Gingival recession and root coverage outcomes in smokers

Mehmet Gümüş Kanmaz1, Burcu Kanmaz2, Nurcan Buduneli3

ABSTRACT
Smoking is considered as the major environmental risk factor for periodontal diseases. Smokers have a higher risk for severe periodontitis with more periodontal tissue destruction, more gingival recession, and more susceptibility to tooth loss. The clinical outcomes of periodontal treatment are also adversely affected by smoking. The aim of this narrative review is to provide up-to-date evidence on the clinical outcomes of root closure in smokers. Electronic databases were searched for studies that compare the clinical outcomes in smokers and non-smokers following surgical procedures for root coverage. The clinical studies published before February 2022 were included in the review. Similar or significantly better root coverage rates have been reported in non-smokers compared to smokers. Although there are controversial findings in the literature, the majority of clinical follow-up studies suggest that non-smokers respond better than smokers to surgical interventions aiming at root closure. Smokers tend to respond less favorably to surgical interventions performed for root coverage. Smokers may be encouraged to quit smoking during non-surgical periodontal treatment that precedes surgical interventions.

INTRODUCTION
Cigarette smoke contains a wide variety of chemicals. Carbon monoxide, hydrogen cyanide and nitrogen oxides, formaldehyde, acrolein, benzene and N-nitrosamines, nicotine, phenol, polyaromatic hydrocarbons, and tobacco-specific nitrosamines are among these chemicals that are hazardous to human health1.

During smoking, the harmful chemicals first encounter the tissues of the oral cavity. Melanosis, which mostly occurs in the form of benign pigment increase in the vestibular gingiva and interdental papilla in the anterior region of both upper and lower jaws, is perceived as an annoying aesthetic problem2. Smoking is among the major aetiological factors for melanosis together with genetic factors, various drugs or various systemic diseases3. Smoking affects gene expressions of the epithelium in the respiratory tract, decreases immune resistance of the oral mucosa and damages buccal mucosa4. Moreover, nicotine, with its pharmacokinetic effect, stimulates the sympathetic nervous system and facilitates plaque accumulation on the tooth and soft tissue surfaces by reducing saliva secretion5.

A relationship between periodontal diseases and smoking was first reported by Pindborg6 in terms of a higher prevalence of acute necrotizing ulcerative gingivitis in smokers. Smokers have a much higher risk for periodontitis7 and when individuals with similar plaque levels were compared, pocket depth and attachment loss are higher in the smokers8. Smoking negatively affects the immune system. While the number of leukocytes in the circulation increases in smokers, fewer defence cells can migrate into the gingival groove/pocket7,9. Circulation in periodontal tissues deteriorates, formation and functions of vascular structures are adversely affected10.

Clinical findings from a study comparing non-smokers, smokers, and passive smokers provided further support for the adverse and dose-dependent effect of tobacco products consumption on periodontal health11. The prevalence of Treponema denticola was higher in smokers possibly explaining at least partially the increased occurrence and
severity of periodontal tissue destruction. In a recent study, the outcomes of non-surgical periodontal treatment were investigated in smoker and non-smoker patients with Stage III and IV periodontitis in terms of clinical periodontal, microbiological, and biochemical parameters and follow-up at 6 months revealed that Gram-negative bacteria colonise faster in smokers.

**DEVELOPMENTS**

**Gingival recession**

Pink aesthetics and smile design are popular, particularly among young individuals. Gingival recession (GR) that makes the teeth look longer disturbs the pink aesthetics and is a common finding in the adult population. According to the Glossary of the American Academy of Periodontology, gingival recession is described as the exposure of the root surface by an apical shift of the gingiva with respect to the cemento-enamel junction. By definition, gingival recession is always associated with clinical attachment loss and can be localized or generalized affecting one tooth or several teeth in the same patient and may involve one or more surfaces of the same tooth.

Gingival recession can be associated with gingivitis or periodontitis, or it may develop due to traumatic occlusion, tissue trauma, proliferation of the pocket epithelium into the gingival connective tissue and its subsequent anastomosis with the outer epithelium as an extension of periodontal inflammation, traumatic tooth brushing and/or iatrogenic factors. The primary pathogenic factors (i.e. periodontal inflammation) and the local anatomic factors, which are environmentally conductive, not only affect the formation but also the quality and morphology of gingival recession lesions.

