedigree of Family 8. Asterisks mark the 3 subjects recruited in this study. Their genotypes are II:4 +/+, III:3 +/+, and III:4 +/-. C: Summary chart showing the sites of tooth agenesis (X) for subjects III:3 and III:4. The proband (III:4) lacked 15 permanent teeth and was included in the data analysis. D: Chromatograms of subjects III:4 and III:3. Sequences altered by mutation are in magenta. Y = T or C. The EDAR chromatograms showed that subject III:4 was heterozygous for the EDAR sequence variation and subject III:3 was wild-type (EDAR sequence was identical to the reference). The mother (II:4) was also wild-type (not shown). No other EDAR sequencevariations were observed. The NCBI reference designations this EDAR variant NG 008257.1: sequence for are g.83352C>T; NG 008257.1(EDAR v001): c.581C>T, p.(Thr194Ile).

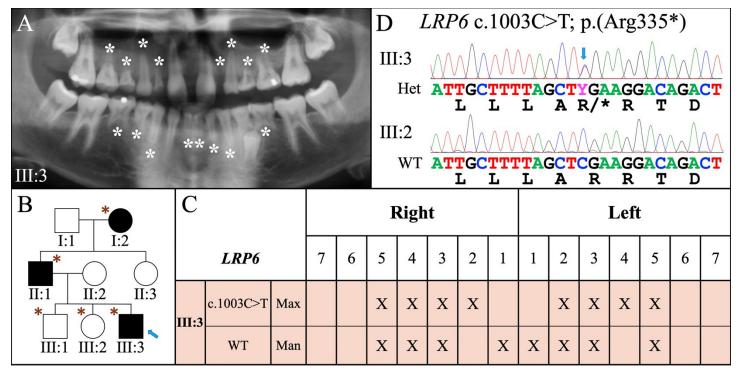


Figure S9. Oligodontia Family 9 Heterozygous with the *LRP6* defect: c.1003C>T, p.(Arg335*). *A*: Panoramic radiograph of the proband (III:3) taken at age 12 years 2 months. A star marks the location of each absent (undeveloped) permanent tooth. *B*: Pedigree of Family 9. Asterisks mark the 5 subjects recruited in this study. Their genotypes are I:2 +/-, II:1 +/-, III:1+/+, III:2 +/+, and III:3 +/-. The mandibular first molars of III:3 show taurodontism. *C*: Summary chart showing the sites of tooth agenesis (X) for the proband (III:3), who lacked 16 permanent teeth and was includedin the data analysis. Subjects I:2 and II:1 were reported to have tooth agenesis, and were heterozygous for the same *LRP6* defect as the proband (not shown), but no radiographs were provided to ascertain the extent of the tooth agenesis. *D*: The chromatogram shows the heterozygous *LRP6* stop gain variant, and the wild-type reference sequence from subject III:2. Sequences altered by mutation are in magenta. Y = T or C. Subject III:1 was also wild-type (not shown) and affected subjects I:2 and II:1 were heterozygous for the *LRP6* defect (not shown), but we were unable to ascertain their numbers of teeth missing due to agenesis. The NCBI reference sequence designations for this *LRP6* variant are (NG_016168.2: g.90465C>T; NG_016168.2(LRP6_v001): c.1003C>T, p.(Arg335*).

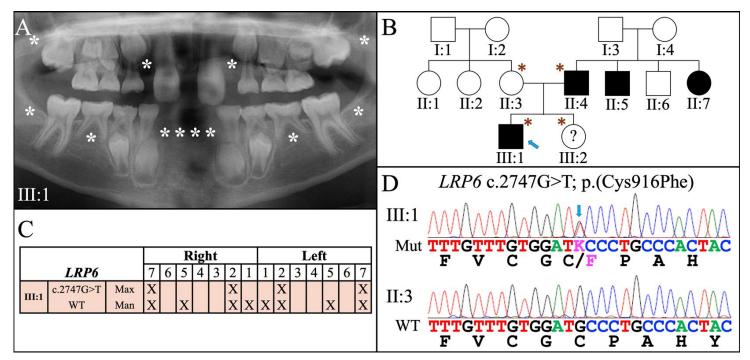


Figure S10. Oligodontia Family 10 Heterozygous with the *LRP6* defect: c.2747G>T, p.(Cys916Phe). *A*: Panoramic radiograph of the proband (III:1) taken at age 10 years. A star marks the location of each absent (undeveloped) permanent tooth. *B*: Pedigree of Family 10. Asterisks mark the 4 subjects recruited in this study. Their genotypes are II:3 +/+, II:4 +/-, III:1+/-, and III:2 +/-. *C*: Summary chart showing the sites of tooth agenesis (X) for the proband (III:1), who lacked 12 permanent teeth and was included in the data analysis. Subject II:4 was reported to have tooth agenesis, but no radiographs were provided, and subject III:2 (at age 3) was too young to assess agenesis of permanent teeth. Both of these subjects were heterozygous for the same *LRP6* defect as the proband (III:1) and reference sequence in subject II:3. Sequences altered by mutation are in magenta. K = T or G. The NCBI reference sequence designations for this *LRP6* variant are (NG 016168.2: g.113005G>T; NG 016168.2(LRP6 v001): c.2747G>T, p.(Cys916Phe).

Figure S11A Family 1 Chromatograms. *WNT10A* Exon 1: NG_012179.1:g.5562_5568dup; NM_025216.2:c.99_105dup; NP_079492.2:p.(Met36Cysfs*).

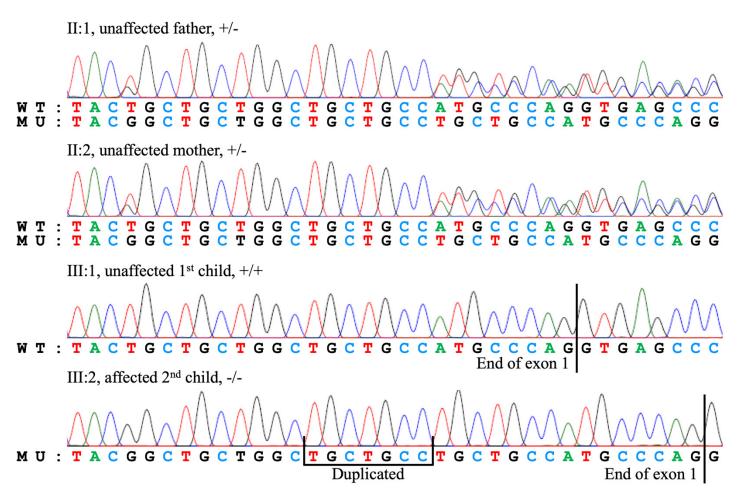


Figure S11B. Family 2 Chromatograms. *WNT10A* Exon 3: NG_012179.1:g.14508G>A; NM_025216.2:c.433G>A; NP_079492.2:p.(Val145Met)



