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Biologic width dimensions – a systematic review

Julia C. Schmidt[1], Philipp Sahrmann[2], Roland Weiger[1], Patrick R. Schmidlin[2] and Clemens Walter[1]

[1]Department of Periodontology, Cariology and Endodontology, University of Basel, Basel, Switzerland; [2]Department of Preventive Dentistry, Periodontology and Cariology, Center of Dental Medicine, University of Zurich, Zurich, Switzerland

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Abstract

Background: Consideration of the biologic width in restorative dentistry seems to be important for maintaining periodontal health.

Objective: To evaluate the dimensions of the biologic width in humans.

Materials and Methods: A systematic literature search was performed for publications published by 28 September 2012 using five different electronic databases; this search was complemented by a manual search. Two reviewers conducted the study selection, data collection, and validity assessment. The PRISMA criteria were applied. From 615 titles identified by the search strategy, 14 publications were included and six were suitable for meta-analyses.

Results: Included studies were published from the years 1924 to 2012. They differed with regard to measurements of the biologic width. Mean values of the biologic width obtained from two meta-analyses ranged from 2.15 to 2.30 mm, but large intra- and inter-individual variances (subject sample range: 0.2 – 6.73 mm) were observed. The tooth type and site, the presence of a restoration and periodontal diseases/surgery affected the dimensions of the biologic width. Pronounced heterogeneity among studies regarding methods and outcome measures exists.

Conclusions: No universal dimension of the biologic width appears to exist. Establishment of periodontal health is suggested prior to the assessment of the biologic width within reconstructive dentistry.

Key words: biologic width; junctional epithelium; connective tissue attachment; crown margins; crown lengthening

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The placement of a restoration margin seems to be of importance for periodontal health (Kois 1996, Amiri-Jezeh et al. 2006). In addition to the influence of several risk factors (Kinane et al. 2006), the position of the restoration margin may affect the initiation and progression of periodontal diseases (Matthews and Tabesh 2004). Interactions between dental crowns and periodontal tissues were recently evaluated in a systematic review (Kosyfaki et al. 2010). The results of this study indicated that a crown margin with a supragingival location was the most beneficial restoration type in terms of periodontal health. In contrast, restorations with equigingival and subgingival margin terminations resulted in increased plaque accumulation, potentially leading to more severe gingival inflammation followed by periodontal destruction with increased pocket depths, loss of attachment, and gingival recessions (Lang et al. 1983, Schätzle et al. 2001, Reitemeier et al. 2002).

These inflammatory processes seem to be associated with a breach of the biologic width. The biologic width is defined as the junctional epithelium and supracrestal connective tissue attachment surrounding every tooth (Ingber et al. 1977, Amiri-Jezeh et al. 2006). The suggested physiological function of the biologic width is that of a protective barrier for the subjacent periodontal ligament and the supporting alveolar bone (Bosshardt & Lang 2005). The subgingival placement of crown margins may therefore affect the homeostasis of the periodontal tissues. However, several views and/or data exist concerning the ideal dimensions of the biologic width, leading to difficulties with respect to the development of clinical recommendations.

Conflict of interest and source of funding statement

The authors declare that they have no conflicts of interest. No external funding was obtained for this review.

Review of Current Literature

Objective

The purpose of the present systematic review was to evaluate the

dimensions of the biologic width and its compartments in humans.

The specific questions addressed in this systematic review were as follows:

What are the dimensions of the biologic width and its compartments (junctional epithelium and connective tissue attachment) around permanent teeth in

a. humans without a reported history of periodontal disease?
b. humans with a history of periodontal disease?

Material and Methods

Protocols

The present systematic review considers the PRISMA (Preferred Reporting Items for Systematic Review and Meta-analyses) criteria (Liberati et al. 2009, Moher et al. 2009) (Appendix S1). The research questions were adapted using the PICO (Population, Intervention, Comparison, Outcomes) criteria (Miller & Forrest 2001).

Eligibility criteria

The search was limited to original studies on the dimensions of the biologic width and its compartments that were performed in humans. No language or time restrictions were applied.

Information sources

The electronic databases MEDLINE, EMBASE, Cochrane, Web of Science and Biosis were searched for studies published prior to 28 September 2012. A manual search was performed on *Journal of Clinical Periodontology*, *Journal of Periodontology*, *Journal of Periodontal Research* and *Periodontology 2000* from January 1990 to September 2012. Moreover, the references of studies examined for inclusion and review articles on the biologic width were thoroughly analyzed to search for additional studies.

Literature search

The search protocols in the different databases were validated and created as identical as possible. Combinations of the search terms (biologic* NEAR/1 width), [(gingiva* OR ligament OR periodontal) NEAR/2 width], and (oral OR dent* OR odont* OR periodont*) were applied (Appendix S2).

Study selection

The combinations of search terms resulted in a list of 615 titles, that is, 569 titles from the electronic databases and 46 titles from the hand search (Fig. 1). Two of the authors (J. S. and P. S.) screened the titles and abstracts for compliance with the inclusion criteria and selected 34 studies for full text analysis. A third reviewer (C. W.) reassessed both the included and excluded studies.

Data collection process

Data items

The following data were collected in data extractions files: Proband characteristics (ethnicity, age, inclusion criteria), # Probands, # Tooth sites, Methods, Measurements, Outcomes (tooth type, tooth site, presence of restorations, gingival inflammation, probing depths, attachment loss, altered passive eruption, surgical crown lengthening).

Risk of bias in individual studies

The methodological and reporting quality of included studies was evaluated by modified items from the Cochrane Collaboration's Tool for assessing risk of bias (Graziani et al. 2012) and the STROBE statement (Pjetursson et al. 2012) (Appendix S3). Considering the adequacy in the respective studies, the items were graded and the percentage of positively graded items was calculated (Graziani et al. 2012).

Summary measures

Most studies reported distance measurements using mean values with corresponding standard deviations, but also individual values or the range of individual values.