**Root coverage procedures**

Root coverage procedures which have been used for the treatment of gingival recession are successful and predictable interventions in periodontics. Aesthetics, dental hypersensitivity, and the prevention of caries and non-carious cervical lesions are considered the main indications for root coverage procedures. A number of different surgical techniques have been described and used for root coverage: lateral sliding flap, double papilla positioned flap, free gingival graft, lateral positioned flap, coronally advanced flap with free gingival graft, coronally advanced flap with a subepithelial connective tissue graft, semilunar flap, and coronally positioned flap. Coronal advanced flap (CAF) with subepithelial connective tissue graft is considered as the gold standard since it offers a greater probability of achieving complete root coverage when compared with other techniques. The major goal for successful root coverage is to move the gingival margin up to the CEJ with a probing sulcus depth of 2 mm and no bleeding on probing, no hypersensitivity issues. Several factors affect the outcome of root coverage procedures and smoking is one of the most important factors.

**Effects of smoking on root coverage procedures**

Numerous studies have evaluated the possible effects of smoking on mucogingival surgical interventions (Table 1). The first study investigating the relationship between root coverage and smoking was published by Tolmie et al.. They observed no adverse effects with cigarette smoking and obtained 100% root coverage in 11 of 12 (92%) sites in smokers. However, the number of cigarettes smoked or the duration of smoking was not reported. In another study, gingival recession in non-smokers, light-smokers ($\leq$10 cigarettes/day), and heavy-smokers ($\geq$10 cigarettes/day) were treated by double pedicle graft and subepithelial connective tissue graft. No difference in clinical outcomes was found between the three study groups. On the other hand, in a clinical study by Trombelli et al., reported that the rate of root coverage was lower in smoker patients than that obtained in non-smokers. In another clinical study, connective tissue graft was applied with the envelope technique modification and attachment gain was negatively affected by smoking. Zucchelli et al., treated 54 teeth with gingival recession, and stated that smoking decreased the expected root coverage amount by 0.52 mm. In contrast, Amarante et al. reported that 62% of heavy smokers ($\geq$20 cigarettes/day) had complete root coverage in comparison to 42% of non-smokers in the non-membrane group, and 37.5% of heavy smokers ($\geq$20 cigarettes per day) had complete root coverage in comparison to 16.7% of non-smokers in the membrane group. On the other hand, Hirsch et al. found similar root coverage rates in smokers and non-smokers.

In time, complete coverage became the desired ultimate goal of root coverage interventions. Therefore, the effects of smoking on complete root coverage have been investigated in more recent studies. Martins et al. found less root coverage, less clinical attachment gain, and deeper probing depth values in the smokers in a 6-month follow-up clinical study. Furthermore, no complete root coverage was obtained in the smokers. Likewise, in another study, the rate of root coverage was found to be lower in smoker patients. Moreover, the recession depths were statistically significantly larger in smokers than those in non-smokers. Silva et al. presented the 6- and 24-month results comparing root coverage in smokers and non-smokers using the coronally repositioned flap technique. In the 6 months results, the smoker patients presented greater residual recession depths and a lower rate of root coverage. Silva et al. found similar root coverage rates in smokers and non-smokers. According to the 24-month results of the study, recession depths significantly increased in both groups (in the smokers: from 0.84 ± 0.49 mm to 1.28 ± 0.58 mm; and in the non-smokers: from 0.22 ± 0.29 mm to 0.50 ± 0.41 mm) between follow-up at 6 and 24 months. Half of the smokers and 10% of the non-smokers exhibited between 0.5 and 1.0 mm of recurrence of gingival recession during this period. Smokers had significantly greater residual...
Table 1. Studies evaluating the effects of smoking on mucogingival surgical interventions