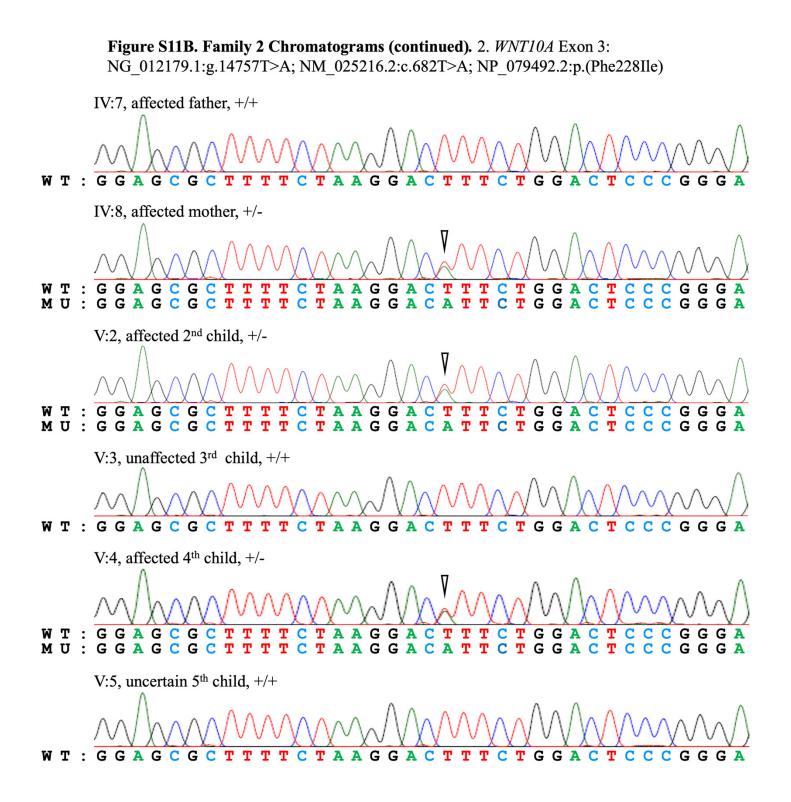


Figure S11C. Family 3 Chromatograms. *WNT10A* Exon 3: NG_012179.1:g.14508G>A; NM_025216.2:c.433G>A; NP_079492.2:p.(Val145Met)

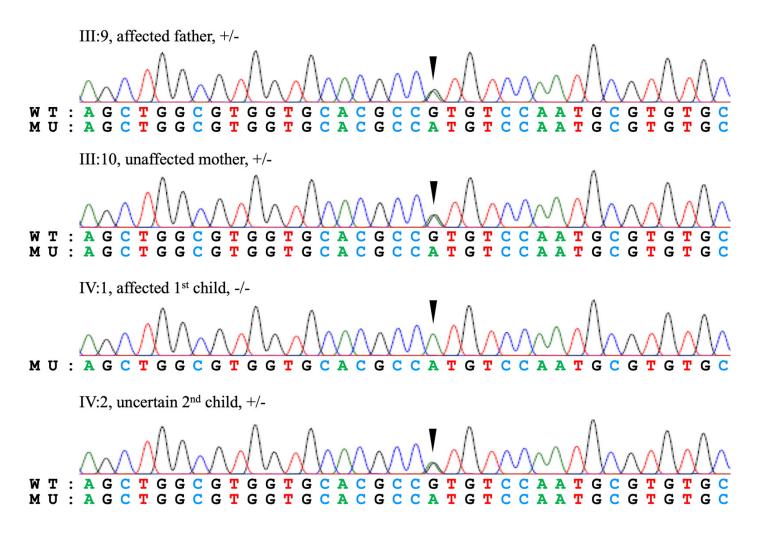
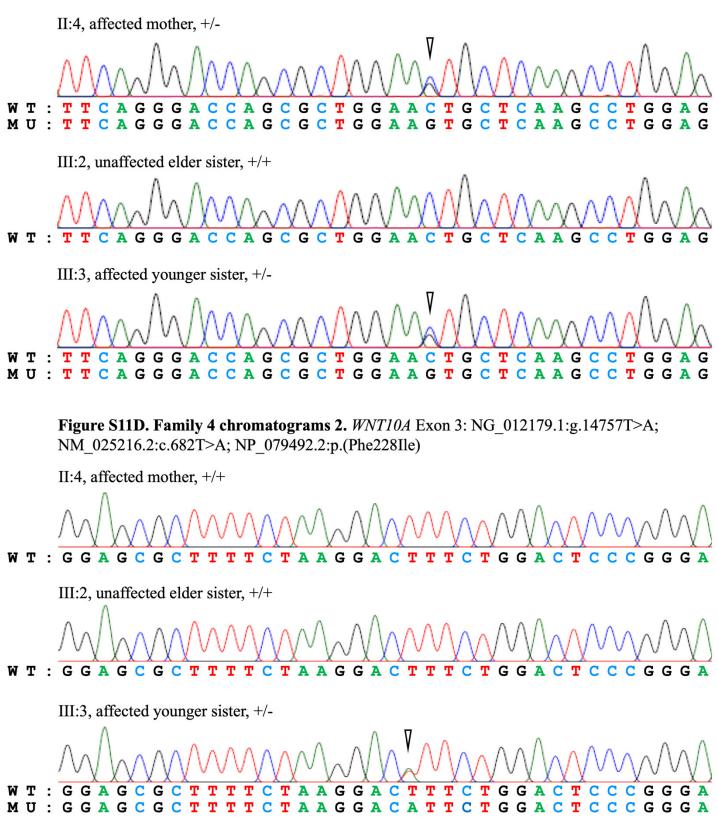
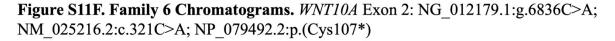


Figure S11D. Family 4 Chromatograms 1. *WNT10A* Exon 2: NG_012179.1:g.6833C>G; NM_025216.2:c.318C>G; NP_079492.2:p.(Asn106Lys)





