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A meta-analysis of the prevalence of dental agenesis of permanent teeth

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Abstract – *Objective:* To gain more insight into the prevalence of dental agenesis. *Methods:* Data from Caucasian populations in North America, Australia and Europe were included in a meta-analysis. For the prevalence of African American, Chinese and Arab groups only indications could be reported because of a limited number of studies. *Results:* Agenesis differs by continent and gender: the prevalence for both sexes was higher in Europe (males 4.6%; females 6.3%) and Australia (males 5.5%; females 7.6%) than for North American Caucasians (males 3.2%; females 4.6%). In addition, the prevalence of dental agenesis in females was 1.37 times higher than in males. The mandibular second premolar was the most affected tooth, followed by the maxillary lateral incisor and the maxillary second premolar. The occurrence of dental agenesis was divided into three main groups: common ($P2_i > I2_s > P2_s$), less common ($I1_i > I2_i$ & $P1_s > C_s$ & $M2_i$) and rare ($M2_s$ & $M1_s > C_i > M1_i$ & $I1_s$). Unilateral occurrence of dental agenesis is more common than bilateral occurrence. However, bilateral agenesis of maxillary lateral incisors is more common than unilateral agenesis. The overall prevalence of agenesis in the maxilla is comparable with that in the mandible, but a marked difference was found between both jaws regarding tooth type. Absence of one or two permanent teeth is found in 83% of the subjects with dental agenesis. A practical application of the results of the meta-analysis is the estimation of dental treatment need.

Key words: dental agenesis; meta-analysis; prevalence

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Introduction

Various definitions are used to describe the phenomenon of congenitally missing teeth: hypodontia, oligodontia, anodontia, congenitally missing teeth and dental agenesis. Anodontia stands for patients with complete absence of teeth, oligodontia for patients with the absence of six or more teeth, apart from the third molars (1). The authors prefer the term dental agenesis as it describes more accurately the developmental disorder involved.

Large differences in the prevalence of dental agenesis have been reported, varying from 0.3 (2) to 36.5% (3). The relationship between the prevalence of missing teeth and dental consumption seems obvious. The actual number of dental agenesis in a community is therefore not only interesting for dentists but also for public health departments and health insurance companies. In most studies the sample size is too small to reach valid conclusions regarding the distribution of agenetic teeth for gender and site. The data presented in the literature have not been analyzed by an integrated approach. The aim of this study is to increase the insight in the prevalence of dental agenesis and its implication for dental consumption in communities by the method of meta-analysis (4, 5). In addition, meta-analysis enables

the study of determinants such as gender, site and race for the prevalence and more reliable predictions of dental consumption because of the number of teeth to be replaced.

Materials and methods

Source of material, inclusion and exclusion criteria

In November 2002 a literature search of prevalence reports on dental agenesis, catalogued in Medline, Silverplatter and EMBase, was conducted with the key words 'hypodontia', 'oligodontia', 'anodontia', 'agenesis' and 'prevalence or incidence'. Papers dealing with patients with craniofacial syndromes or developmental disorders were excluded. After this search 125 papers remained. Two independent observers (BP and AK) rated these papers according to the following criteria.

The inclusion criteria were:

- Presence of an English abstract
- Sample is representative for the underlying general population
- Diagnosis 'dental agenesis' based on a radiographic examination
- Report presents information on the ethnic background
- Report presents prevalence of agenesis except third molars

The exclusion criteria were:

- Study limited to an orthodontic patient group, or patient groups with craniofacial syndromes or developmental disorders
- Isolated populations were regarded as non-representative
- Incomplete radiographic examination
- Report with no proper data analysis
- A second report on the same population

An excellent interobserver agreement was obtained (inter-observer kappa = 0.96) and 35 publications were included. Furthermore, literature references in the 125 papers reporting on prevalence of hypodontia were checked. The result of this manual search was 16 papers.

The concerning 51 publications are presented in the reference list (6–56). The next step was a thorough evaluation of the whole text of these papers by the same two observers.

From the 35 computer searched papers 19 were excluded, from the 16 manual searched papers one was excluded, resulting in a total of 31 papers (inter-observer kappa = 1.00).

Populations in the meta-analysis

One study concerned two different European populations and could be split up into two substudies: schoolchildren and students (54). In two reports concerning White as well as African–American populations, the studies were split up to evaluate White and African–American populations separately (46, 48).

