Examining peri-implant and periodontal conditions co-occur: a cross-sectional study (#105496)

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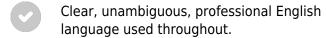
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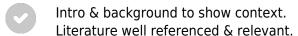
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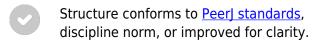
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Rigorous investigation performed to a high technical & ethical standard.

Methods described with sufficient detail & information to replicate.

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I commend the authors for their extensive data set, compiled over many years of detailed fieldwork. In addition, the manuscript is clearly written in professional, unambiguous language. If there is a weakness, it is in the statistical analysis (as I have noted above) which should be improved upon before Acceptance.



Examining peri-implant and periodontal conditions co-occur: a cross-sectional study

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Introduction: Both peri-implant and periodontal conditions have underlying main factors, risk factors, microbiology, immunology, and treatments. Aims: This study aims to investigate the potential co-occurrence of peri-implant and periodontal conditions **Design:** One hundred twenty-three implants were divided into three groups: peri-implantitis (41 implants), peri-implant mucositis (41 implants), and peri-implant health (41 implants). Periimplant and periodontal statuses were assessed using the 2017 AAP / EFP World Workshop on Classification of Periodontal and Peri-implant Diseases and Conditions. One-way Analysis of Variance was used to compare the study groups according to the data. An assessment was conducted regarding the coexistence of periodontal and peri-implant conditions. Results: While patients with peri-implant mucositis mostly had gingivitis and patients with peri-implant health had periodontal health, those with peri-implantitis mostly had gingivitis and relatively less periodontitis. A significant difference was observed between the peri-implant and periodontal groups (p=0.003). Significant differences were observed between peri-implant and periodontal evaluations for plague indices, gingival indices, probing depth, gingival recession, and clinical attachment level (p=0.001), (p=0.006). **Conclusions:** The findings of this study underscore the intricate influence of implant treatment on periodontal health. This observation emphasizes the importance of elucidating underlying factors to improve clinical management and outcomes in patients with periodontal and peri-implant diseases, highlighting this research's relevance and potential impact in the field.



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2	Examining Peri-İmplant and Periodontal Conditions Co-
3	occur: A Cross-Sectional Study
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35	implant diseases, highlighting this research's relevance and potential impact in the field.
36	Reywords: Periodontal disease; peri-implant health; peri-implant mucositis; peri-implantitis
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Introduction

- 39 Establishing and maintaining oral health involves monitoring both peri-implant and periodontal
- 40 conditions. Periodontal health and peri-implant health are defined by the absence of bleeding,
- 41 swelling, or suppuration on probing, coupled with the absence of clinically evident
- 42 inflammation. In peri-implant mucositis, akin to gingivitis in natural dentition, the initiation of
- 43 inflammation occurs with microbial plaque accumulation; however, there is no extension of
- increased probing depth to the alveolar bone (Berglundh et al., 2018), (Chapple et al., 2018).
- 45 Peri-implant mucositis is distinguished from peri-implantitis by triggering a localized
- 46 inflammatory response and ultimately leading to the loss of supporting bone around the implant
- 47 (Berglundh et al., 2018), (Renvert et al., 2018), (Lee & Wang, 2010). Likewise, periodontitis, an
- 48 enduring inflammatory condition, is typified by dysbiotic plaque biofilms and an immune
- 49 -- dysregulation that promotes the destruction of the periodontal ligament and alveolar bone
- 50 (Sedghi, Bacino & Kapila, 2021), (Papapanou et al., 2018).