Synthesis of results

A pronounced heterogeneity regarding methods and outcome parameters in the included studies occurred (Appendix S4). Six studies were included in meta-analyses with respect to histological and clinical data (Appendix S5).

Results

Study selection

A total of 569 titles from the electronic databases and 46 titles from the hand search were identified (Fig. 1). The titles and abstracts were screened by two reviewers (observed agreement = 91.38%, kappa = 0.622; intra-class correlation coefficient (ICC) = 0.623). The full texts of 34 publications (selected by at least one reviewer) were further analyzed. Full text analysis led to exclusion of further 24 studies (Appendix S6). Finally, 10 publications from the electronic and hand search satisfied the inclusion criteria. Four additional publications were identified by screening references in the studies evaluated for inclusion (Orban & Köhler 1924, Stanley 1955, Vacek et al. 1994, Al-Rasheed et al. 2005). The 14 included studies were published in the period from 1924 to 2012.

Description of characteristics, results, and quality assessment

The study characteristics (Appendix S7) and the results related to outcome parameters (Tables 1, 2, 3) are summarized in Tables. In the following, study characteristics relevant for the original research questions are described. Risk of bias within studies and the quality scores (Cochrane Collaboration's Tool for assessing risk of bias, STROBE statement) for included studies are presented in Appendix S8.

Summary of characteristics (PICO – Population, Intervention/Comparison, Outcomes)

Population – number and characteristics of subjects and tooth sites. Most studies included probands between the ages of 19 and 50 years. Orban & Köhler (1924) additionally analyzed deciduous teeth of probands at the age of 1.75 years and of 3.5 years. In three studies, probands older than 70 years were also analyzed. In one study, proband age was not available (Stanley 1955). The ethnic background of probands was provided in four studies. One study examined a Caucasian population, whereas three other studies assessed probands of Asian origin.

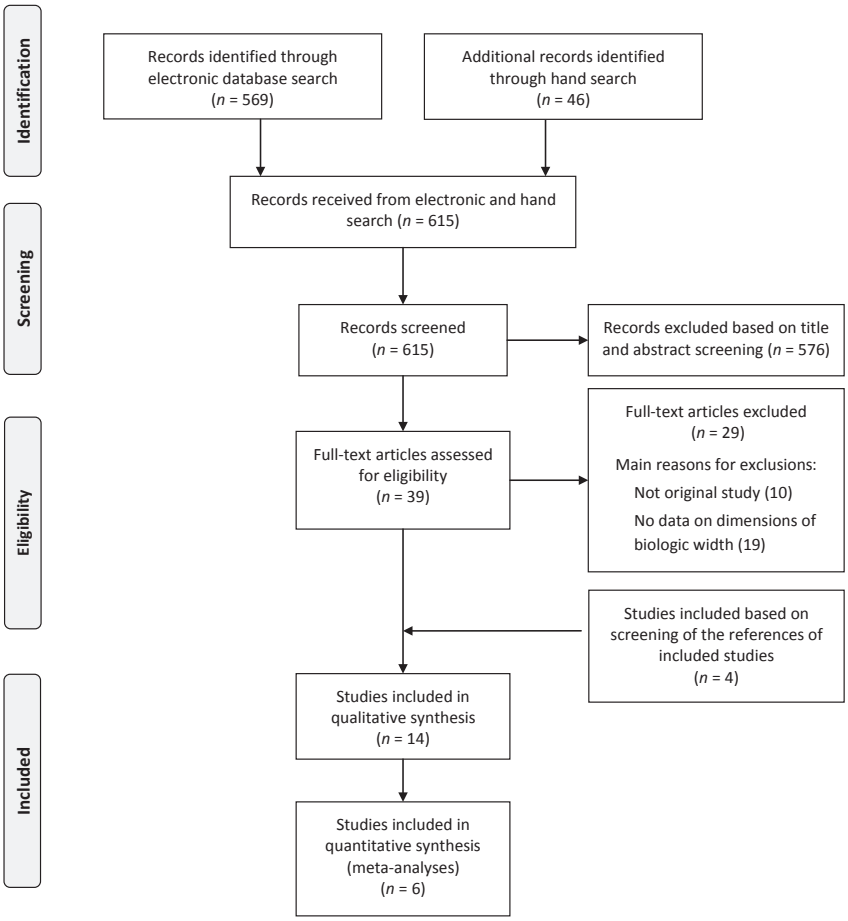

Fig. 1. Selection process of the studies included.

The remaining publications did not disclose the ethnicity of subjects.

Inclusion criteria were described at the patient level and were occasionally specified with respect to the analyzed tooth. Three studies presupposed the absence of restorations, periodontal pathology, recent orthodontic treatment, and any systemic pathology influencing the periodontium. Three other studies reported the periodontal status, including probing depth and clinical attachment loss, and others demanded healthy conditions without further specification. Two studies stated exclusion criteria in terms of the presence of epithelial ulceration (Orban & Köhler 1924) and the presence of increased tooth mobility and probing depths, bone loss, unrestorable teeth, local or systemic contraindications to surgery, and furcation involvement (Shobha et al. 2010). Four studies assigned neither inclusion nor exclusion criteria for the analyzed subjects.

Overall, the dimensions of the biologic width were analyzed in more than 3000 tooth sites. Alpiste-Illueca (2012) analyzed a total of 123 probands. The number of included probands in the remaining studies ranged from 5 to 88 individuals. The number of tooth sites analyzed for biologic width measurements varied among the included studies. Three studies analyzed one tooth site in each patient, two studies noted two sites per tooth, one study included three sites per tooth in the assessment and eight studies included four sites per tooth.

Intervention/comparison – methods and measurements. Measurements of the biologic width were accomplished either using histological techniques, clinical methods or a combination of both clinical and radiographic methods. All histological samples were obtained from human jaws of autopsy cadaver specimens. The histometric analysis was performed using light microscopy. The preferred radiographic method was the parallel profile radiograph technique comprised of two radiographs in frontal and lateral projections. Clinical measurement of the sulcus depth was performed to supplement this method and provide an estimate of the biologic width. As an exclusive method of performing biologic width measurements, clinical measurements recorded sulcus depths and attachment levels via periodontal probing and alveolar bone levels via transgingival probing under local anesthesia.