The data of two papers could not be used completely (see Table 1, references 42 and 51). The group of 6–8-years old children was not radiographically examined and could not be included in the meta-analysis (51). In another report concerning orthodontic patients as well as a random sample, only the data of the latter was used (42). Two reports regarded the same population (50, 43). Only the final study was included (43). Data from five reports on African American, Saudi Arab and Chinese populations were too limited for inclusion in the meta-analysis (18, 20, 24, 46, 48). On these populations only the reported outcomes and averages are presented (Table 1). Finally, 28 reports on White populations from North America, Australia and Europe could be used for further meta-analysis. The flow diagram leading to the final sample of 28 reports is presented in Fig. 1.

Outcome measures and determinants

The studies included in the meta-analysis often reported on the prevalence of affected patients, the site of agenetic teeth, the average number of missing teeth per patient and unilateral versus bilateral agenesis. Influencing determinants for the recorded prevalence of agenesis may be: age, gender, maxilla versus mandible, continent (North America, Europe and Australia) and the year of publication (1936–2002). The data needed for the analysis were retrieved from tables, figures or text; sometimes calculations were needed.

Consideration of bias

A large variation in sample sizes of the studies is seen, varying between 396 and 36 000 (Table 1). It is more likely to overlook dental agenesis in larger samples, especially when agenesis is not the only aim of the study (see Table 1; aim). The observed prevalence of dental agenesis therefore may vary with sample size, leading to information bias. Furthermore small studies resulting in a low prevalence of dental agenesis are less likely to be submitted or accepted for publication. Considering

Table 1. Included studies on prevalence of dental agenesis (% of persons with one or more agenetic teeth) by first author, country, sample composition, aim of the study, age range of observations, sample size, gender distribution, number of affected teeth and percentages of prevalences. In addition the tables and figures of the present study that deal with the analysis of the material as indicated later on are specified