Periodontitis and peri-implantitis are inflammatory conditions caused by biofilms that can result in tooth and oral implant loss if not addressed (Lasserre, Brecx, Toma, 2018). Compared with healthy areas in the same individual, inflamed regions (peri-implantitis and periodontitis) harbor unique dysbiotic subgingival microbial ecosystems (Barbagallo et al., 2022). Periodontitis and peri-implantitis have been reported to be associated with a notable increase in microbial stability within the subgingival microbiome (Zhang et al., 2021). Studies investigating peri-implant biofilms have predominantly focused on recognized periodontal pathogens like Porphyromonas gingivalis (P. gingivalis) and Treponema denticola. Furthermore, these results have highlighted similarities between the subgingival microbiota of periodontitis and periimplantitis (Kotsakis & Olmedo, 2021). In contrast, certain studies refute the notion of microbiota similarity between peri-implantitis and periodontitis (Koyanagi et al., 2013), (Maruyama et al., 2014). In a broad sense, risk factors encompass patient-related, environmental, or practitioner-related elements. Patient-related risk factors encompass socio-economic status, smoking habits, substance abuse disorders, diabetes, dietary habits and supplementation, mental health conditions, advanced age, inadequate home dental care, limited understanding of the importance of proper oral hygiene, genetic polymorphisms, and medication usage (Darby, 2022), (Kinane & Hart, 2003), (Vaz et al., 2012). Moreover, according to the report of the 6th Conference European Association for Osseointegration, prosthesis over-contouring and implant surface characteristics increase the risk of peri-implantitis (Schwarz et al., 2021).

Individuals with a history of periodontitis are more susceptible to peri-implant infections and complications (*Renvert & Persson, 2009*), (*Ferreira et al., 2018*). A history of periodontitis can be assessed by evaluating periodontal bone loss on radiographs, examining dental records, or talking to the patient to determine the cause of tooth loss. The patient can also tell the cause of tooth loss. It is reasonable to include the stage and extent of periodontal disease in this assessment as it influences the development and progression of peri-implant disease (*Heitz-Mayfield, Heitz & Lang, 2020*).

The peri-implant sulcus is histologically and immunologically distinct from the subgingival sulcus (*Robitaille et al.*, 2016). Increasing evidence has been obtained on the



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development and causes of periodontal and peri-implant disorders. Although there are some 79 80 similarities in the host's reactions in both settings, their differences can be attributed to the distinct composition of tooth-periodontium and implant-alveolar bone biointerfaces (Larsson et 81 al., 2022). The host response, pivotal in delineating the genetic basis of diseases like 82 83 periodontitis and peri-implantitis, necessitates an examination of cytokines, chemokines, growth 84 factors, and their receptors, crucial in understanding the pathogenesis of periodontal and peri-85 implant diseases (Turkmen & Firatli, 2022), (Genco, 1992).

Periodontal and peri-implant diseases are mainly managed using manual instrumentation to reduce the bacterial load and improve the patient's at-home cleanliness (Meffert, 1996). If required, further antibiotic therapies and laser treatments may be employed (Mombelli, Lang, 1992), (Hammami & Nasri, 2021), (Diwan et al., 2024), (Ashnagar et al., 2024). Regenerative treatment can also be applied (Larsson et al., 2016). Periodontitis and peri-implantitis reportedly have similar etiology and similar therapeutic interventions are performed in patients with the two entities (Robitaille et al., 2016).

Since there are some commonalities in primary factors, risk factors, microbiology, immunology, and treatment interventions, it is hypothesized that periodontitis may co-occur in the presence of peri-implantitis, gingivitis may manifest in the presence of peri-implant mucositis, and periodontal health may be observed in the presence of peri-implant health. This study hypothesizes that there is a significant association between the presence of periodontitis and peri-implantitis, gingivitis and peri-implant mucositis, and periodontal health and periimplant health. Therefore, this study aims to investigate the potential presence of periodontitis in patients with peri-implantitis, gingivitis in those with peri-implant mucositis, and periodontal health in those with peri-implant health.

Materials & Methods

This study complied with the World Medical Association Declaration of Helsinki for medical research (Emanuel, 2013).

Patients who were previously examined for periodontal/peri-implant status, except excluded ones, at Bolu Abant İzzet Baysal University between July 2022 and November 2023, were included in the study. This clinical study was registered at ClinicalTrials.gov (NCT06128850/23.07.2024). - The registration shows March 31,2023.