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<tr>
<th>Study</th>
<th>Study design and duration</th>
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<tr>
<td>Tolmie et al. 23 1991</td>
<td></td>
<td>74 Patients (69 male and 5 female)</td>
<td>Double pedicle graft + SCTG • Non-smokers • Light-smokers (≤10 cigarettes/day) • Heavy-smokers (≥10 cigarettes/day)</td>
<td>GRH</td>
<td>Saw no adverse effects of cigarette smoking. They obtained 100% root coverage in 11 of 12 (92%) sites in smokers. Their mean root coverage for smokers was 97.9%. However, no mention was made as to how much the patients smoked.</td>
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<tr>
<td>Harris 24 1994</td>
<td>Clinical study, 8 to 72 weeks follow-up (mean 23 weeks)</td>
<td>74 Patients (69 male and 5 female) Age 18–48 years 100 recession defects Miller’s Class I and II recessions</td>
<td>Double pedicle graft + SCTG • Non-smokers • Light-smokers (≤10 cigarettes/day) • Heavy-smokers (≥10 cigarettes/day)</td>
<td>GRH</td>
<td>No difference between non-smokers, light-smokers (≤10 cigarettes/day), and heavy-smokers (≥10 cigarettes/day) Complete root coverage obtained 89% RC Non-smokers 97.6% RC Light-smokers 96.6% RC Heavy-smokers 98.5%</td>
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<td>Trombetti et al. 25 1997</td>
<td>Retrospective study, 6 months duration</td>
<td>22 Patients Aged 23–40 years Miller’s class I and class II recessions</td>
<td>Guided tissue regeneration with e-PTFE membrane 9 Smokers (&gt;10 cigarettes/day at initial examination) 13 Non-smokers</td>
<td>PD</td>
<td>Membrane exposure significantly greater in smokers Newly formed tissue gain is not statistically different between groups Smokers significantly less RD reduction (2.5 ± 1.2 mm vs 3.6 ± 1.1 mm) and root coverage (57% vs 78%) Complete root coverage was observed in one smoker (11%) and five non-smokers (38%)</td>
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<td>Müller et al. 26 1998</td>
<td>Clinical trial study, 12 months duration</td>
<td>22 Patients (4 patients dropped-out from smokers) Aged 22–73 years 18 Patients 28 recession sites included Miller’s class I and class II recessions</td>
<td>Connective tissue graft + envelope technique modification 3 Smokers 15 Non-smokers</td>
<td>PD</td>
<td>Attachment level alteration during the postoperative observation period was negatively influenced by the location of the recession in the maxilla and by cigarette smoking (R²=0.395, p&lt;0.001)</td>
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| Zuchelli et al. 1998  | Randomized clinical trial, 12 months duration | 54 Subjects (29 female and 25 male) Aged 23–33 years 54 recession defects Miller’s Class I and II recessions | Compare the clinical efficacy of 3 surgical approaches  
GTR + bioabsorbable membrane  
GTR + non-resorbable membrane  
CAF + SCTG  16 Patients were smokers (≥10 cigarettes/day) | PD       | Smoking decreases the expected coverage to 0.52 mm |
| Amarente et al. 2000  | Controlled clinical trial, 6 months duration | 20 Patients, mean age 38.4 years Bilateral Miller Class I and II recessions Total 40 sites | Coronally positioned flap, alone or combined with bioabsorbable membrane  
8 Smokers (≥20 cigarettes/day)  
12 Non-smokers | PD       | 62% of heavy smokers (≥20 cigarettes/day) had complete root coverage compared to 42% of non-smokers in the non-membrane group  
37.5% of heavy smokers (≥20 cigarettes/day) had complete root coverage compared to 16.7% of non-smokers in the membrane group |
| Hirsch et al. 2001   | Clinical study, mean follow-up 32.68 months | 25 Patients (17 male and 8 female) Aged 23–48 years Miller Class I and II recessions 44 recessions | Subepithelial connective tissue graft with coronally positioned flap  
9 Non-smokers  
16 Smokers  
11 Patients less than 10 cigarettes/day  
5 Patients 10–20 cigarettes/day | PD       | No significant differences in root coverage between smokers and non-smokers |
| Martins et al. 2004  | Prospective clinical study, 6 months duration | 15 Patients Aged 27–55 years Miller’s Class I and II recessions 18 recessions defects | Coronally positioned flap with subepithelial connective tissue graft  
7 Smokers (20 cigarettes/day for >5 years)  
8 Non-smokers | PD       | Lower RC in smokers 58.84 ± 13.68% vs non-smokers 74.73 ± 14.72%  
Less CAL gain in smokers 2.00 ± 1.04 mm vs non-smokers 2.54 ± 0.79 mm  
Deeper PD in smokers 2.35 ± 0.67 mm vs non-smokers 1.56 ± 0.53 mm  
Post-op 4th month smokers presented more keratinized tissue 4.50 ± 1.16 mm vs 3.30 ± 0.86 mm p<0.05  
Complete root coverage was observed in 35% of the non-smokers and apparently not in the smokers |