Year	Author	Country	Literature number	Population	Aim[a]	Population[b]	Age	Sample size	Males	Females	Affected	Prevalence (%)	Table	Figure
1936	Dolder	Switzerland	56	Schoolchildren	p	Eur. White	6–15	10 000	?	?	340	3.4	1, 2, 3, 4, 5	1, 2, 3, 4
1956	Grahnen	Sweden	54	Schoolchildren	p	Eur. White	11–14	1006	531	475	61	6.1	1, 2, 3, 4, 5	1, 2, 4
1956	Grahnen	Sweden	54	Schoolchildren	p	Eur. White	17–43	1064	547	517	53	5.0	1, 2, 3, 4, 5	1, 2, 4
1961	Glenn	USA	53	Dental clinic	p	Am. White	3–16	777	405	327	40	5.1	1, 2, 4, 5	1, 2, 4
1963	Gimnes	Norway	52	Schoolchildren	p	Eur. White	6–15	36 000	?	?	1626	4.5	1, 2, 4, 5	1, 2, 3, 4
1963	Volk	Austria	51	Schoolchildren	p	Am. White	9–15	9533	4801	4732	911	9.6	1, 2, 3, 4, 5	1, 2, 3, 4
1964	Glenn	USA	49	Dental clinic	p	Am. White	3–16	925	431	494	47	5.1	1, 2, 4, 5	1, 2, 4
1966	Castaldi	Canada	36	Dental clinic	p	Am. White	6–9	457	226	231	19	4.2	1, 2, 3, 4, 5	1, 2, 3, 4
1967	Blayney	USA	48	Schoolchildren	p&o	Am. White	12–14	11 713	5825	5888	450	3.8	1, 2, 4, 5	1, 2
1968	Davies	Australia	47	Schoolchildren	p	Aust. White	12–14	2170	1220	950	136	6.3	1, 2, 3, 4, 5	1, 2, 3
1970	Muller	USA	46	Students	p	Am. White	>18	13 459	6696	6763	467	3.5	1, 2, 3, 4, 5	1, 2, 4
1971	Egermark-Erikson	Sweden	45	Schoolchildren	p	Eur. White	10–16	3327	1692	1635	209	6.3	1, 2	1, 2
1971	Haaviko	Finland	44	Schoolchildren	p	Eur. White	5–13	1041	527	514	83	8.0	1, 2, 4, 5	1, 2, 4
1973	Hunstadbraten	Norway	43	Schoolchildren	p	Eur. White	7–14	1295	645	650	131	10.1	1, 2, 4, 5	1, 3
1973	Thilander	Sweden	42	Schoolchildren	p&o	Eur. White	7–13	5459	2664	2795	332	6.1	1, 2	1, 2
1974	Bachmann	Switzerland	40	Schoolchildren	p	Eur. White	9–10	8694	4438	4256	670	7.7	1, 2, 3, 4, 5	1, 2, 3, 4
1974	Brook	UK	32	Schoolchildren	p&o	Eur. White	11–14	1115	572	543	49	4.4	1, 2	1, 2
1974	Thompson	Canada	41	Schoolchildren	p	Am. White	6–12	1191	615	576	88	7.4	1, 2, 3, 4, 5	1, 2, 3, 4
1974	Wisth	Norway	38	Schoolchildren	p	Eur. White	9	813	428	385	55	6.8	1, 2	1, 2, 4
1977	Bergstrom	Sweden	39	Schoolchildren	p&o	Eur. White	8–9	2589	1314	1275	192	7.4	1, 2, 4, 5	1, 2, 4
1977	Magnusson	Iceland	31	Schoolchildren	p	Eur. White	8–16	1116	521	595	88	7.9	1, 2, 4, 5	1, 2, 4
1980	Rolling	Denmark	26	Schoolchildren	p	Eur. White	9–10	3325	1668	1657	258	7.8	1, 2, 4, 5	1, 2, 3, 4
1989	Lo Muzio	Italy	22	Dental clinic	p	Eur. White	7–14	1529	789	740	79	5.2	1, 2, 4, 5	1, 2
1990	Lynham	Australia	17	Recruits	p	Aust. White	16–26	662	535	127	42	6.3	1, 2, 4, 5	1, 2
1993	Polastri	Italy	37	Recruits	p	Eur. White	19–26	700	700	0	25	3.6	1, 2, 3, 4, 5	1, 2, 3
1993	Aasheim	Norway	13	Schoolchildren	p	Eur. White	7–10	1953	993	960	127	6.5	1, 2, 4, 5	1, 2
1997	Johannsdottir	Iceland	10	Schoolchildren	p&o	Eur. White	6–7	396	204	192	17	4.3	1, 2, 4, 5	1, 2
2001	Backmann	Sweden	6	Schoolchildren	p	Eur. White	7	739	371	368	55	7.4	1, 2, 3, 4, 5	1, 2, 3, 4
1967	Blayney	USA	48	Schoolchildren	p&o	Afr. Am.	12–14	1320	682	638	54	4.1	1, 2	1, 2
1970	Muller	USA	46	Students	p&o	Afr. Am.	>18	1481	777	704	54	3.6	1, 2	1, 2
1989	Salem	Saudi Arabia	20	Dental clinic	p&o	Arab	4–14	2393	1433	960	53	2.2	1, 2	1, 2
1990	Al Emran	Saudi Arabia	18	Schoolchildren	p	Arab	13–14	500	500	0	20	4.0	1, 2	1, 2
1987	Davis	Hong Kong	24	Schoolchildren	p	Chinese	12	1093	561	532	75	6.9	1, 2	1, 2

[a]Aim of the study: p, prevalence of dental agenesis as only aim of the study; p&o, prevalence and other aims.
[b]Am., American; Aust., Australian; Afr., African; Eur., European.