Ethical considerations

Bolu Abant İzzet Baysal University Clinical Researches Ethics Committee approved the study (2022/163-28/06/2022). The participants were informed about the procedures and signed the informed consent form.

Sample size calculation

It was planned to have three groups according to peri-implant health status in the study, and the sample size was calculated according to the study of Barwacz et al. (Barwacz, 2018) (2018). According to the results of power calculation using F test, fixed effects, special effects, main effects, and interaction analysis (G*Power 3.1 software; Heinrich Heine University,



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- Dusseldorf, Germany), with α (margin of error) = 0.05, power (1- β) = 0.90 and effect size (f)= 0.4, the required sample size for the three groups was 117, and the required sample size for each subgroup was at least 39. The effect size value was determined regarding the proposed large effect size convention.
 - Study design and participants

The study included 123 implants with fixed prostheses that had survived for at least six months following functional prosthetic loading, with the exception of patients with uncontrolled medical issues and referred clinical bruxism. Implants are 2–5 years old. The implant's fixed prosthesis is 1.5–4.5 years old. Two hundred twenty-four patients were evaluated, and 123 implants that fulfilled the inclusion criteria were included in the study. The plaque index (Silness & Löe, 1964), gingival index (Loe & Silness, 1963), probing depth, bleeding on probing (Ainamo & Bay, 1975), clinical attachment loss (CAL), and gingival recession were recorded for the teeth and implants of patients from the mesiobuccal, distobuccal, mid-buccal, mesiopalatinal/lingual, mid-palatinal/lingual, and distolingual/palatinal regions. All indices were taken during the examination. The peri-implant and periodontal health statuses of the patients were examined. Healthy gingiva, displaying an intact periodontium, exhibits minimal bleeding on probing (<10%) and shallow periodontal pocket depths (≤3 mm). In contrast, gingivitis is characterized by increased bleeding on probing ($\geq 10\%$) with pocket depths remaining ≤ 3 mm. Descriptions of periodontitis should encompass metrics such as the prevalence of bleeding on probing, the proportion of teeth with probing depths surpassing specified thresholds (commonly >4 mm and \geq 6 mm), and teeth exhibiting clinical attachment loss (CAL) of \geq 3 mm and \geq 5 mm (*Papapanou* et al., 2018). Peri-implant health was characterized by the absence of erythema, bleeding on probing, swelling, and suppuration. The main clinical characteristic of peri-implant mucositis is bleeding on gentle probing. Erythema, swelling, and/or suppuration may also occur. As outlined in the 2017 World Workshop on Periodontology guidelines, in cases where prior examination data is unavailable, diagnosing peri-implantitis may rely on concurrent indications such as bleeding or suppuration during gentle probing, probing depths measuring 6 mm or greater, and bone resorption levels reaching 3 mm or beyond apically from the most coronal aspect of the intra-osseous section of the implant (Berglundh et al., 2018). The implants were divided into three groups: peri-implantitis, peri-implant mucositis, and peri-implant health. Each group was evaluated according to periodontal status (periodontal health, gingivitis, and periodontitis). A single clinician (T.Ş.) recorded all measurements in a single session.

An assessment was conducted regarding the coexistence of periodontal and peri-implant conditions.

Inclusion and exclusion criteria

This study included systemically healthy patients aged 18-70 who had undergone at least six months and at most five years of functional prosthetic loading of one or more dental implants with a fixed prosthesis.

Pregnant or lactating women, patients with a history of chronic use of anti-inflammatory agents, and those on immunosuppressive drugs or drugs that impact the mucosa and bones were



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158	not included in the study. Patients who underwent treatment of peri-implant disease after implant
159	placement, those with residual cement residue and prosthesis design, and those with
160	malpositioned implants were also excluded from the study. Patients undergoing active
161	periodontal treatment or treatment after implantation, diabetic patients, patients with mucosal
162	diseases, and smokers were excluded.
163	Statistical analyses

Research analysis was conducted using the SPSS 26 (SPSS Inc., Chicago, IL, USA) statistical program. Shapiro Wilk normality tests were performed to determine whether the data met parametric test criteria. The study compared the three groups according to peri-implant health status. Paired sample t-tests were utilized to assess each group's implant and periodontal indices. The chi-square test was used to analyze categorical data. The level of significance was set at p<0.05. A total of 123 implants were analyzed, with 41 implants in each group based on peri-implant health status.