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| Erley et al. 31 2006| A comparative clinical study, 6 months duration | 17 Patients (16 male and 1 female) Aged 27–45 years Miller's class I and II recessions 22 recession defects | Connective tissue graft Smokers (10–20 cigarettes/day and >10 ng/mL cotinine level) Non-smokers (0–10 ng/mL cotinine level) | PD  RCAL  GRH  GRW  KTW  RC  CRC  Saliwary cotinine level | • RD 6 months statistically significant larger for smokers than non-smokers (1.0 ± 0.85 mm and 0.20 ± 0.42 mm, respectively)  
• RC at 6 months was 82.33 ± 14.90% for smokers and 98.3 ± 4.42% for non-smokers. This was statistically significant (p=0.001)  
• Only 25% of smokers healed with complete root coverage compared to 80% of non-smokers |
| Silva et al. 32 2006| Prospective clinical trial, 6 months duration | 20 Patients Aged 22–53 years Miller’s class I recessions | Coronally positioned flap 10 Smokers (>10 cigarettes/day at least for 5 years) 10 Non-smokers | PD  CAL  GRH  KTW  RC  CRC | • Smokers presented greater residual RD at 6 months (0.84 ± 0.49 mm and 0.22 ± 0.29 mm, respectively) and lower percentage of root coverage (69.3% vs 91.3%, p<0.05)  
• No smokers obtained complete root coverage compared to 50% of non-smokers |
| Silva et al. 33 2007| Prospective controlled clinical trial, 24 months duration | 20 Patients Aged 22–53 years Miller’s class I recessions | Coronally positioned flap 10 Smokers (>10 cigarettes/day at least for 5 years) 10 Non-smokers | PD  CAL  GRH  KTW  RC  CRC | • RD significantly increased in smokers (from 0.84 ± 0.49 mm to 1.28 ± 0.58 mm) and in non-smokers (from 0.22 ± 0.29 mm to 0.50 ± 0.41 mm) between 6 and 24 months  
• 50% of smokers and 10% of non-smokers lost between 0.5 and 1.0 mm of root coverage in the same period  
• Smokers had significantly greater residual recession (p=0.001) at 24 months  
• Both smokers and non-smokers lost CAL and experienced decreases in KT |
| Souza et al. 34 2008| Controlled clinical trial, 6 months duration | 30 Patients (20 male and 10 female) Aged 24–47 years Miller's class I and II recessions | Subepithelial connective tissue graft with coronally positioned flap 15 Smokers (>10 cigarettes/day) 15 Non-smokers | PD  RCAL  GRH  KTW  RC  CRC | • Smokers had less root coverage than non-smokers (58.02 ± 19.75% versus 83.35 ± 18.53%, p<0.05)  
• Smokers had more GR (1.48 ± 0.79 mm vs 0.52 ± 0.60 mm) than the non-smokers (p<0.05)  
• Histomorphometry of the donor tissue revealed a blood vessel density of 49.01 ± 11.91 vessels/200x field for non-smokers and 36.53 ± 10.23 vessels/200x field for smokers (p<0.05)  
• CRC was 6.7% in smokers compared to 53.3% in non-smokers |