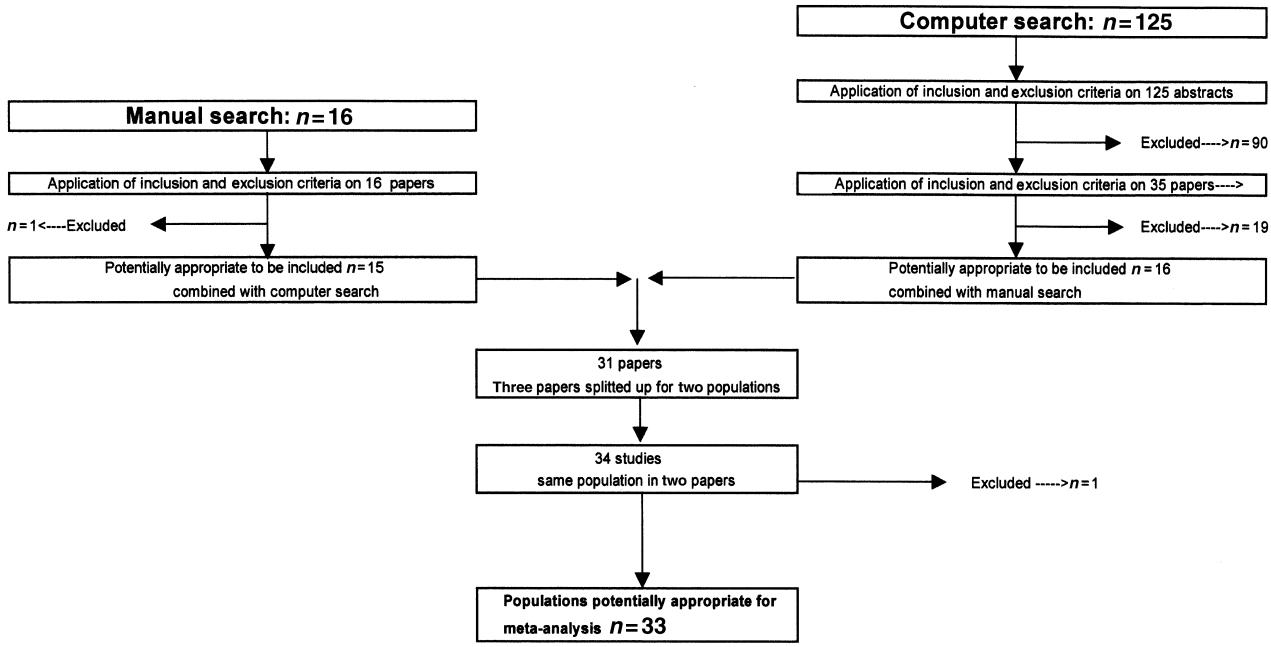

Fig. 1. Quorum statement flow diagram of literature search.

such possible effects, the influence of sample size on the prevalence has to be analyzed.

Age at diagnosis

An important issue is the age at diagnosis. Visibility of tooth germs on radiographs depends on their mineralization stage. Major differences in mineralization stages and dental age occur among subjects of the same chronological age. Tooth buds with a late onset of mineralization (mandibular second premolars) could give false-positive diagnosis of agenesis on radiographs. On the average, the mineralization of the mandibular second premolar starts at the age of 3–3.5 years, but it may also begin many years later (57, 58). A mandibular second premolar, diagnosed as agenetic at the age of seven showed to develop after the age of 10 years (59, 60). The diagnosis of dental agenesis of a mandibular second premolar before the age of seven is probably not conclusive (61). The age range in the selected studies is 3–43 years. If any relation is found between age of the investigated populations and prevalence of dental agenesis, further exclusion criteria based on age has to be formulated.

Statistical analysis

Multiple regression-analysis (weighted least squares) was applied to evaluate the influence of chronological age, sample size, continent and year of publication. Statistical significance was established at $P < 0.05$. The prevalence of agenesis per tooth type, affected patients and number of missing teeth per patient was calculated as far as reported in the papers. For the comparison of the prevalence for males and females, the relative risk (RR) was calculated (Mantel-Haenszel).

The number of teeth to be replaced because of agenesis for communities with 100 inhabitants was estimated. The standard error was calculated by the 'jack knife method' (leave one out method).

Results

The prevalence of dental agenesis derived from the 33 studies are presented in Table 1. It was found that the size of the investigated sample was negatively related to the reported prevalence of dental agenesis ($P = 0.01$). The largest study ($n = 36\,000$) should be regarded as an influential point for this phenomenon. After exclusion of this study the effect was no longer significant ($P = 0.44$). Therefore a correction for sample size was not seen as necessary. No significant increase of prevalence in populations including lower ages (7-years old or younger) was observed (Pearson correlation test $P = 0.42$).

A curvelinear relationship with publication year was found (polynomial regression