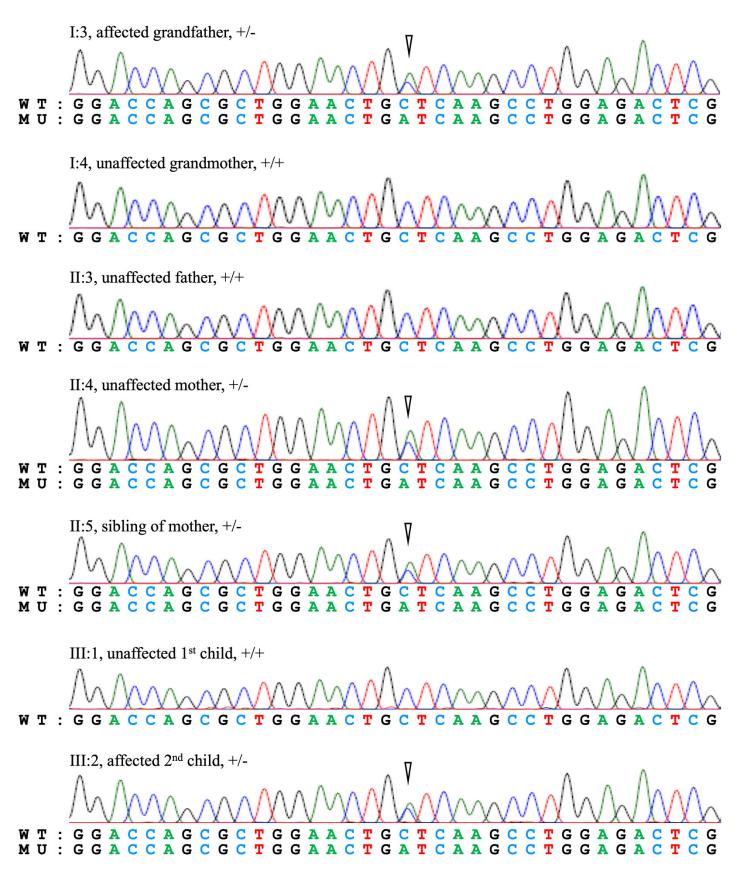


Figure S11G. Family 7 Chromatograms. *WNT10A* Exon 3: NG_012179.1:g.14757T>A; NM_025216.2:c.682T>A; NP_079492.2:p.(Phe228Ile)

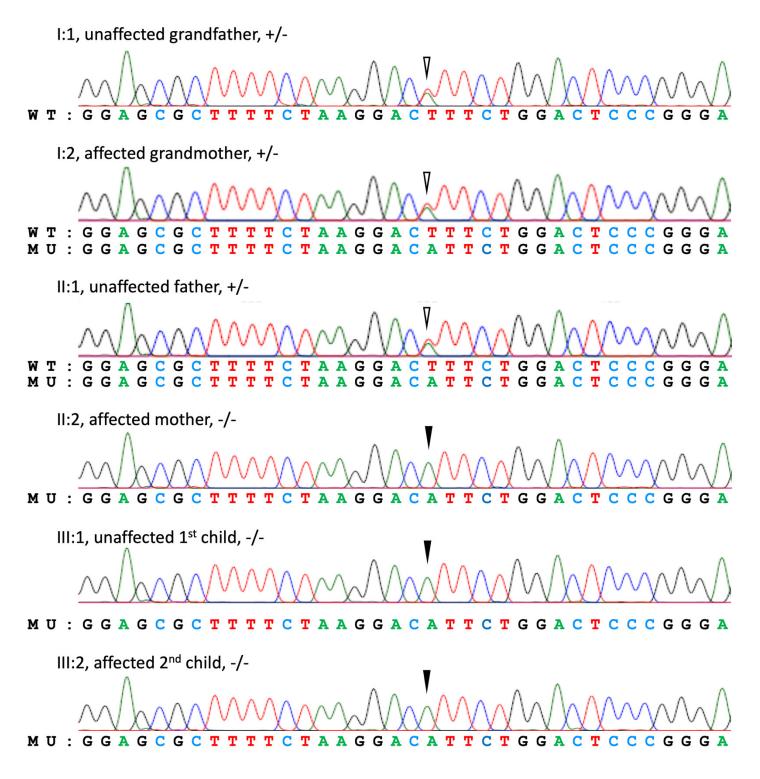


Figure S11H. Family 8 Chromatograms. *EDAR* Exon 7: NG_008257.1:g.83352C>T; NM_022336.3:c.581C>T; NP_071731.1:p.(Thr194Ile)

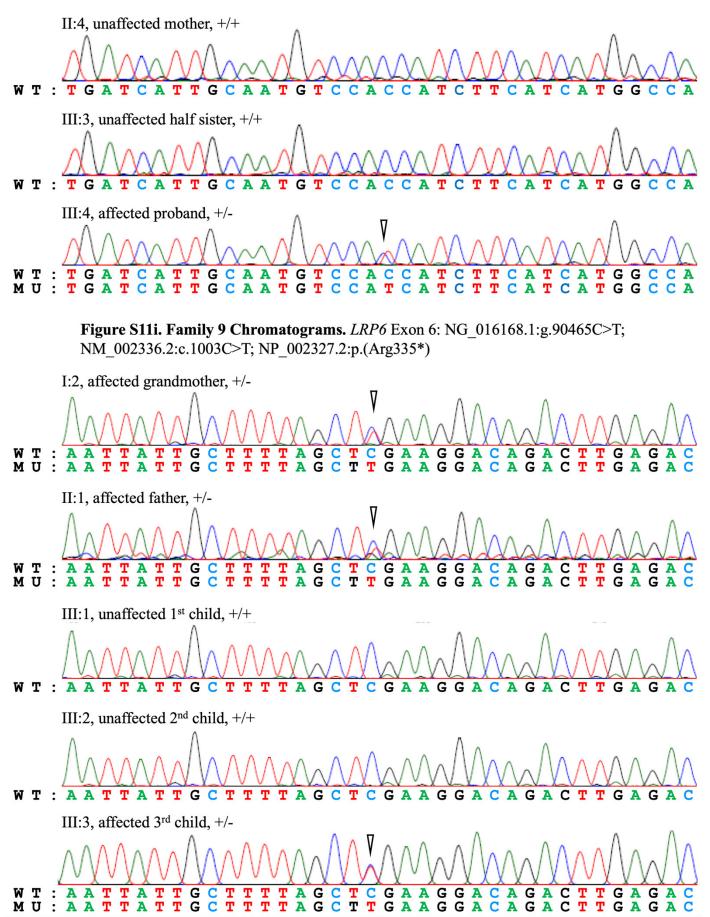


Figure S11J. Family 10 Chromatograms. *LRP6* Exon 12: NG_016168.1:g.113005G>T; NM_002336.2:c.2747G>T; NP_002327.2:p.(Cys916Phe)