Twelve studies assessed distances corresponding to the defined biologic width. Three of these studies additionally quantified distances for the junctional epithelium and supracrestal connective tissue attachment. In two studies, the dimensions of the junctional epithelium and the connective tissue attachment were separately measured. The length of the biologic width results from the addition of both dimensions. Conversely, studies using clinical or combined clinical and radiographic methods did not differentiate between the compartments of the biologic width.

Outcomes – patient- and tooth-related factors. Most studies differentiated among several clinical parameters with potential impacts on the dimensions of the biologic width. These parameters were different tooth types and tooth sites, the existence of subgingivally located restorations or altered passive eruption and varied healing periods following surgical crown lengthening. Six studies correlated the biologic width with the existing attachment loss. The attachment loss was explained according to the historical biologic understanding regarding different stages of "passive eruption" of a tooth in two studies (Orban & Köhler 1924, Gargiulo et al. 1961). Another publication assessed the biologic width in patients with severe, generalized chronic periodontitis (Novak et al. 2008). The

Table 1. Influence of patient- and tooth-related factors on the dimensions of the biologic width according to tooth type and tooth site. Mean values ± standard deviations (range of individual values) in mm

		Method of analysis			Tooth type				Tooth site				
Author (year of publication)	Total	Histologically	Clinically	Clinically/ radio-graphically	Anterior	Posterior	Premolars	Molars	Mesial	Distal	Buccal	Oral	Approximal (mesial + distal)
Orban & Köhler (1924)*	(0.50–6.73)	(0.50–6.73)	–	–	(0.50–6.73)	–	(0.80–3.66)	(0.65–6.40)	(0.58–4.67)	(0.88–4.34)	(0.65–6.73)	(0.50–4.90)	(0.58–4.67)
Stanley (1955)	1.50 (0.2–2.9)	1.50 (0.2–2.9)	–	–	nr	nr	nr	nr	nr	nr	–	–	1.50 (0.2–2.9)
Gargiulo et al. (1961)†	2.04‡	2.04‡	–	–	nr	nr	nr	nr	nr	nr	nr	nr	nr
Vacek et al. (1994)	(0.75–4.33)	(0.75–4.33)	–	–	1.75 ± 0.56 (0.75–3.29)	nr	1.97 ± 0.67 (0.78–4.33)	2.08 ± 0.55 (0.84–3.29)	nr	nr	nr	nr	nr
Lanning et al. (2003)	2.26 ± 0.13§ 2.36 ± 0.08¶ 2.23 ± 0.11**	–	2.26 ± 0.13§ 2.36 ± 0.08¶ 2.23 ± 0.11**	–	nr	nr	nr	nr	nr	nr	–	–	2.26 ± 0.13§ 2.36 ± 0.08¶ 2.23 ± 0.11**
Alpiste-Illueca (2004)	2.00 ± 0.72 (0.5–3.8)	–	–	2.00 ± 0.72 (0.5–3.8)	2.00 ± 0.72 (0.5–3.8)	–	–	–	–	–	2.00 ± 0.72 (0.5–3.8)	–	–
Al-Rasheed et al. (2005)	1.25 ± 0.19 (0.89–1.61)	–	1.25 ± 0.19 (0.89–1.61)	–	1.30 ± 0.41‡‡ 1.17 ± 0.32§§	nr	1.27 ± 0.41¶¶ 1.32 ± 0.35***	1.19 ± 0.28††† 1.22 ± 0.38‡‡‡	1.42 ± 0.31	1.22 ± 0.28	1.10 ± 0.25	–	nr
Xie et al. (2007)	2.17 ± 0.18	2.17 ± 0.18	–	–	2.10 ± 0.16	2.22 ± 0.18	nr	nr	2.22 ± 0.17	2.21 ± 0.16	2.16 ± 0.21	2.08 ± 0.16	nr
Xie & Chen (2007)	2.17 ± 0.19	2.17 ± 0.19	–	–	2.07 ± 0.14	nr	2.23 ± 0.17	2.25 ± 0.19	2.11 ± 0.17	2.20 ± 0.16	2.20 ± 0.15	2.16 ± 0.13	nr
Novak et al. (2008)	3.95 ± 1.04	–	–	3.95 ± 1.04	nr	nr	nr	nr	nr	nr	–	–	3.95 ± 1.04
Shobha et al. (2010)	1.80 ± 0.77§ 1.73 ± 0.88¶ 1.53 ± 0.74**	–	1.80 ± 0.77§ 1.73 ± 0.88¶ 1.53 ± 0.74**	–	nr	nr	nr	nr	nr	nr	–	–	1.80 ± 0.77§ 1.73 ± 0.88¶ 1.53 ± 0.74**
Galgali & Gontiya (2011)	(0.8–2.2)	–	(0.8–2.2)	(0.94–2.11)	(0.8–2.2)	–	–	–	–	–	(0.8–2.2)	–	–
Ganji et al. (2012)	1.95, 2.55, 2.7	–	1.95, 2.55, 2.7	–	nr	nr	nr	nr	nr	nr	nr	nr	nr
Alpiste-Illueca (2012)	2.16 ± 0.85 (0.5–4.9)	–	–	2.16 ± 0.85 (0.5–4.9)	2.16 ± 0.85 (0.5–4.9)	–	–	–	–	–	2.16 ± 0.85 (0.5–4.9)	–	–

*values calculated from Tables 2 to 5 in Orban & Köhler (1924).
†material in Gargiulo et al. (1961) included measurements of specimens published by Orban & Köhler (1924).
‡calculated from Table 12 in Gargiulo et al. (1961).
§treated sites.
¶non-adjacent sites.
**adjacent sites.
††range of mean values.
‡‡maxillary left central incisors.
§§mandibular right central incisors.
¶¶maxillary left first premolars.
***mandibular right first premolars.
†††maxillary right first molars.
‡‡‡mandibular left first molars; nr, data not reported.
–, no sites presenting the related outcome parameter in the studies included.