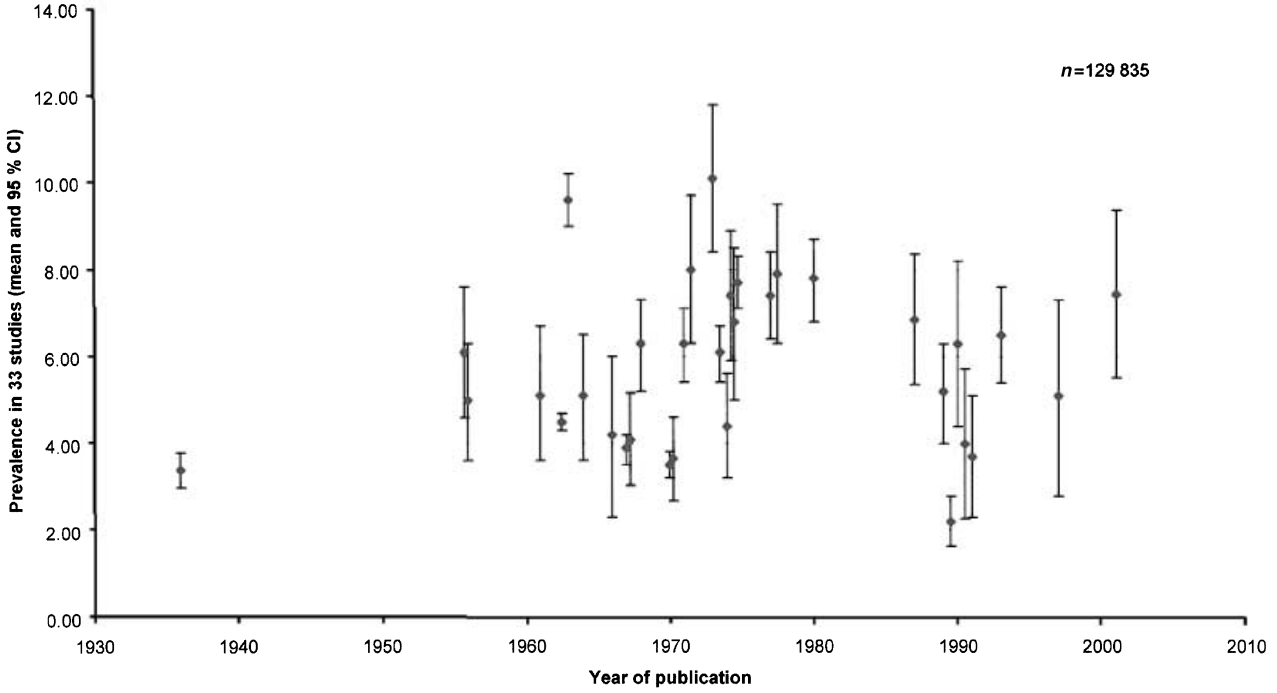

Fig. 2. Plot of the calculated recorded prevalence with 95% confidence intervals of agenetic teeth in 33 publications according to year of publication (see Table 1 for sources).

Table 2. Prevalence of dental agenesis by continent, race and gender in percentages (and 95% CI)

	Males	Females	Total
Europe (White)	4.6 (4.5–4.8)	6.3 (6.1–6.5)	5.5 (5.3–5.6)
North America (White)	3.2 (2.9–3.5)	4.6 (4.2–4.9)	3.9 (3.7–4.1)
North America (African American)	3.2 (2.2–4.1)	4.6 (3.5–5.8)	3.9 (3.1–4.6)
Australia (White)	5.5 (4.4–6.6)	7.6 (6.0–9.2)	6.3 (5.4–7.2)
Saudi Arabia (White)	2.7 (2.0–3.4)	2.2 (1.2–3.1)	2.5 (1.9–3.1)
Chinese (Mongoloid)	6.1 (4.0–8.1)	7.7 (5.4–10.0)	6.9 (5.3–8.4)

$P = 0.002$). Higher prevalence values were found in studies published in the period 1970–80. The calculated prevalence of dental agenesis against the year of publication are plotted in Fig. 2. This figure clearly shows heterogenity of the samples. It was decided to combine the information of comparable populations in the meta-analysis. Prevalence of dental agenesis appeared to be lower in North America than in Europe and Australia ($P = 0.0007$).

For further research questions in this meta-analysis not all papers could be used, because of lacking information. Table 1 indicates the papers that were used in the different questions.

The prevalence of dental agenesis for females was significantly larger than for males, RR = 1.37 (95% CI for RR = 1.28–1.45) As significant differences existed between males and females and populations, prevalence of dental agenesis are presented separately (Table 2).