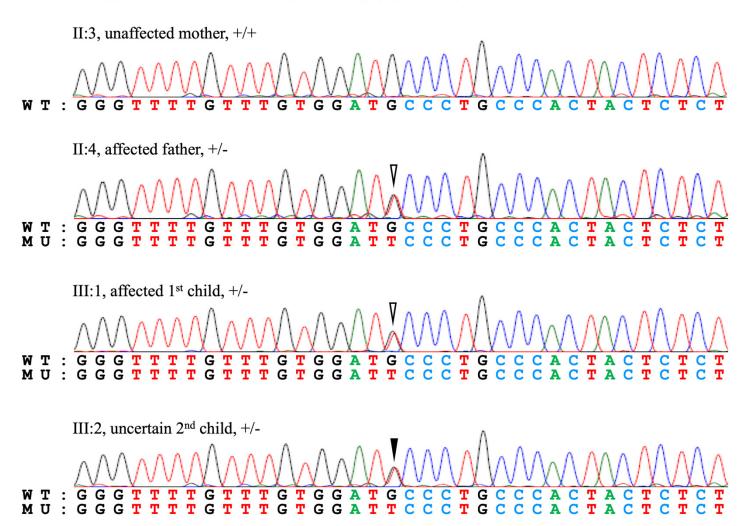


Table S1. List of articles reviewed in this study with brief summaries of their specific findings.