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| Andia et al. 2008 | A controlled clinical study, 24 months    | 22 Patients Aged 22–55 years Miller’s class I and II recessions           | Subepithelial connective tissue graft with coronally positioned flap 11 Smokers (>20 cigarettes/day for >5 years) 11 Non-smokers | PD, CAL, GRH, KTW, KTT, RC, CRC | • At 24 months postoperatively, statistical analysis showed that smokers presented poorer outcomes regarding PD, GR, and CAL (p<0.05); in addition, a more satisfactory stabilization of the gingival tissue was found in the non-smoker group  
• RC after 2 years was 50% (1.8 mm, range of residual recession: to 2.6 mm) and 77.8% (2.8 mm; range of residual recession: to 1.8 mm) for smokers and non-smokers, respectively  
• CRC was found in 27% of the non-smokers, whereas none of the smokers presented CRC |
| Reino et al. 2012 | A controlled clinical trial, 6 months     | 20 Patients (10 male and 10 female) Aged 35–50 years 40 gingival recessions | Subepithelial connective tissue graft with coronally positioned flap One side Langer-Langer technique, One side Barros technique  
All heavy smokers (≥20 cigarettes/day for >5 years) | PD, CAL, GRH, KTW, RC, CRC, Saliva cotinine analysis | • Both techniques promoted low root coverage (Control group: 43.18% and Test group: 44.52%)  
• No difference was found in root coverage between the techniques  
• CRC occurred in 2 cases at 6 months (5%) |
| Alves et al. 2012 | Randomized, controlled, split mouth design, 6 months | 19 Patients (12 female and 7 male) Aged 30–50 years 40 gingival recessions | Acellular dermal matrix graft + Emdogain vs Acellular dermal matrix graft  
All smokers (consuming ≥10 cigarettes/day for >5 years) | PD, RCAL, GRH, GRW, KTW, KTT, RC, CRC | • The percentage of root coverage was 55.4% for the ADMG + EMD and 44.0% for the ADMG group  
• The ADMG + EMD group showed CRC in three gingival recessions, whereas the ADMG group showed in one gingival recession  
• Considering the number of sites with CRC, there was a statistical difference between the groups |
| Nanavati et al. 2013 | Controlled clinical trial, 6 months        | 20 Patients (14 male and 6 female) Aged 22–53 years Miller’s class I recessions | Coronally positioned flap 10 Smokers (≥10 cigarettes/day for >5years) 10 Non-smokers | PD, CAL, GRH, GRW, KTW, RC, CRC | • No smokers obtained complete root coverage compared to 30% of non-smokers (p<0.05)  
• Smokers presented greater residual RD at 6 months and lower percentage of root coverage (60.09% vs 76.05%, p<0.05) |