The absolute percentage of agenesis for individual teeth is given in Table 3, based on 10 studies. The distribution of agenetic teeth according to tooth type is presented in Table 4. The overall prevalence of agenesis in the maxilla is comparable with that in the mandible. However, a marked

Table 3. Prevalence in percentages and 95% CI of dental agenesis of individual teeth derived from 48 274 persons (10 studies, see Table 1)

	Maxilla		Mandible	
	n	Prevalence (95% CI)	*n*	Prevalence (95% CI)
I1	3	0.00–0.01	143	0.25–0.35
I2	804	1.55–1.78	102	0.17–0.25
C	47	0.07–0.13	8	0.01–0.03
P1	100	0.17–0.25	66	0.10–0.17
P2	722	1.39–1.61	1479	2.91–3.22
M1	17	0.02–0.05	6	0.00–0.02
M2	21	0.03–0.06	47	0.07–0.13

Table 4. Distribution of 11 422 agenetic teeth according to tooth type in 112 334 persons (24 studies, see Table 1)

	Maxilla		Mandible	
	Number	Percentage	Number	Percentage
I1	18	0.2	403	3.5
I2	2620	22.9	282	2.5
C	149	1.3	39	0.3
P1	320	2.8	161	1.4
P2	2423	21.2	4687	41.0
M1	81	0.7	31	0.3
M2	67	0.6	141	1.2
Total	5703	49.7	5761	50.3

Table 5. Sequence of most to least affected teeth, divided in three main groups

	Prevalence (%)	Sequence
Common	1.5–3.1	$P2_i > I2_s > P2_s$
Less common	0.1–0.3	$I1_i > I2_i$ & $P1_s > C_s$ & $M2_I$
Rare	0.01–0.04	$M2_s$ & $M1_s > C_i > M1_i$ & $I1_s$

s, maxilla; i, mandible.

difference exists between both jaws regarding the frequency of agenesis of the various tooth types (Table 4).

Table 5 (based on 24 studies) shows the occurrence of dental agenesis subdivided into three categories: common, less common and rare.

Comparing bilateral and unilateral agenesis (based on nine studies), the occurrence of bilateral agenesis for the four most affected teeth was estimated. Bilateral agenesis of maxillary lateral incisors occurred more often (95% CI = 50.9–57.0%) than unilateral agenesis. For the other teeth unilateral agenesis was more common (Fig. 3). Bilateral agenesis expressed as a percentage of the sum of unilateral and bilateral agenesis provided the following data. Bilateral agenesis of mandibular second premolars was calculated as 43.5–47.7% (95% CI), that of maxillary second premolars as 46.3–52.2% (95% CI) and that of lower central incisors as 30.5–51.9% (95% CI). The number of congenitally missing teeth in patients with dental agenesis is presented in Fig. 4 (based on 17 studies). In most patients dental agenesis involved only one (48%) or two teeth (35%). In 2.6% of the affected patients six or more teeth were missing (overall prevalence of 0.14%). Based on the included studies in this meta-analysis the number of missing teeth needing replacement per 100 inhabitants was estimated for Europe 10,5 (SE = 0.2) and for North America 6.5 (SE = 0.2).

Discussion

The etiology of dental agenesis is still not quite clear. Several hypotheses have been postulated. It has been demonstrated that genetic factors with a marked degree of penetrance play a major role in dental agenesis (54). The linkage of dental agenesis and human genes is established. A mutation in human genes causes selective tooth agenesis (62–63). The influence of hereditary and environmental factors on the reduction of tooth number in human dentitions is illustrated by the prevalence of 36.5% found in a genetic and religious isolated population in North America (3). Developmental anomalies, endocrine disturbances, local factors as pathology, facial trauma and medical treatment have also been mentioned as etiological factors (25, 64). A developmental relationship between nerve,