Table 2. Influence of patient- and tooth-related factors on the dimensions of the biologic width according to presence of a restoration, gingival inflammation, probing depth and attachment loss. Mean values ± standard deviations (range of individual values) in mm

	Restoration		Gingival inflammation		Probing depth				Attachment loss		
Author (year of publication)	Existent‡‡‡	Non-existent‡‡‡	Existent‡‡‡	Non-existent‡‡‡	<2 mm	2–4 mm	>4–7 mm	>7 mm	<3 mm	3–6 mm	>6 mm
Orban & Köhler (1924)*	–	–	–	–	(0.50–6.73)†	(1.30–3.10)†	0.97, 1.44†	–	(0.56–6.73)	(0.50–6.40)	3.04
Stanley (1955)	–	–	–	–	nr	nr	nr	nr	nr	nr	nr
Gargiulo et al. (1961)‡	–	–	–	–	nr	nr	nr	nr	2.43, 2.17, 1.80§	1.77¶	–
Vacek et al. (1994)	–	–	–	–	(0.75–4.33)***	–	–	–	(0.75–4.33)***	–	–
Lanning et al. (2003)	–	–	–	–	–	2.26 ± 0.13**·*** 2.36 ± 0.08††·*** 2.23 ± 0.11‡‡·***	–	–	–	–	2.26 ± 0.13**·*** 2.36 ± 0.08††·*** 2.23 ± 0.11‡‡·***
Alpiste-Illueca (2004)	–	2.00 ± 0.72 (0.5–3.8)	–	–	2.00 ± 0.72 (0.5–3.8)	–	–	–	nr	nr	nr
Al-Rasheed et al. (2005)	–	–	1.25 ± 0.19 (0.89–1.61)***	–	–	1.25 ± 0.19 (0.89–1.61)	–	–	nr	nr	nr
Xie et al. (2007)	–	–	–	–	2.17 ± 0.18†††	–	–	–	nr	nr	nr
Xie & Chen (2007)	–	–	–	–	nr	nr	nr	nr	nr	nr	nr
Novak et al. (2008)	–	–	–	–	5.02 ± 2.48	4.16 ± 1.32	3.33 ± 1.17	3.21 ± 1.29	5.35 ± 1.50	3.83 ± 1.18	3.05 ± 0.96
Shobha et al. (2010)	–	1.80 ± 0.77** 1.73 ± 0.88†† 1.53 ± 0.74‡‡	–	–	1.73 ± 0.88††·*** 1.53 ± 0.74‡‡·***	1.80 ± 0.77§§·***	–	–	–	1.80 ± 0.77**·*** 1.73 ± 0.88††·*** 1.53 ± 0.74‡‡·***	–
Galgali & Gontiya (2011)	–	(0.8–2.2)	–	–	nr	nr	nr	nr	nr	nr	nr
Ganji et al. (2012)	–	1.95, 2.55, 2.7	–	–	nr	nr	nr	nr	nr	nr	nr
Alpiste-Illueca (2012)	–	2.16 ± 0.85 (0.5–4.9)	–	2.16 ± 0.85 (0.5–4.9)	nr	nr	nr	nr	nr	nr	nr

*values calculated from Tables 2 to 5 in Orban & Köhler (1924).
†relating to histological pocket depth.
‡material in Gargiulo et al. (1961) included measurements of specimens published by Orban & Köhler (1924).
§mean values of groups I-III (attachment loss ranged from 0.00 to 2.36 mm).
¶mean value of group IV (attachment loss ranged from 0.39 to 6.08 mm).
**treated sites.
††non-adjacent sites.
‡‡adjacent sites.
§§range of mean values.
¶¶probing depth ranged from 1.89 to 3.06 mm.
***related outcome parameter (bleeding index, plaque index, probing depth or attachment loss) available as mean ± SD of all sites.
†††probing depth <3 mm.
‡‡‡bar (–) means that data are not available; nr, data not reported; –, no sites presenting the related outcome parameter in the studies included.

Table 3. Influence of patient- and tooth-related factors on the dimensions of the biologic width according to presence of altered passive eruption and healing periods following surgical crown lengthening. Mean values ± standard deviations (range of individual values) in mm

	Altered passive eruption**		Post-surgery		
Author (year of publication)	Existent††	Non-existent††	3–6 weeks††	3 months††	6 months††
Orban & Köhler (1924)	–	–	–	–	–
Stanley (1955)	–	–	–	–	–
Gargiulo et al. (1961)	–	–	–	–	–
Vacek et al. (1994)	–	–	–	–	–
Lanning et al. (2003)	–	–	–‡‡	1.96 ± 0.05* 1.94 ± 0.03† 1.94 ± 0.04‡	2.19 ± 0.06* 2.14 ± 0.06† 2.08 ± 0.07‡
Alpiste-Illueca (2004)	–	–	–	–	–
Al-Rasheed et al. (2005)	–	–	–	–	–
Xie et al. (2007)	–	–	–	–	–
Xie & Chen (2007)	–	–	–	–	–
Novak et al. (2008)	–	–	–	–	–
Shobha et al. (2010)	–	–	1.73 ± 0.59* 1.93 ± 1.03† 1.93 ± 0.88‡	1.67 ± 0.62* 1.93 ± 0.80† 1.87 ± 0.64‡	1.87 ± 0.83* 1.80 ± 0.56† 1.60 ± 0.51‡
Galgali & Gontiya (2011)	–	–	–	–	–
Ganji et al. (2012)	–	–	1.25§, 1.85§ 1.15¶, 1.65¶	1.8§ 2.5¶	–‡‡
Alpiste-Illueca (2012)**	2.61 ± 1.0 (0.7–4.9)	2.00 ± 0.72 (0.5–3.8)	–	–	–

*treated sites.
†non-adjacent sites.
‡adjacent sites.
§after ostectomy.
¶after gingivectomy.
**altered passive eruption is defined as gingival overlap on the anatomical crown (Alpiste-Illueca 2012).
††bar (–) means that data are not available.
‡‡follow-up time not analyzed; nr, data not reported; –, no sites presenting the related outcome parameter in the studies included.

biologic width was correlated with probing depth and attachment loss in this subject sample. Al-Rasheed et al. (2005) measured the biologic width in the presence of gingival inflammation. One study considered the influence of local pathologic factors in terms of supra- and subgingival calculus, epithelial ulceration and inflammation of the lamina propria on the analyzed dimensions (Stanley 1955). Five studies did not compare the biologic width measurements under distinct clinical conditions.