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| Jankovic et al. 2013 | Controlled clinical trial, 3 years duration | 55 Patients (29 male 26 female) Aged 30–41 years Miller’s Class I and II recessions | Subepithelial connective tissue graft with coronally positioned flap 30 Non-smokers 25 Smokers (≥20 cigarettes/day) 19 gen+ patients 36 gen- patients | GI CAL GRH IL-1 genotype RC CRC | • RC was similar in gen+ (92%) and gen- (93.2%) subjects within smoking and non-smoking groups after 1 year  
• For non-smokers, RC was obtained 75% in gen+ subjects and 88% for gen- subjects. Statistically significant difference detected  
• For non-smokers, CRC was for gen- 75%, gen+ 70% at 1 year After 3 years, these values decreased 55% and 30%, respectively  
• For smokers, RC was obtained 86% in gen+ subjects and 92% for gen- subjects. At 3 years, 57% root coverage for gen+ and 79% for gen- subjects obtained. Statistically significant difference detected  
• For smokers, CRC was for gen- 68.75%, gen+ 55.55% at 1 year. After 3 years, these values decreased 25% and 0%, respectively  
• In a 3-year period, non-smokers with IL-gen+ lost approx. 20% of the root coverage gained at 1 year and almost four times more inferior compared with gen- group  
• Patients who smoked and had a positive IL-1 gen+ lost approx. 35% of the gained root coverage. IL-1 polymorphism and smoking habit did not affect gingival recession at 1 year but had a great impact on long-term stability  
• Smokers who were presented with IL gen- and gen+ significantly increased risk for root coverage failure compared with non-smoking patients, 3 years after surgical treatment |
| Kaval et al. 2014 | Controlled clinical trial, 6 months duration | 32 Patients (11 male and 21 female) Aged 18–52 years Miller’s class I and II recessions 2 Patients dropped out from smoker group 36 defects 18 each | Coronally advanced flap (microsurgical) 15 Smokers (>10 cigarettes/day for >5 years) 15 Non-smokers | PD CAL GRH GRW KTW KTT RA RC CRC Cotinine level GCF samples | • CAL gain, percentage of root coverage and complete root coverage rates were similar in the study groups  
• RC at 6 months, 90.33 ± 17.84% smokers, 94.11 ± 12.00% non-smokers  
• CRC at 6 months, 66.70% smokers, 72.20% non-smokers not statistically different |
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<tr>
<td>Reino et al. 41</td>
<td>A pilot comparative clinical study, 12 months duration</td>
<td>20 Patients Bilateral Miller's class I and II gingival recessions 40 recession sites</td>
<td>CPF + SCTG vs EFT + SCTG 20 Smokers</td>
<td>PD, CAL, GRH, KTT, KTW, RC, CRC</td>
<td>• Percentage of root coverage, CPF group 48.60% and EFT group 54.28%</td>
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<tr>
<td>Costa et al. 42</td>
<td>The randomized clinical trial, 12 months duration</td>
<td>19 Patients Aged 30–50 years Bilateral Miller's class I and class II recessions 38 recession sites</td>
<td>Extended flap technique with Acellular dermal matrix graft + Emdogain or Acellular dermal matrix graft alone 19 Smokers (≥10 cigarettes/day for &gt;5 years)</td>
<td>PD, RGAL, GRH, GRW, KTT, KTW, RC, CRC</td>
<td>• Percentage of root coverage, ADMG + EMD group 59.7% and ADMG group 52.8%</td>
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<tr>
<td>Dwarakanath et al. 43</td>
<td>A pilot comparative clinical study, 6 months duration</td>
<td>20 Patients Aged 19–58 years Miller’s class I and class II recessions</td>
<td>Subepithelial connective tissue graft with coronally advanced flap 10 Non-smokers (≥5 cigarettes/day for ≥5 years) 10 Smokers • 7 Light smokers (5–10 cigarettes/day) • 3 Moderate smokers (10–20 cigarettes/day)</td>
<td>PD, CAL, GRH, GRW, KTT, RA, RC, CRC</td>
<td>• 60% non-smokers and 30% smokers showed CRC  • MRC was 71.2% in non-smokers and 38% in smokers</td>
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<td>Romanos et al. 44</td>
<td>Prospective case series, 12 months period</td>
<td>18 Patients Mean age 36.7 years Multiple recessions with Miller’s Class I, II and III 133 recession sites</td>
<td>Modified coronally advanced tunnel flap + Acellular dermal matrix graft 8 Smokers (&gt;10 cigarettes/day for ≥5 years) 10 Non-smokers</td>
<td>RD, KTW, VAS pain, RC, CRC</td>
<td>• RC, 82.0 ± 20.2% for smokers and 90.5% ± 16.2% for non-smokers  • CRC, 48.1% for smokers and 70.9% for non-smokers</td>
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<th>Results</th>
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| Saima et al.   | Controlled clinical trial, 6 months duration | 20 Patients (14 male and 6 female) Aged 22–53 years Miller’s Class I recessions | Coronally positioned flap 10 Smokers (≥10 cigarettes/day ≥5 years) 10 Non-smokers | PD       | • At 6 months, RD in the smoker group was significantly greater than the non-smoker group, when the average root coverage percentage was compared, smokers had a significantly lower percentage than non-smokers  
  • The frequency of complete root coverage was significantly greater in the non-smoker group  
  • In the smoker group average RC was 60%, while for non-smokers was 76%  
  • In the smoker group, CRC was 0%, while in the non-smoker group it was 30% at 6 months |
| Tawfik et al.  | Controlled clinical trial, 6 months duration | 22 Patients Aged 20–35 years Miller’s Class I and II gingival recessions | Free Gingival Graft 12 Non-smokers 10 Smokers (≥10 cigarettes/day) | PD       | • The non-smokers group showed a significant decrease of recession width and graft shrinkage area compared to the smokers group. The clinical parameters showed improvement in the non-smoker group more than smoker group, but the difference was not statistically significant |

recession (p=0.001) at 24 months. In another clinical study\textsuperscript{35}, root coverage rates were less in the smokers than in the non-smokers (58.02 \pm 19.75\% versus 83.35 \pm 18.53\%, p=0.003) and complete root coverage was obtained in 6.7\% of the recession defects in the smokers compared to 53.3\% in the non-smokers. Accordingly, Andia et al.\textsuperscript{36} stated that smokers responded poorly to the root coverage intervention and complete root coverage was not obtained in the 24-month follow-up clinical study.