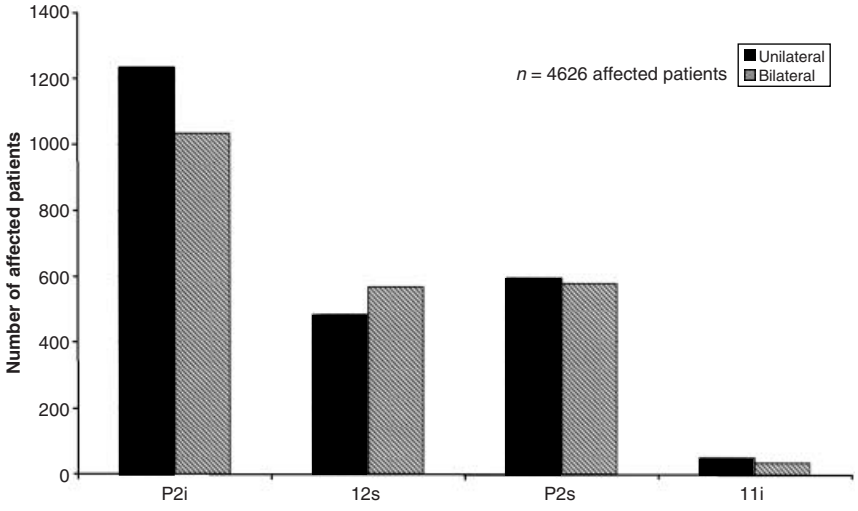

Fig. 3. Unilateral and bilateral occurrence of agenesis for the four most affected teeth in 10 studies (4626 affected patients) (see Table 1 for sources). P2i = mandibular second premolar; I2s, maxillary lateral incisor; P2s, maxillary second premolar; I1i, mandibular central incisor.

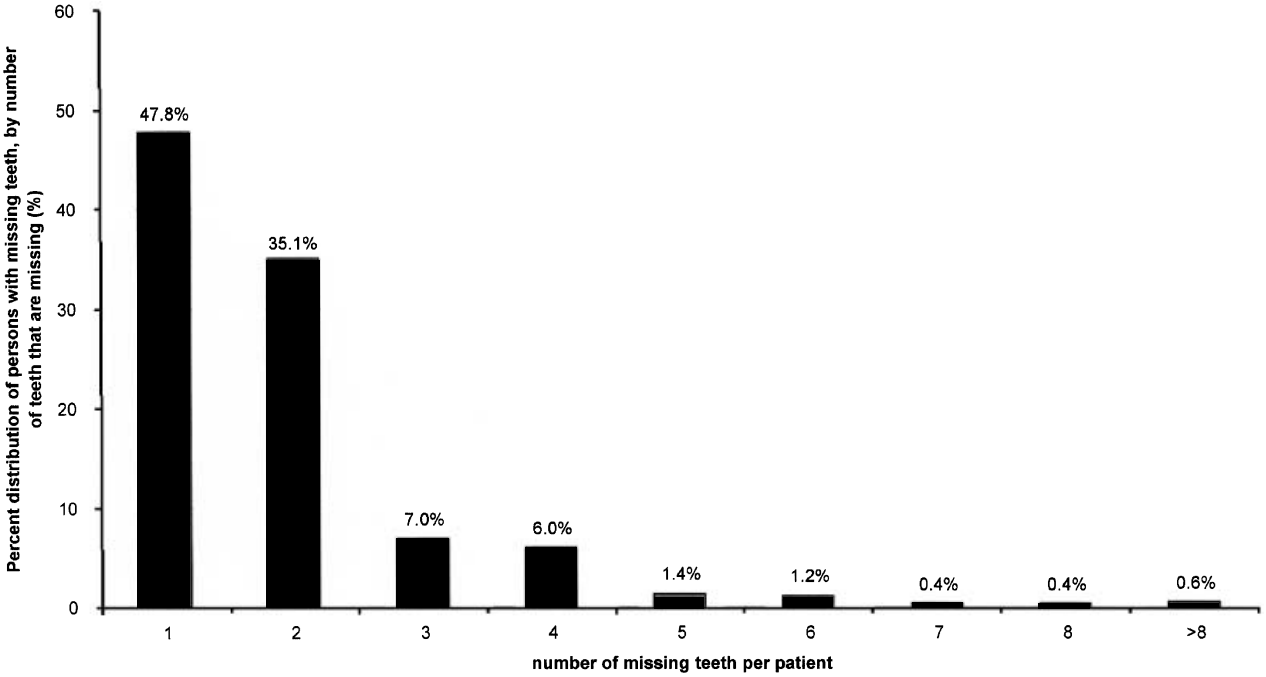

Fig. 4. Percent distribution of persons with missing teeth, by number of teeth that are missing.

oral mucosa, supporting tissues and hard tissue has been proposed (65, 66). This multi-factorial nature of dental agenesis may explain the variability in reported prevalence. Such a heterogenity is often a problem in meta-analysis. In this situation where local differences seem to play a role, the best approach is to accept such local differences. The meta-analysis may then be considered as a cluster sample for Europe.