What are the dimensions of the biologic width and its compartments (junctional epithelium and connective tissue attachment) around permanent teeth in

a) *humans without a reported history of periodontal disease?* The mean dimensions of the biologic width ranged from 1.5 to 2.7 mm (Table 1). The smallest biologic width measurement was 0.2 mm. Tooth type and tooth site influenced the dimensions of the biologic width. In three studies, the biologic width around anterior teeth was smaller than that around posterior teeth. The mean biologic width calculated in the meta-analysis amounted 2.15 mm (CI 95% 2.01, 2.29) (Appendix S5a).

The mean dimensions of the junctional epithelium ranged from 0.57 mm (Subject sample range: 0.1 – 1.4 mm) to 1.14 ± 0.49 mm (Subject sample range: 0.32 – 3.27 mm) (Table 4). The highest variability in individual values was observed by Orban & Köhler (1924), ranging from 0.08 to 3.72 mm. Tooth type, tooth site and, presence of a restoration affected the measured dimensions of the junctional epithelium (Table 5). A discrete analysis of anterior and posterior teeth revealed greater dimensions of the junctional epithelium around posterior teeth, with the highest values found for molars. The mean dimensions around anterior teeth were 0.97 ± 0.13, 0.99 ± 0.14 mm and 1.03 ± 0.45 mm. In contrast, the mean values around posterior teeth ranged from 1.12 ± 0.19 mm to 1.22 ± 0.46 mm. Differences with regard to distinct tooth sites were observed. The dimensions of junctional epithelium at mesial and distal sites exceeded measurements at buccal and oral sites. The mean values at the approximal sites were >1 mm, whereas the mean values at the oral sites were <0.9 mm. Vacek et al. (1994) reported higher measurements for the junctional epithelium around restored teeth compared with non-restored teeth (Table 5).

The mean dimensions of connective tissue attachments ranged from 0.77 ± 0.29 mm (Subject sample range: 0.29 – 1.84 mm) to 1.10 ± 0.13 mm (Table 6). Orban & Köhler (1924) observed the highest variability in individual values, which ranged from 0.00 to 6.52 mm. The tooth type, tooth site, and presence of a restoration influenced the dimensions of the connective tissue attachment (Table 7). Vacek et al. (1994) detected differences in such dimensions between anterior and posterior teeth, with mean values of 0.71 ± 0.24 mm around anterior teeth and 0.77 ± 0.31 mm and 0.89 ± 0.31 mm around premolars and molars, respectively. In contrast, Xie et al. (2007) did not observe such differences between anterior and posterior teeth. Tooth site differentiation revealed greater dimensions of the connective

tissue attachment at buccal and oral sites compared with mesial and distal sites. The mean values at buccal and oral sites ranged from 1.13 ± 0.13 mm to 1.31 ± 0.12 mm. In contrast, the mean mesial and distal dimensions ranged from 0.95 ± 0.13 mm to 1.05 ± 0.09 mm. The mean dimensions of the connective tissue attachments were 0.84 ± 0.26 and 0.76 ± 0.29 mm for restored and non-restored teeth, respectively (Table 7).

The mean dimensions of the junctional epithelium exceeded those of the connective tissue attachment at approximal sites (Xie & Chen 2007, Xie et al. 2007). At buccal and oral sites, higher mean values of connective tissue attachment compared with junctional epithelium were reported (Xie & Chen 2007, Xie et al. 2007). Compared with epithelial measurements, two studies observed a greater variability in values of connective tissue attachments (Orban & Köhler 1924, Gargiulo et al. 1961). In contrast, Vacek et al. (1994) observed lower variability in connective tissue measurements compared with those of junctional epithelium.

b) humans with a history of periodontal disease? The mean dimensions of the biologic width ranged from 1.25 ± 0.19 mm to 3.95 ± 1.04 mm (Table 1). The smallest biologic width measurement was 0.5 mm and the greatest amounted 6.40 mm. Attachment loss and increased probing depths influenced the dimensions of the biologic width (Table 2). Gargiulo et al. (1961) compared the measurements with respect to periodontal diseases and reported a mean biologic width of 2.43 mm at sites without attachment loss. In contrast, the mean biologic width was reduced to 1.71 mm at sites with an attachment loss of up to 6.08 mm. Novak et al. (2008) observed similar findings in untreated patients with severe, generalized chronic periodontitis. The mean biologic width was reduced to 3.05 ± 0.96 mm at sites with an attachment loss of more than 6 mm, whereas the mean biologic width measured 5.35 ± 1.50 mm at sites with an attachment loss of up to 2 mm. Similarly, the mean biologic width was smaller at sites with increased probing depths. However, in comparison to tooth sites without a reported

Table 4. Influence of patient- and tooth-related factors on the dimensions of the junctional epithelium according to tooth type and tooth site. Mean values ± standard deviations (range of individual values) in mm