Instead of comparing smokers and non-smokers, Reino et al.\textsuperscript{37} compared subepithelial connective tissue grafts with a coronally positioned flap and used Langer-Langer technique on one side and Barros technique on the other side, in heavy smokers. Both techniques were found to be unsuccessful in this group of patients. Similarly, Alves et al.\textsuperscript{38} compared the possible benefits of enamel matrix derivative (Emdogain\textsuperscript{®}) combined with acellular dermal matrix graft and only acellular dermal matrix graft application in smokers and reported that the clinical outcomes were slightly improved in the combination group. Complete root coverage was observed in three gingival recession defects in the combination group, whereas the acellular dermal matrix graft group showed complete root coverage only in one defect. Nanavati et al.\textsuperscript{39} compared the effects of smoking on coronally positioned flap outcomes and reported that no complete root coverage was obtained in the smokers, whereas 30\% complete root coverage was detected in the non-smokers (p<0.05) and smokers revealed greater residual recession depth and lower percentage of root coverage (60.09\% vs 76.05\%, p<0.05) at 6 months follow-up.

Jankovic et al.\textsuperscript{40} investigated the effects of smoking and IL-1 genotype on root closure outcomes with a 1-year follow-up study. Root coverage rates were similar in genotype+ (92\%) and genotype- (93.2\%) individuals within the smoker and non-smoker groups. Patients who smoked and were positive for IL-1 genotype lost approximately 35\% of the obtained root coverage. IL-1 polymorphism and smoking habit did not affect gingival recession at 1 year, but follow-up at 3 years revealed less stability in smokers. In another study\textsuperscript{41}, the coronally repositioned flap was performed with microsurgical technique in smokers and non-smokers with Miller I - II gingival recessions. The smoking status of the patients was chemically validated by salivary cotinine levels. At 6 months evaluation, complete root coverage rate was 66.70\% and 72.20\% in smokers and non-smokers, respectively, without statistically significant difference.

On the other hand, Reino et al.\textsuperscript{42} included only smoker patients and compared the coronally positioned flap and extended flap technique with regard to clinical success and reported similar outcomes. Accordingly, possible benefits of enamel matrix derivative (Emdogain\textsuperscript{®}) were investigated in a split-mouth study conducted only on smokers\textsuperscript{43}. While the extended flap technique and acellular dermal matrix grafts were applied on one side, enamel matrix derivative was applied additionally on the other side. No significant difference was found between the two treatment approaches. The possible effects of smoking on root coverage surgery were investigated in another study and 60\% of the non-smokers and 30\% of the smokers showed complete root coverage\textsuperscript{44}. Romanos et al.\textsuperscript{45} investigated the effects of smoking on the modified coronally advanced flap technique with a cellular dermal matrix graft procedure and reported rather low success rates in smokers compared to non-smokers with complete root coverage ratios of 48.1\% and 70.9\%, respectively.

Coronally advanced flap was used in another study comparing the clinical outcomes of root coverage between smoker and non-smoker patients\textsuperscript{46}. At 6 months follow-up, gingival recession depth in the smoker group was significantly greater than in the non-smoker group with a significantly lower percentage of root coverage in smokers. The non-smoker group exhibited a significantly greater rate of complete root coverage. The mean root coverage rate was 60.09\% in smokers and 76.05\% in non-smokers. In another study\textsuperscript{47}, using the free gingival graft procedure, significantly better outcomes were obtained in the non-smokers in terms of the decrease in recession width and graft shrinkage compared to the smokers.

**CONCLUSION**

Within the limits of the available studies and within the context of a non-systematic narrative literature review, it may be concluded that smoking adversely affects the clinical success of various surgical techniques used for root coverage. This fact may encourage smokers who are concerned about dental aesthetics to quit smoking along with non-surgical periodontal treatment.

**REFERENCES**


CONFLICTS OF INTEREST
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AUTHORS’ CONTRIBUTIONS
NB provided primary oversight to the organization of the review, as well as specific sections on periodontal treatment. MGK and BK provided the content for the review.

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