Article	Year	Patients' region	Gene name	Number of cases	Mo2	Mol	PM2	PMI	Ca	Ш	CI	Mo2	Mol P	M2 PN	MI	Ca	u	CI	syndromic phenotype	
Functional analysis of a novel missense mutation in AXIN2 associated with non-syndromic tooth	2016	China	AXIN2	1	0	0	2	2	0	0	0	0	_	2 1	_	-	_	2	1 	
agenesis Axis inhibition protein 2 polymorphisms may be a risk factor for families with isolated oligodontia	2010	China	AXIN2 AXIN2	2	2	0	4	3	4	3	0	2					2	2	1	
A Novel AXIN2 Missense Mutation Is Associated with Non-Syndromic Oligodontia	2015	China	AXIN2	ĩ	0	0	2	2	0	0	0	0		2 1	1		0	0	<i>I</i>	
Novel missense mutations in the AXIN2 gene associated with non-syndromic oligodontia Isolated Oligodontia Associated With Mutations in EDARADD, AXIN2, MSX1, and PAX9 Genes	2014 2011	China Sweden	AXIN2 AXIN2	2 3	0	0	4	3	1	4	2	0	0		2	1	3	2 4	/	
AXIN2-Associated Autosomal Dominant Ectodermal Dysplasia and Neoplastic Syndrome	2011	USA	AXIN2	1	2	0	0	0	2	0	0	2	0	0 0	0	1	2	2	sparse eyebrows, slightly upslanting palpebral fissures, and thin upper vermillion;multiple fundic gland	Ectodermal Dysplasia and Neoplas Syndrome
Phenotypic confirmation of oligodontia, colorectal polyposis and cancer in a family carrying an exon 7	2019	Australia	AXIN2	2	4	2	2	2	2	2	2	0	3	1 3				2		Syndrome
nonsense variant in the AXIN2 gene Mutations in AXIN2 Cause Familial Tooth Agenesis and Predispose to Colorectal Cancer	2019	Finnish	AXIN2 AXIN2	2	4	2	2	2	4	2	2	0		1 3				2	colorectal nooplasia	
Mutations in AXIN2 Cause Familial Tooth Agenesis and Predispose to Colorectal Cancer	2004	Finnish	AXIN2	4	5	4	8	5	2	8	0	3		8 2		3	7	7	/	
Targeted next-generation sequencing (NGS) analysis of mutations in nonsyndromic tooth agenesis candidate genes	2021	Turkey	AXIN2	1	0	0	2	0	0	2	0	0	0	2 (0	0	0	0	/	
unnymatenase novan agonese canzanar gunes A new Hypo/Oligodomia Syndrome: Carvaja/nazos Syndrome Secondary to Desmoplakin-dominant Mutations	2011	France	DSP	1	2	0	2	0	2	1	0	2	0	2 0	0	0	0	0	weelly hair, palmoplantar koratederma, and dilated cardiemyopathy	Carvajal/Naxos syndrome
Peleterious Variants in WNT10A, EDAR, and EDA Causing Isolated and Syndromic Tooth Agenesis: A Structural Perspective from MolecularDynamics Simulations	2019	Pakistan, Egypt, Saudi Arabia,	EDA	1	0	0	0	0	0	2	0	0	0	2 0	0	0	2	2	i.	
Whole Genome Sequencing Reveals Novel Non- Synonymous Mutation in Ectodysplasin A (EDA)	2014	and Syria Indian	EDA	3	2	0	2	2				0		3 3		5	6	6	/	
Associated with Non-Syndremic X-Linked Dominant Congenital Tooth Agenesis Oligodontia and early Hair Occur with Ectodysplasin-A Mutations	2014	koren	EDA	1	1	0	6	2	6	6	2	3	2	6	4	2	6	4		
Non-syndromic Tooth Agenesis Associated with a Nonsense Mutation in Ectodysplasin-A (EDA)	2013	Sweden	EDA	2	0	0	0	0	3	3	0	0	0	1 (0	0	4	4	/	
Candidate Gene Analysis of Tooth Agenesis Identifies Novel Mutations in Six Genes and Suggests Significant Role for WNT and EDA Signaling and Allele Combinations	2013	Finland	EDA	3	0	0	2	5	2	6	0	0	0	1 3	2	0	6	4	/	
EDA Gene Mutations Underlie Non-syndromic Oligodontia	2009	China	EDA	4	2	0	6	6	6	8	1	1	0	6 6	6	3	6	5	/	
Novel mutations identified in patients with tooth agenesis by whole - exome sequencing	2019	China	EDA	1	2	2	2	2	2	2	2	2	2	2 2	2	2	2	2	thin or wrinkled skin sparse or curly hair Hypohidrosis	Ectodermal Dysplasia
Novel missense mutation in the EDA gene in a family affected by oligodontia	2016	Germany	EDA	2	0	0	0	2	2	4	0	0	0	1 1	1	0	4	4	one patient presents sparse, wiry hair. The other one presents an abnormal ear shape and sparse, wiry hair	Ectodermal Dysplasia
Missense mutation of the EDA gene in a Jeedunian family with X-linked hypohiskosic ectodermal dysplasic phenotypic appearance and speech problems	2010	Jordanian	EDA	-1	2	2	0	0	0	2	0	2	1	2 1	2	2	0	2	heat intolerance, sparse hair (hypotrichosis);speech problems, and damaged eccrine glands, resulting in reduced sweating (anhidrosis)	
A novel EDAR missense mutation identified by whole-ecome sequencing with non-syndromic tooth agenesis in a Chinese family	2021	China	EDA	3	0	0	4	0	1	6	0	0	0	6 1	1	0	6	6	· · · · · · · · · · · · · · · · · · ·	
Comparative analysis of rare EDAR mutations and tooth agenesis pattern in EDAR - and EDA - associated nonsyndromic	2020	China	EDAR	8	5	4	9	12	8	7	0	4	2		5	4	9	6	1	
oligodentia																				
Novel EDAR mutation in tooth agenesis and variable associated features Candidate Gene Analysis of Tooth Agenesis Identifies Novel Mutations in Six Genes and Suggests	2020	Turkey	EDAR	2	0	0	0	0	0	4	0	0						1	/	
Significant Role for WNT and EDA Signaling and Allele Combinations	2013	Finland	EDAR	3	0	0	4	1	4	6	0	0	0	4 3	2	0	4	0	<u>/</u>	
A novel EDAR missense mutation identified by whole-exome sequencing with non-syndromic tooth agenesis in a Chinese family	2021	China	EDAR	1	2	2	2	0	1	1	2	2	1	2 (0	0	1	2	/	
Candidate Gene Analysis of Tooth Agenesis Identifies Novel Mutations in Six Genes and Suggests	2013	Finland	EDARADD	4	0	0		4		4	0	3	0	8 .		0	0	0	1	
Significant Role for WNT and EDA Signaling and Allele Combinations Isolated Oligodontia Associated With Mutations in EDARADD, AXIN2, MSX1, and PAX9 Genes	2013	Sweden	EDARADD	4	0	0	0	4	3	4	0							0	1	
	2011	Sweden	EDARADD	1	0	0	2	0	0	0	0	2	0	2 (0	0	0	0	'	
Whole-Exome Sequencing Identifies Novel Variants for Tooth Agenesis	2018	Turkey	KREMEN1	1	2	0	0	2	0	2	0	1	0	1	1	1	2	2	í.	