171 Results

1.1. **Demographic characteristic**

The demographic characteristics of the study population are presented in Table 1.

1.2. **Distribution of groups**

Implants were most commonly placed in relation to #16 (12.2%), #36 (11.4%), and #46 (8.9%). The peri-implantitis, peri-implant mucositis, and peri-implant health groups comprised an equal number of patients in (Table 2).

Comparison of groups

Individuals with peri-implantitis had higher rates of gingivitis and periodontitis, respectively. Periodontal health was primarily detected in patients diagnosed with peri-implant health and gingivitis was primarily detected in those with peri-implant mucositis. The most significant number of people diagnosed with peri-implant mucositis had gingivitis and periodontal health. A significant difference was observed between the peri-implant and the

periodontal groups (p=0.003) (Table 3). Lack of specificity: What kind of difference? In what neadure?

Comparison of indices

When peri-implant health and conditions were analyzed concurrently for plaque indices a significant difference was observed between the peri-implant and periodontal evaluations (p=0.001), (II=0.05-0.13, PI=0.19-0.30). The plaque index during periodontal evaluation was found to be greater than that during implant evaluation (Table 4).



190 A significant difference was observed in the context of gingival indices between periimplant and periodontal evaluations in patients with peri-implantitis and peri-implant mucositis 191 (p=0.001), (II=0.07-0.76, PI=0.07-0.14). The gingival index during implant evaluation was 192 found to be greater than that during periodontal evaluation (Table 4). 193 194 In patients diagnosed with peri-implantitis, a statistically significant variance in probing 195 depth was observed between assessments of peri-implant and periodontal regions (p=0.001), (II=3.24-4.59, PI=2,09-2,25). The probing depth during implant evaluation was found to be 196 197 greater than that during periodontal assessment. Regarding the probing depth, there was not any 198 significant difference between implant and periodontal assessments in patients with peri-implant 199 mucositis and peri-implant health (p=0.165), (p=0.837), (II=2.00-2.28, PI=1.70-2.26), (II=1.82-200 2.12, PI=1.98-2.14) (Table 4). 201 In the context of gingival recession, a significant difference was observed between peri-202 implant and periodontal recession in patients with peri-implantitis and peri-implant mucositis 203 (p=0.001), (p=0.014), (II=0.27-1.17, PI=0.06-0.31), (II=0.04-0.46, PI=0.05-0.18). Periodontal 204 evaluation revealed that gingival recession in implants was greater than that in teeth. 205 Furthermore, no notable distinction was found regarding gingival recession between assessments 206 of implants and periodontal health in patients diagnosed with peri-implant health (p>0.05), (p=0.410), (II=0.02-0.29, PI=0.08-0.38) (Table 4). 207 208 A significant difference was observed in terms of CAL between peri-implant and periodontal evaluations in patients with peri-implantitis (p=0.001), (II=3.28-4.67, PI=2.16-2.39). 209 210 The CAL at the time of implant evaluation was more significant than that at the periodontal 211 evaluation. There was no notable contrast in attachment loss values identified between 212 assessments of implants and periodontal conditions in patients diagnosed with either peri-implant mucositis or peri-implant health (p=0.869), (p=0.971), (II=1.92-2.26, PI= 1.66-2.30), (II=1.79-213 214 2.17, PI=1.80-2.23) (Table 4). As per the assessment of implants, a notable distinction in gingival recession was observed 215 216 among the peri-implant groups (p=0.016). The peri-implantitis group exhibited the highest level of recession, which differed significantly from the other two groups, whereas the lowest gingival 217 recession value was observed in the peri-implant health group. 218