Producing a list of published prevalence studies without confidence intervals or critical evaluation is of limited value as compared with analysis of available data. The total number of persons in this meta-analysis was large enough to be conclusive on several issues. The difference between Europe and Australia on one hand and North America on the other hand was clearly significant. Ethnic background is suggested as an important factor for prevalence of dental agenesis (2, 67, 68). Unfortunately African–American populations were only reported in four studies (46, 48, 69, 70). Two of these four studies were included after application of the inclusion and exclusion criteria. The prevalence reported in these two studies did not differ from the prevalence of the White population studies in North America. The large variation in prevalence of the included White population studies (3.4–10.1%) is another indication that difference in ethnic background is not the explanation of differences in prevalence between populations.

Studies including children under the age of 7 years tended to nonsignificant counter correlation i.e. a lower prevalence in younger children. This finding justified the decision not to exclude these studies for further evaluation. The remaining variation of reported prevalence (3.4–10.1%) is possibly explained by differences in sample size, inaccurate observations and different local etiological factors. The relationship with sample size turned out to be weak. After leaving out the largest sample, the relationship with sample size completely disappeared.

The prevalence of dental agenesis in the period 1936–2002, is significantly higher in the years 1971–80, a period dominated by studies in Scandinavian countries. This unexpected relationship with year of publication may be explained by the intensity of research in this area (12 of 28 papers). The high prevalence of the large Swiss study (40), is also worth mentioning. This raises the question of common determinants for agenesis between Switzerland and Scandinavian countries. Very local, genetic and environmental factors limited to those countries in the studied samples could be an explanation of this finding. Despite this heterogenity, it was decided to present an overall prevalence for the different continents (Table 2) as local differences are not important for national health policies.

Females seem to be 1.37 times more susceptible to dental agenesis than males. Most authors reported

a small non-significant sex-difference (46, 54, 55). Rose (71) is an exception, reporting a significant larger frequency in females than in males. Although this sample of 6000 was probably large enough to be conclusive, it consisted of orthodontic patients and therefore could not be regarded as representative for the population.

The mandibular second premolar is clearly the most frequently absent tooth, followed by the maxillary lateral incisor and the maxillary second premolar. In some studies a different sequence from most to least affected teeth is found. Sample size or incomplete examination may explain this difference. Agenesis of maxillary central incisors, maxillary and mandibular first molars and mandibular cuspids are very rare. Whenever these teeth are missing, loss of teeth because of trauma, caries and extraction must be carefully excluded before the diagnosis of agenesis is confirmed. Most patients (83%) with dental agenesis have absence of one or two permanent teeth. The absence of more than six missing permanent teeth is very rare (0.14%). For patients with oligodontia, defined as dental agenesis of six or more teeth, other factors than only prevalence are important (inheritance, reduction in size and form of teeth, reduction in size and shape of the alveolar process, combination with syndromes) (1, 72).

No overall differences in dental agenesis were found between the mandible and the maxilla. Only for maxillary lateral incisors prevalence of unilateral agenesis was lower than bilateral agenesis.

The prevalence of agenesis has a direct consequence for the costs of tooth replacement. Those with more missing teeth require more replacement, which means higher costs. On the assumption that every missing teeth needs replacement, it is clear that 'rare' patients with multiple agenesis are far more expensive than 'common' patients with only one missing teeth. In Fig. 4 is illustrated that 85% of the consumption of dental care related to agenesis in a community is needed for patients with up to four missing teeth. The consumption for patients with more than eight missing teeth is limited (3%). Many other factors are involved in the complex process of treatment planning. Not only the number but also the distributions of the missing teeth are important variables in the estimation of treatment need. A few Swedish studies indicate a higher orthodontic treatment need for patients with dental agenesis than for those in which all teeth are present (42, 73). Great objective need of orthodontic treatment exists for patients with missing anterior teeth or with two or more missing teeth in the same quadrant (13). The clinical significance of number and location of dental agenesis and the relation with size and shape abnormalities of the other teeth is still not fully clear. Most publications on treatment of dental agenesis are case-presentations or anadotical reports. Therefore further research with emphasis on long-term results and cost-benefit analysis is needed.

Conclusions

This meta-analysis presents clear facts on dental agenesis by narrow confidence intervals. It is shown that prevalence of dental agenesis in Europe and Australia is higher than in North America. In addition, the prevalence of dental agenesis in females is 1.37 times higher than in males for all three continents. Furthermore the meta-analysis demonstrates that mandibular second premolars are affected most frequently, followed by maxillary lateral incisors and maxillary second premolars.

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