Whole-Exome Sequencing Identifies Novel Variants for Toeth Agenesis	2018	Turkey	KREMEN1	2	4	0	2	4	2	4	0	3	0	3	3	3	4	4	mild clinical findings of ectodermal dysplasia (sparse hair, dry skin, sparse cycleows and cyclashes, pro_x005f_x0002_truded lips, and heat intolerance)	Ectodermal Dysplasia
tutation of KREMEN1, a modulator of Witt signaling, is responsible for extodermal dysplasia including oligodomin in Palestinian families	2016	Palestine	KREMEN1	9	2	0	0	2	2	18	2	2	0	2	2	16	18	18	facial forures include abovemb law distribution of the scalp, low hairline with forehead fazzness, broad and low nose bridge, columella extending with age, thick lips, slight ocult hypertections, and down and sating of the paipebral fissures.	Ectodermal Dysplasia
luation of KREMEN1, a modulator of War signafing, is responsible for ectodernal dysplasia including oligodeonia in Palestiniant families Lrp6 Dynamic Expression in Tooth	2016	Palestine	KREMEN1	3	0	0	0	0	0	8	0			0 0				8	,	1
Development and Mutations in Oligodontia	2020	China	LRP6	4	4	0	7	5	7	6	0	6	1	6 1	1	1	3	2	<u>(</u>	
Whole-Exome Sequencing Identifies Novel Variants for Toeth Agenesis Loss-of-Function Mutations in the WNT Co-receptor LRP6 Cause Autosomal-Dominant Oligodontia	2018	Turkish	LRP6	1	0	0	2	2	0	2	0	0	0	2 2	2	2	2	2	/	
Lipó Dynamic Expression in Tooth Development and Mutations in Oligodontia	2015	Netherland	LRP6	4	2	2	2	2	0	2	0	0	0	2 2	2	0	2	2	/ sparse hair and hypoblidrosis	Ectodermal Dysplasia
oncurrent manifestation of oligodontia and thremhocytopenia caused by a contiguous gene deletion in 12p13.2: A three-generation clinical report	2019	Netherlands	LRP6	3	0	0	0	0	6	6	0	0	0	0 0	0	5	5	6	r -	
A novel missense mutation of LRP6 identified by whole-exome sequencing in a Chinese family with non-syndromic tooth agenesis	2021	China	LRP6	1	2	0	2	2	0	2	0	2	0	2	2	0	1	2	1	
Two novel mutations in MSX1 causing oligodontia	2020	China	MSX1	2	4	0	3	4	2	2	2	4	0	2 2	2	1	2	2	<i>i</i>	
A novel mutation of MSX1 inherited from maternal mosaicism causes a severely affected child with nonsyndromic oligodontia	2020	China	MSX1	1	0	0	2	2	0	0	0	1	2	2 0	0	2	0	2		
a novel mutation of MSX1 in oligodontia inhibits odontogenesis of dental pulp stem cells via the ERK	2018	China	MSX1	5	0	0	12	10	0	1	0	0	,	10	0	0	0	0	1	
pathway Next generation sequencing reveals a novel nonsense mutation in MSX1 gene related to oligodontia	2018	GERMANY	MSX1 MSX1	2	2	0	12 A	4	0	4	0	2	0	4 4	0			2	· /	
a novel MSX1 intronic mutation associated with autosomal dominant non- syndromic oligodontia in a	2018	China	MSXI	7		4	5	10	4	1	3		6	7	7			4	1	
large Chinese family pedigree Mutations in MSX1, PAX9 and MMP20 genes in Saudi Arabian patients with tooth agenesis	2016	Saudi Arabian	MSX1 MSX1	2	0	0	4	4	0	0	0	0	0	4 4	2	0	0	0		
an Aberrant Splice Acceptor Site Due to a Novel Intronic Nucleotide Substitution in MSX1 Gene Is the	2016	Janpan	MSX1 MSX1	3	2	0	6	1	1	1	1	4	3	4	1	1	1	3	1	
Cause of Congenital Tooth Agenesis in a Japanese Family Characterization of Novel MSX1 Mutations Identified in Japanese Patients with Nonsyndromic Tooth	2013	Japan	MSXI	3	1	2	4	4	1	2	2	0	2	6 6	6	2		2	r /	
Agenesis A novel non-stop mutation in MSX1 causing autosomal dominant non-syndromic oligodontia	2014	China	MSX1	1	0	0	2	2	1	2	1	2	0	2 (0	1	1	2	1	
ovel nensense mutation in MSX1 in familial nonsyndromic eligodontia: subcellular localization and role of homeodomain/MH4	2014	Japan	MSX1	2	2	0	4	4	0	0	0	2	2	4 0	0			4	/	
Clinical and genetic evaluation of a Chinese family with isolated oligodontia	2013	China	MSX1	2	0	0	4	3	1	4	0		0					4	1	
Candidate Gene Analysis of Tooth Agenesis Identifies Novel Mutations in Six Genes and Suggests Significant Role for WNT and EDA Signaling and Allele Combinations	2013	Finland	MSX1	2	0	0	4	4	0				0					4	/	
solated Oligodontia Associated With Mutations in EDARADD, AXIN2, MSX1, and PAX9 Genes	2011	Sweden	MSX1	2	2	2	4	3	0	3	0	2	2	4 0	0	0	1	2		
Mutations in the Human Homeobox MSX1 Gene in the Congenital Lack of Permanent Teeth leutification of a novel missense mutation of MSX1 gene in Chinese family with autosomal-dominant	2009	Poland	MSX1	2	0	0	2	0	0	2	0	0	0	4 3	2	0	0	2	1	
Intrification of a novel missense mutation of MSX1 gene in Chinese family with autosomal-dominant objecteria A novel c.581C>T transition localized in a highly conserved homeobox sequence of MSX1: is it	2008	Chinese	MSX1	2	2	0	1	0	3	4	2	0	0	4 0	0	2		2	<u> </u>	
A novel c.581C>T transition localized in a highly conserved homeobox sequence of MSX1: is it responsible for oligedontia? Novel MSX1 Frameshift Causes Autosomal-dominant Oligodontia	2006	Poland	MSX1	1	0	0	1						0					2	/	
rover MSAT Prantstant Causes Autosomar-asimati Ongodenna A novel missense mutation in MSX1 underfles autosomal recessive oligodontia with associated dental anomalies	2006	Korea	MSX1	2	0	0	4	2					0			0		0	· · · · · · · · · · · · · · · · · · ·	
in Pakistani families																				Wolf-Hirschhorn syndrome
MSX1 Gene is Deleted in Wolf-Hirschhorn Syndrome Patients with Oligodoatia Targetet next-generation sequencing (NGS) analysis of mutations in programmedomic teach incomercia candidate as non-	2003	Finish Turkey	MSX1 MSX1	5	5	4	10	9	0									6 2	mental and growth returdation, seizures, and hypospudia /	
nonsyndromic tooth agenesis candidate genes Novel MSX1 variants identified in families with nonsyndromic	2021	China	MSX1	8				14										11		
oligodentia	2021	Cama	212.4.1			4	10	- 14		· ·	. 0	10			~ I	· .			1	