	Method of analysis				Tooth type				Tooth site				
Author (year of publication)	Total	Histologically	Clinically	Clinically/ radiographically	Anterior	Posterior	Premolars	Molars	Mesial	Distal	Buccal	Oral	Approximal (mesial + distal)
Orban & Köhler (1924)	(0.08–3.72)	(0.08–3.72)	–	–	(0.08–2.90)	nr	(0.14–2.65)	(0.16–3.72)	(0.16–3.72)	(0.20–2.90)	(0.14–2.80)	(0.08–2.15)	(0.16–3.72)
Stanley (1955)	0.57 (0.1–1.4)	0.57 (0.1–1.4)	–	–	nr	nr	nr	nr	nr	nr	–	–	0.57 (0.1–1.4)
Gargiulo et al. (1961)*	0.97 (0.08–3.72)	0.97 (0.08–3.72)	–	–	nr	nr	nr	nr	(0.44–1.56)†	(0.88–1.37)†	(0.63–1.35)†	(0.53–1.14)†	(0.44–1.56)†
Vacek et al. (1994)	1.14 ± 0.49 (0.32–3.27)	1.14 ± 0.49 (0.32–3.27)	–	–	1.03 ± 0.45 (0.38–2.48)	nr	1.20 ± 0.53 (0.32–3.27)	1.22 ± 0.46 (0.44–2.30)	nr	nr	nr	nr	nr
Xie et al. (2007)	1.07 ± 0.18	1.07 ± 0.18	–	–	0.99 ± 0.14	1.12 ± 0.19	nr	nr	1.20 ± 0.16	1.16 ± 0.16	1.03 ± 0.13	0.88 ± 0.09	nr
Xie & Chen (2007)	1.07 ± 0.18	1.07 ± 0.18	–	–	0.97 ± 0.13	nr	1.12 ± 0.18	1.14 ± 0.19	1.16 ± 0.16	1.18 ± 0.14	1.03 ± 0.12	0.85 ± 0.11	nr

*material in Gargiulo et al. (1961) included measurements of specimens published by Orban & Köhler (1924).
†range of mean values; nr, data not reported; –, no sites presenting the related outcome parameter in the studies included.

Table 5. Influence of patient- and tooth-related factors on the dimensions of the junctional epithelium according to presence of a restoration, probing depth and attachment loss. Mean values ± standard deviations (range of individual values) in mm

Author (year of publication)	Restoration		Probing depth				Attachment loss		
	Existent**	Non-existent**	<2 mm	2–4 mm	>4–7 mm	>7 mm	<3 mm	3–6 mm	>6 mm
Orban & Köhler (1924)	–	–	(0.08–3.72)	(0.34–1.60)	(0.38, 0.45)	–	(0.08–3.72)	(0.20–2.08)	2.24
Stanley (1955)	–	–	nr	nr	nr	nr	nr	nr	nr
Gargiulo et al. (1961)*	–	–	nr	nr	nr	nr	1.35, 1.10, 0.74[†] (0.16–3.72)	0.71[‡] (0.08–2.65)	–
Vacek et al. (1994)	1.32 ± 0.47 (0.69–2.29)	1.11 ± 0.49 (0.32–3.27)	1.32 ± 0.47 (0.69–2.29)[¶]	–	–	–	1.32 ± 0.47 (0.69–2.29)[¶]	–	–
Xie et al. (2007)	–	–	1.07 ± 0.18[§]	–	–	–	nr	nr	nr
Xie & Chen (2007)	–	–	nr	nr	nr	nr	nr	nr	nr

*material in Gargiulo et al. (1961) included measurements of specimens published by Orban & Köhler (1924).
[†]mean values of groups I-III (attachment loss ranged from 0.00 to 2.36 mm).
[‡]mean value of group IV (attachment loss ranged from 0.39 to 6.08 mm).
[§]probing depth <3 mm.
[¶]related outcome parameter (probing depth or attachment loss) available as mean ± SD of all sites.
**bar (–) means that data are not available; nr, data not reported; –, no sites presenting the related outcome parameter in the studies included.

history of periodontal disease, the mean biologic width in tooth sites with a history of periodontal disease was in most cases increased. In the presence of gingival inflammation, the average biologic width was 1.25 ± 0.19 mm (Al-Rasheed et al. 2005). Lanning et al. (2003) observed an increase in the biologic width after surgical crown lengthening in sites with a mean attachment loss >6 mm (Table 3). The mean biologic width was 1.96 ± 0.05 mm 3 months post-surgery, and this value increased to 2.19 ± 0.06 mm 6 months post-surgery. Shobha et al. (2010) also detected a re-establishment of the biologic width after surgical crown lengthening in sites with a mean attachment loss of 3 – 6 mm. The mean value of the biologic width increased from 1.67 ± 0.62 mm 3 months post-surgery to 1.87 ± 0.83 mm 6 months post-surgery. According to the meta-analysis, the mean biologic width in subjects with attachment loss $\geq$3 mm amounted to 2.30 mm (CI 95% 2.19, 2.41) (Appendix S5b).

The presence of attachment loss was reported to influence the dimensions of the junctional epithelium (Orban & Köhler 1924, Gargiulo et al. 1961). Whereas the mean junctional epithelium around teeth without attachment loss was 1.35 mm, it was reduced to 0.71 mm in the case of an attachment loss of up to 6.08 mm (Gargiulo et al. 1961). The lower limiting values of the junctional epithelium obtained in this study were primarily based on teeth with attachment loss, whereas the upper maximum values were derived from teeth without attachment loss.

Differences in the mean values of the connective tissue dimensions were not documented for teeth with and without attachment loss (Gargiulo et al. 1961). However, significant variability of individual values of the connective tissue dimensions was observed.

Discussion

The marginal compartments of the periodontium have been analyzed and debated for several decades (Schroeder & Listgarten 2003). In the early part of the last century, Bernhard Gottlieb (1921) described a strong association between the tooth surface and the gingival epithelium. The first publication identified in this systematic review reported data from this Vienna group on the dimensions of the junctional epithelium and connective tissue attachment (Orban & Köhler 1924). In contrast, Jens Waerhaug (1952) reported a weak attachment of epithelial cells to the adjacent tooth. Improvements in electron microscopy were milestones in periodontal research for improving the understanding of the structure of the junctional epithelium. Using this technique, Schroeder and Listgarten (1971) published data on the components and structure of the junctional epithelium. They described the attachment of the epithelium to the tooth surface via a basement lamina and hemidesmosomes.