(Continued on next page)

Functional study of novel PAX9 variants: The paired domain and non-syndromic oligodontia																			
	2020	China	PAX9	3	6	4	2	5	4	3	2	6 1	5	0	0	0	4	1	
Familial oligedontia and regional odontodysplasia associated with a PAX9 initiation codon mutation Nine Novel PAX9 Mutations and a Distinct Tooth Agenesis Genotype-Phenotype	2019 2018	Finland	PAX9 PAX9	4	7	6	6	4	2	4	1	8 4	4	2	0	0	4		T.
A novel G to A transition at initiation codon and exon-intron boundary of	2018	China Indian	PAX9 PAX9	10	19	20	91	1		2	4		3	0	4	0	5		I. I.
PAX9 identified in association with familial isolated eligodontia Characterization of PAX9 variant P20L identified in a Japanese family with tooth agenesis	2017	Janpan	PAX9	2	4	4	3	0	0	0	0	4 4	0	0	0	0	0	I	
A novel PAX9 mutation causing oligodontia	2017	Malta	PAX9	1	2	2	0	0	0	0	0	2	0	0	0	0	2	/	
Mutations in WNT10B Are Identified in Individuals with Oligodontia A novel initiation codon mutation of PAX9 in a family with oligodontia	2016 2015	China China	PAX9 PAX9	2 5	4	4 8	2 6	2	2	2	0 4	4 :	2	0	0	0	2		
A Nonsyndromic Autosomal Dominant Oligodontia with A Novel Mutation of Pax9-A Clinical and	2015	Indian	PAX9	2	2	0	2	0	3	4	0	2 0	3	0	4	2	2	i i	
Genetic Report A screen of a large Czech cohort of oligodontia patients implicates a novel	2015	Czech	PAX9	3	6	6	6	0	0	4	0	6	2	0	0	0	1	1	
Novel PAX9 mutations cause non-syndromic tooth agenesis	2014	Japan	PAX9	2	4	4	2	0	0	0	0	4 0	0	0	0	0	2	1	
Novel missense mutation in PAX9 gene associated with familial tooth agenesis	2012	Brazil	PAX9	3	1	0	-4	5	2	0	0	0 0	5	4	0	0	-1	1	
Candidate Gene Analysis of Tooth Agenesis Identifies Novel Mutations in Six Genes and Suggests Significant Role for WNT and EDA Signaling and Allele Combinations	2013	Finland	PAX9	2	4	2	2	0	0	0	0	2 4	2	0	0	0	2	/	1
Novel missense mutations in PAX9 causing oligodentia	2012	China	PAX9	4	8	8	2	0	7	1	0	7	0	0	2	0	1		1
Sequence analysis of PAX9, MSX1 and AXIN2 genes in a Chinese oligodontia family	2011	China	PAX9	1	0	0	0	0	2	2	0	0	0	0	2	2	2	1	
Isolated Oligodontia Associated With Mutations in EDARADD, AXIN2, MSX1, and PAX9 Genes Mutations in the PAX9 gene in sporadic oligodontia	2011	Sweden	PAX9 PAX9	4	6	4	6	4	2	1	0	6 :	5	2	0	0	1		1
Identification and Functional Analysis of Two Novel PAX9 Mutations	2010 2009	Poland	PAX9 PAX9	6	1	4	1	1	0	4	0	2	1	7	4	0	0	1	
Identification of a novel mutation in the PAX9 gene in a family affected by oligodontia and other dental		China			2	2	0	2	0	0	0	2	0	0	0				
anomalies	2007	Spain	PAX9	4	8	8	5	2	0	0	0	8 :	2	0	0	0	0	1	
A novel nonsense mutation in PAX9 is associated with marked variability in number of missing teeth	2007	Denmark	PAX9	3	6	4	5	1	1	0	0	6 1	4	0	0	2	3	/	
A novel mutation in PAX9 causes familial form of molar eligodontia	2006	Poland	PAX9	5	10	10	10	0	0	0	0	10 1	0 10	0	0	0	10	1	
Molecular characterization of a novel PAX9 missense mutation causing posterior tooth agenesis Novel Mutation	2006	USA	PAX9	2	4	4	4	0	0	0	0	3 4	4	0	0	0	0	/	
of the Initiation Codon of PAX9 Causes Oligodontia	2005	Chinese	PAX9	2	4	4	4	2	2	0	0	4 4	4	0	0	0	2	/	
A novel missense mutation in the paired domain of PAX9 causes non-syndromic oligodontia. Novel mutation in the paired box	2004	USA	PAX9	1	2	2	2	0	2	0	0	2	0	0	0	0	2	/	
sequence of PAX9 gene in a sporadic form of oligodontia A missense mutation in PAX9 in a family with distinct phenotype of oligodontia	2003 2003	Poland Finland	PAX9 PAX9	1	0	0	2	0	1	2	0	0 0	2	0	0	1	1	/	
Mutational Analysis of Families Affected with Molar Oligodentia	2002	USA	PAX9	8	8	4	14	0	4	2	0	8 1	6) 14	6	0	0	4	1	
Identification of a nonsense mutation in the PAX9 gene in molar oligodontia Novel PAX9 and COL1A2 Missense Mutations Causing Tooth Agenesis and Ol/DGI without Skeletal	2001	Finland	PAX9	5	10	7	8	4	4	10	0	9 (5	2	4	3	5	1	
Novel PAX9 and COL1A2 Missense Mutations Causing Tooth Agenesis and OL/DGI without Skeletal Abnormalities	2012	European	PAX9	1	2	0	2	1	0	0	0	2 (1	0	0	0	2	1	
Intragenic duplication a novel causative mechanism for SATB2-associated syndrome	2014	Sweden	SATB2	1	0	0	2	0	2	0	0		2	0	2	2	0	intellectual disability, speech and language impairment, cleft palate, malformed teeth, and oligodontia.	SATB2-Associated Syndrome : moderat severe intellectual disability (ID)
					-		-		-						-			and the second	Lourany (10)
Recessive ofigodontia linked to a homozygous loss-of- function mutation in the SMOC2 gene Homozygosity Mapping and Candidate Prioritization Identify Mutations, Missed by Whole-Exome	2013	Pakistan	SMOC2	3	0	0	6	1	4	2		0 0		6	6	6	0		
Sequencing, in SMOC2, Causing Major Dental Developmental Defects	2011		SMOC2	2	0	0	4	0	0	0	0	0 0	4	3	4	2	0	1	
Targeted next-generation sequencing (NGS) analysis of mutations in	2021	Turkey	SMOC2		2	0	2	0			0	, I.,			0	0	0	1	
nonsyndromic tooth agenesis candidate genes	2021	Turkey	SMOC2		2	0	2	0	0	0	0	· ['	2	1	U	U	0	0	
Novel TSPEAR mutations in non-syndromic oligodontia	2020	Korea	TSPEAR	1	1	2	0	0	0	2	0	0 0	0	0	0	0	2	1	
TSPEAR variants are primarily associated with ectodermal dysplasia	2021	Canada,USA,Fra	TSPEAR	3	4	2	2	4	4	6	0	,	,	2	4	6	6	patient#17:dry skin, eczema, taurodontism; patient#18:scoliosis; patient#15: attention disorder, large	ectodermal draplasia
and tooth agenesis but not hearing loss: A novel cohort study		nce				0.50	- C			1		- I - I	1			- C	1	anteverted ears, prognathism, speech difficulties	
Actiological Evaluation of Oligodontia in a Three-Generation Family	2020	Turkey	WNT10A	2	1	0	2	1	0				2		0	0	2		
Novel mutations identified in patients with tooth agenesis by whole - exome sequencing	2019	China	WNT10A	1	2	0	1	2	2	2	0	2 (0	0	2	2	2	7	
Deleterious Variants in WNT10A, EDAR, and EDA Causing Isolated and Syndromic Tooth Agenesis: A	2019	Pakistan, Egypt,	WNT10A	1	2	0	2	2	1	2	0	2	1	0	2	2	2	1	
Structural Perspective from MolecularDynamics Simulations		Saudi Arabia,																	
Whole-Exome Sequencing Identifies Novel Variants for Toeth Agenesis	2018	Turkish	WNT10A	5	8	2	6	7	8	9	0	9	8	7	7	8	10	7	
Role of WNT10A in failure of tooth development in humans and zebrafish Dental and extra-oral clinical features in 41 patients with WNT10A gene mutations: A multicentric	2017	USA	WNT10A	1	0	2	2	2	1	0	0	0 3	1	0	0	0	1	/	
genotype-phenotype study	2017	French	WNT10A	1	0	0	2	2	0	0	0	0 0	2	2	0	0	0	6	
WNT10A mutations account for ½ of population-based isolated oligodontia and show phenotypic correlations.	2013	Sweden.	WNT10A	28	10	15	48	30	4	24	0	12 1	42	27	3	6	14	1	
Candidate Gene Analysis of Tooth Agenesis Identifies Novel Mutations in Six Genes and Suggests	2013	Finland	WNT10A	25	10	0	47	34	15	28	0	13 1	42	20	9	13	16	1	
Cananame Gene Analysis of 100th Agenesis identifies Novel Mutations in Six Genes and Suggests																			
Significant Role for WNT and EDA Signaling and Allele Combinations												1 0	0	0	2	2	2	hypotrichosis, sparse hair, eczema	HED (Hypohidrotic Ectodermal Dysplay