The junctional epithelium is an important part of the protective physiological barrier termed the biologic width by Cohen (1962). The biologic width is defined as the junctional epithelium and supracrestal connective tissue attachment – without the depth of the gingival sulcus – surrounding every tooth. This complex protects the subjacent periodontal ligament and the alveolar bone from the attack of a pathogenic biofilm present in the oral cavity (Bosshardt & Lang 2005). Evidence from different types of studies and a recent review suggests that a breach of the biologic width have an impact on periodontal health (Newcomb 1974, Tal et al. 1989, Günay et al. 2000, Padbury et al. 2003).

Table 6. Influence of patient- and tooth-related factors on the dimensions of the connective tissue attachment according to tooth type and tooth site. Mean values ± standard deviations (range of individual values) in mm

Author (year of publication)	Total	Method of analysis			Tooth type				Tooth site				
		Histologically	Clinically	Clinically/ radio-graphically	Anterior	Posterior	Premolars	Molars	Mesial	Distal	Buccal	Oral	Approximal (mesial + distal)
Orban & Köhler (1924)	(0.00–6.52)	(0.00–6.52)	–	–	(0.00–6.52)	–	(0.00–2.56)	(0.10–5.92)	(0.00–2.48)	(0.00–2.98)	(0.16–6.52)	(0.00–3.76)	(0.00–2.98)
Stanley (1955)	0.99 (0.1–2.3)	0.99 (0.1–2.3)	–	–	nr	nr	nr	nr	nr	nr	–	–	0.99 (0.1–2.3)
Gargiulo et al. (1961)*	1.07 (0.00–6.52)	1.07 (0.00–6.52)	–	–	nr	nr	nr	nr	(0.75–1.02)†	(0.69–1.10)†	(1.01–1.53)†	(1.03–1.49)†	(0.69–1.10)†
Vacek et al. (1994)	0.77 ± 0.29 (0.29–1.84)	0.77 ± 0.29 (0.29–1.84)	–	–	0.71 ± 0.24 (0.35–1.34)	nr	0.77 ± 0.31 (0.29–1.84)	0.89 ± 0.31 (0.40–1.77)	nr	nr	nr	nr	nr
Xie et al. (2007)	1.10 ± 0.13	1.10 ± 0.13	–	–	1.11 ± 0.12	1.10 ± 0.14	nr	nr	1.02 ± 0.10	1.05 ± 0.09	1.13 ± 0.13	1.21 ± 0.12	nr
Xie & Chen (2007)	1.10 ± 0.13	1.10 ± 0.13	–	–	1.09 ± 0.12	nr	1.09 ± 0.14	1.11 ± 0.14	0.95 ± 0.13	1.02 ± 0.16	1.17 ± 0.13	1.31 ± 0.12	nr

*material in Gargiulo et al. (1961) included measurements of specimens published by Orban and Köhler (1924).
†range of mean values; nr, data not reported; –, no sites presenting the related outcome parameter in the studies included.

Recently, the interactions between dental crowns and the marginal periodontal tissues were analyzed in a systematic review (Kosyfaki et al. 2010). It was concluded that the recognition of the biologic width, in terms of crown margin placement, is beneficial for periodontal health. Therefore, knowledge of the dimensions of the junctional epithelium and connective tissue attachment is of clinical relevance.

Herein, we conducted a systematic review to evaluate the dimensions of the biologic width. A total of fourteen publications were included from a systematic literature search. Data were exclusively collected from human studies.

In the included studies, the mean dimensions of the biologic width ranged from 1.15 to 3.95 mm (Novak et al. 2008, Ganji et al. 2012). Intra- and inter-individual variances did not permit the determination of a "magic number" of the biologic width. Therefore, the data from meta-analyses obtained either from studies using histological or clinical measures just provide a statistically calculated value on the dimensions of the biologic width (Appendix S5). The tooth type, tooth site, presence of restoration, healing time after surgical crown lengthening, and periodontal disease, including attachment loss and increased probing depths, were identified as factors that possibly affect the biologic width (Tables 1–7).

With regard to the substantial observed variance in biologic width, several potential confounding factors should be taken into consideration:

(i) The approaches used to measure the biologic width were histological and clinical techniques or a combination of clinical and radiological methods of assessment. The histological approach has been suggested to enable the most precise measurement (Orban & Köhler 1924, Stanley 1955, Gargiulo et al. 1961, Vacek et al. 1994, Xie & Chen 2007, Xie et al. 2007). Histological and clinical data may therefore considered separately as displayed in this review (Table 1, Appendix S5). Qualitative and quantitative discrimination between the junctional epithelium and connective

Table 7. Influence of patient- and tooth-related factors on the dimensions of the connective tissue attachment according to presence of a restoration, probing depth and attachment loss. Mean values ± standard deviations (range of individual values) in mm

	Restoration		Probing depth				Attachment loss		
Author (year of publication)	Existent**	Non-existent**	<2 mm	2–4 mm	>4–7 mm	>7 mm	<3 mm	3–6 mm	>6 mm
Orban & Köhler (1924)	–	–	(0.00–6.52)	(0.48–1.66)	0.52, 1.06	–	(0.00–6.52)	(0.00–5.92)	0.80
Stanley (1955)	–	–	nr	nr	nr	nr	nr	nr	nr
Gargiulo et al. (1961)*	–	–	nr	nr	nr	nr	1.08, 1.07, 1.06[†] (0.02–4.38)	1.06[‡] (0.00–6.52)	–
Vacek et al. (1994)	0.84 ± 0.26 (0.42–1.47)	0.76 ± 0.29 (0.29–1.84)	0.84 ± 0.26 (0.42–1.47)[¶]	–	–	–	0.84 ± 0.26 (0.42–1.47)[¶]	–	–
Xie et al. (2007)	–	–	1.10 ± 0.13[§]	–	–	–	nr	nr	nr
Xie & Chen (2007)	–	–	nr	nr	nr	nr	nr	nr	nr

*material in Gargiulo et al. (1961) included measurements of specimens published by Orban and Köhler (1924).
[†]mean values of groups I-III (attachment loss ranged from 0.00 to 2.36 mm).
[‡]mean value of group IV (attachment loss ranged from 0.39 to 6.08 mm).
[§]probing depth <3 mm.
[¶]related outcome parameter (probing depth or attachment loss) available as mean ± SD of all sites.
**bar (–) means that data are not available; nr, data not reported; –, no sites presenting the related outcome parameter in the studies included.

tissue attachment is possible by histological methods, with the caveat that the laboratory preparation may introduce artifacts into the specimens. The application of a histological method is often precluded in a clinical situation for ethical reasons. Different clinical methods, including the measurement of the gingival margin and the attachment level by periodontal probing and the evaluation of the alveolar bone level by transgingival probing, were employed (Lanning et al. 2003, Al-Rasheed et al. 2005, Shobha et al. 2010, Galgali & Gontiya 2011, Ganji et al. 2012). Transgingival probing following the administration of local anesthesia seems to be an accurate and reliable method for estimating the alveolar bone level and to detect osseous defects (Greenberg et al. 1976, Ursell 1989, Mealey et al. 1994, Perez et al. 2007). However, the probing force determines the penetration depth of the probe into the subjacent periodontal tissues (van der Velden 1979). In addition, the accuracy of periodontal probing depends on the inflammatory state of the periodontal tissues (Armitage 1996). In the presence of inflammation, the periodontal probe may penetrate the junctional epithelium and stop at the most coronal part of the non-inflamed connective tissue ligament (Listgarten et al. 1976, Magnusson & Listgarten 1980). A combined clinical and radiographic method using a gutta-percha point inserted into the gingival sulcus and two radiographic images was also described (Alpiste-Illueca 2004, 2012, Galgali & Gontiya 2011). This approach seems to produce reliable results; however, its use is restricted to anterior teeth for technical reasons, and needs to consider the radiation dose.

(ii) The dimensions of the biologic width seem to differ with respect to periodontal health. In the presence of gingival inflammation, the dimension of the biologic width was decreased compared with the dimensions at non-inflamed sites (Al-Rasheed et al. 2005). In addition, the biologic width appears to differ with respect to attach-

ment loss (Orban & Köhler 1924, Gargiulo et al. 1961) and in patients with untreated chronic periodontitis (Novak et al. 2008). In contrast, two studies state there is no impact of attachment loss or other signs of periodontal disease on the dimensions of the biologic width (Stanley 1955, Vacek et al. 1994). However, they did not report distinctive data.

(iii) According to one study, restored teeth seem to differ in terms of the junctional epithelium and connective tissue attachment dimensions compared with those of non-restored teeth (Vacek et al. 1994). However, the locations of the restoration margins were subgingival, and there were no available data regarding the marginal fit or the time period since insertion of the restorations. Inappropriate adaptation of the restoration margin and breach of the biologic width due to a subgingival restoration location will promote gingival inflammation due to increased local plaque accumulation (Silness 1970, Newcomb 1974, Lang et al. 1983).

(iv) Certain clinical situations require lengthening of the clinical crown by surgical techniques. Three studies provided data regarding periodontal tissue remodeling after surgery. Lanning et al. (2003) and Shobha et al. (2010) suggested that a time period of at least 6 months is needed for the re-establishment of the biologic width after surgical crown lengthening. This timeframe was confirmed by two additional publications. Herrero et al. (1995) showed that the desired amount of supracrestal tooth structure could not be routinely achieved after 2 months postsurgery, and Brägger et al. (1992) demonstrated that stable periodontal conditions were obtained in most of the reported cases after 6 months.

Conclusions and Clinical Relevance

In summary:

(1) There is significant intra- and inter-individual variability in the dimensions of the biologic width.
(2) A "magic number" for the biologic width as a treatment objective cannot be recommended, as the use of mean values could mask the actual clinical situation.
(3) Mean values of the biologic width obtained from two meta-analyses ranged from 2.15 to 2.30 mm.
(4) Periodontal and transgingival probing may be helpful in determining the dimensions of the junctional epithelium and connective tissue attachments.
(5) The dimensions of the biologic width seem to be affected by periodontal diseases.
(6) Periodontal health is supposed to be established prior to assessment of the biologic width.
(7) The completion of remodeling after surgical crown lengthening procedures may require at least 6 months.

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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Appendix S1. PRISMA 2009 checklist.
Appendix S2. Electronic search strategy for the database Web of Science.
Appendix S3. Scores for assessment of methodological and reporting quality.
Appendix S4. Heterogeneity regarding methods and outcome parameters in included studies.
Appendix S5. Meta-analyses. (a) Dimensions of the biologic width on anterior and posterior teeth in humans without a reported history of periodontal disease (histological studies). (b) Dimensions of the biologic width in humans with attachment loss of ≥ 3 mm (clinical studies).
Appendix S6. Studies excluded based on full text analysis, and reason for exclusion.
Appendix S7. Study characteristics of the included studies.
Appendix S8. Quality assessment (methodological and reporting scores) of included studies.
Appendix S9. Directions for further research.

Address:
Clemens Walter
Department of Periodontology, Endodontology and Cariology
University of Basel
Hebelstrasse 3, CH-4056 Basel
Switzerland
Email: clemens.walter@unibas.ch

Clinical Relevance

Scientific rationale for the study: For maintaining periodontal health in reconstructive dentistry, it is suggested to respect the biologic width.
Principal findings: In this systematic review, several patient- and tooth-related confounding factors and a significant intra- and inter-individual variability in the dimensions of the biologic width were identified. Data from meta-analyses on the biologic width may provide a careful estimate for clinical use.
Practical implications: Periodontal and transgingival probing after the application of local anesthesia may be helpful in determining an individual's biologic width. Periodontal health is supposed to be established in advance of biologic width assessment.