Candidate Gene Analysis of Tooth Agencisis Identifies Novel Mutations in Six Genes and Suggests Significant Role for WNT and EDA Signaling and Allele Combinations Eight Mutations of Three Genes (EDA, EDAR, and WNT10A) Identified in Seven Hypohideotic Ecidemul Dysplasis Parients	2016	China	WNT10A	1	2	0	2	0	2	2	0								
Significant Role for WNT and EDA Signaling and Allele Combinations Eight Mutations of Three Genes (EDA, EDAR, and WNT10A) Identified in Seven Hypohidrotic Ectodermal Dysplassis Patients																	-	onychodysplasia, palmoplantar hyperkeratosis, dry skin, hypotrichosis, and hyperhidrosis of the palms	
Significant Role for WNT and EDA Signaling and Allele Combinations Eight Mutations of Three Genes (EDA, EDAR, and WNT10A) Identified in Seven Hypohidrotic	2016 2016	China Denmark	WNT10A WNT10A	1	2		2				0	2	2	2	2	2	2	onychedysplasia, palmoplantar hyperkeratosis, dry skin, hypotrichosis, and hyperhidrosis of the palms and soles	
Significant Flok for WWT and IDA Signafing and Able Combinations Tight Munitors of There Genes (CDA), EDAN, and WWT1010 Alternational Seven Typohabotic Ecodermal Dypohasis have paider to have a seven provide the seven transformed Outsite-oxybod-hermit Applies in a spatient service space for a WWT101A nearestine multi- matifications of excludential dysplasis in carries of the mantion and mild matteriations of excludential dysplasis in carries of the mantion	2016	Denmark	WNT10A	1	2	2	2	2	2	2	0							and soles hyperhidrosis of palms and soles Coarse hair structure and/or light/scarce/britte hair	OODD (odonto-onycho-dermal dysplasi
Significant Role for WNT and EDA Signaling and Allele Combinations Eight Mutations of Three Genes (EDA, EDAR, and WNT10A) Identified in Seven Hypohakotic Ecisdownul Dysplasis Intents Odonto-onycho-dermal dysplasis in a patient homorogons for a WNT10A nonsense matution and mild					2			2	2	2	0				2		2	and soles hyperhidroosis of pains and soles Coarse hair structure and/or light/scarce/brittle hair Nail absormathios	
Significant Flok for WWT and IDA Signafing and Able Combinations Tight Munitors of There Genes (CDA), EDAN, and WWT1010 Alternational Seven Typohabotic Ecodermal Dypohasis have paider to have a seven provide the seven transformed Outsite-oxybod-hermit Applies in a spatient service space for a WWT101A nearestine multi- matifications of excludential dysplasis in carries of the mantion and mild matteriations of excludential dysplasis in carries of the mantion	2016	Denmark	WNT10A	1	2	2	2	2	2	2	0							and soles hyperhidrosis of palms and soles Coarse hair structure and/or light/scarce/britte hair	OODD (odonto-onycho-dermal dysplasi
Significant Flok for WWT and IDA Signafing and Able Combinations Tight Munitors of There Genes (CDA), EDAN, and WWT1010 Alternational Seven Typohabotic Ecodermal Dypohasis have paider to have a seven provide the seven transformed Outsite-oxybod-hermit Applies in a spatient service space for a WWT101A nearestine multi- matifications of excludential dysplasis in carries of the mantion and mild matteriations of excludential dysplasis in carries of the mantion	2016	Denmark	WNT10A	1	2	2	2	2	2	2	0							ind solos Dypothelionis of palins and solos Coarse har structure and/or light/source/bettle har Nal absormation Dypsion	OODD (odento-onycho-dermal dysplasi octodermal dysplasia
Significant Role for WIT and IDA Signifing and Alabi Conbination Fight Munition of These Genes (IDA, IDMA, and WITNIA)) Identified in Seven Hypokelowice Dotation-orphol-admul chaption in a patient Denorgion for 8 WTNTO Anonem mutities and mild munifications of exodermal dysplasis in carriers of the materia Almortand primary and permanent dustitions with ecodormal symptons predict WNT10A defisiency Materian in WNT10A. Arc Frequently Involved in Origodostin Anoximed With Mater Signs of	2016	Denmark	WNT10A	1	2	2	2	2	2	2	0	12 :		14				mit olos hyperblainois of quins and solos Course has trusturis and/or light-successful their Nield accountion Dryckan Dockonychia, dort stature, bittise, fiksing, sundi soot, fian and fugile hair, sparse hair, slow gowekh, and solosowers, son gellway, bullyos, finking and hun, wanning dady, hyperhalassis, kadwaych, dar	OODD (odento-onycho-dermal dysplasi octodermal dysplasia
Significant Role for WIT and IDA Signifies and Alaki Corbinations Tegli Mutation of Theorem (ThA, THA, and WITNI)Al Metallin likes well psycholoxic Ecohomal Dysplana Plantin Odates-oxyshes-Hand Applein is a paint Borospopion for 4WT110A Nonemarcs matisten and mild manifestations of ecohomal dysplans in carties of the mation Absorbed primary and permanent doubtions with octodermal symptoms predict WNT10A. defisionsy	2016	Denmark Swoden	WNTIOA WNTIOA	7	2	2	2	2	2 8	2	0	12 :	14	14	7	12	13	logothadus of palin and solos logothadus of palin and solos Coarse har structure and/or glafos-isoarchetite hair Norm har structure and/or glafos Dyyskin	OODD (odento-onycho-dermal dysplasi octodermal dysplasia
Significant Role for WIT and IDA Signifing and Alabi Conbination Fight Munition of These Genes (IDA, IDMA, and WITNIA)) Identified in Seven Hypokelowice Dotation-orphol-admul chaption in a patient Denorgion for 8 WTNTO Anonem mutities and mild munifications of exodermal dysplasis in carriers of the materia Almortand primary and permanent dustitions with ecodormal symptons predict WNT10A defisiency Materian in WNT10A. Arc Frequently Involved in Origodostin Anoximed With Mater Signs of	2016	Denmark Swoden	WNTIOA WNTIOA	7	2	2	2	2	2 8	2	0	12 :	14	14	7	12	13	and solos Dippethalismis of palam and solos Course have solves and solve applications that Barries and the solvest and solvest and solvest and solvest Drysban Drysban and solvest and solvest fairs of solgab hair, sparse have solve proved and spatients, spatianty, hypothesis, is an annexes to hur, wormand, adays, hypothalassis, Jakobysha, da mat, providend hypothesis, maximum to hur, worman, days, hypothalassis, Jakobysha, da mat, providend hypothesis, and resolvest fairs of program to replace and provide and spatients of the spatial spa	OODD (odento-onycho-dermal dysplasi octodermal dysplasia
Significant Role for WIT and IDA Signifing and Alabi Conbination Fight Munition of These Genes (IDA, IDMA, and WITNIA)) Identified in Seven Hypokelowice Dotation-orphol-admul chaption in a patient Denorgion for 8 WTNTO Anonem mutities and mild munifications of exodermal dysplasis in carriers of the materia Almortand primary and permanent dustitions with ecodormal symptons predict WNT10A defisiency Materian in WNT10A. Arc Frequently Involved in Origodostin Anoximed With Mater Signs of	2016 2016 2013	Denmark Sweden France	WNTIOA WNTIOA	9	2 10 14	2 10	2 12 11	2 14 9	2 8 7	2 14 14	0	12	14	14	8	12	13	mit solos Dyporhikativa (et plans and solos Corare har structure acidor light-cuencebritte har Nail ale accountation Dyskin Dyskin solomychin, but et alemanistic (et al. 1998) Solomychin, but et alemanistic (et al. 1998) Solomychin, but et al. 1998) Solomychin, but et al. 1998 Solomychin, but et al. 1998 Solo	OODD (odento-oxycho-dermal dysplasi octodermal dysplasia HED (Hypobalitotic Ectodermal Dyspla
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