219	Implant evaluation revealed a significant difference in gingival recession between the
220	periodontal groups (p=0.020). The periodontitis group had the highest gingival recession rate,
221	which differed significantly from the other two groups, whereas the periodontal health group had
222	the lowest.
223	Discussion
224	This study tested whether peri-implant and periodontal conditions occurred simultaneously.
225	It found that gingivitis was mainly detected in patients with peri-implant mucositis and peri-
226	implant health. The results show that most patients with peri-implantitis do not have concomitant
227	periodontitis.
228	Vague statement: Specify what kind of "relevance" is being considered. Investigations into peri-implant biofilms consider the relevance of implant-related
229	environmental factors. These factors play a crucial role in facilitating effective, implant-driven
230	therapies for peri-implantitis, which are essential for mitigating the health burden associated with
231	implant-related inflammatory conditions (Kotsakis & Olmedo, 2021). The architectural
232	characteristics of dental implants differ from those of natural teeth, including differences in
233	morphology, surface material, texture, and energy (Robitaille et al., 2016). Furthermore, dental
234	implants differ from natural teeth by being decay-resistant, lacking pulps that could serve as
235	early pathology indicators or contribute to endodontic lesions, and lacking a periodontal
236	membrane (Misch, 2014). Periodontal tissues attach teeth to alveolar bone via the periodontal
237	ligament and supra-bony connective tissues, which include collagen fibers anchored to the root's
238	cementum. In contrast, osseointegrated dental implants lack these connective tissue attachments,
239	with direct bone contact and no intervening connective tissues (Klokkevold & Newman, 2000).
240	When comparing implants to natural teeth, implants are typically conical screws made of
241	titanium and/or ceramic, known for their increased surface roughness and decreased surface
242	energy. Although roughness, energy, and composition are interrelated, each factor can
243	independently influence bacterial colonization, gene expression, and community composition
244	(Larsson et al., 2022). The presence of peri-implantitis in implants led to significantly elevated
245	amounts of dissolved titanium in subgingival plaque compared to healthy implants. This
246	indicates a strong association between titanium dissolution and peri-implantitis (Kotsakis,
247	Olmedo, 2021). Furthermore, the combination of stress, corrosion, and bacteria can also doesn't flow with from the contribute to implant failure (Chatumadi, 2000). Tribocorrosion and metal corrosion impact pari
248	contribute to implant failure (Chaturvedi, 2009). Tribocorrosion and metal corrosion impact peri-
249	implant biofilms, potentially leading to peri-implant inflammation and implant failure through



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250	direct mechanisms (such as immune modulation) or indirect pathways (by disturbing the
251	microbiome) (Kotsakis & Olmedo, 2021). Additional investigation is required to elucidate the
252	factors behind titanium dissolution and the role of titanium corrosion byproducts in the
253	progression of peri-implant inflammation (Safioti, 2017). According to the limited clinical data,
254	the incidence and development of peri-implantitis do not differ between modified and non-
255	modified implant surfaces (Schwarz et al., 2021). Differences in the implant and natural tooth
256	environment affect the simultaneous occurrence of periodontal and peri-implant diseases. In this
257	study, these differences led to the occurrence of not only periodontitis but also gingivitis with
258	peri-implantitis.
259	Meffert et al. (Meffert, 1996) reported that the bacterial flora linked to the implant and native
260	tooth during illness are mostly identical and consist primarily of gram-negative pathogens,
261	including P. gingivalis, Porphyromonas intermedia, and A. actinomycetemcomitans. It indicates
262	that the subgingival microbiota compositions are quite comparable between the distinct clinical
263	groups of periodontitis and peri-implantitis. These similarities encompass potential
264	"periodontopathogens" like Prevotella, Porphyromonas, Tannerella, Bacteroidetes [G5], and
265	Treponema spp. (Yu et al., 2019). In contrast, Dutra et al. (Dutra et al., 2023) observed a varied
266	array of bacteria near infected implants, some of which were unculturable and previously
267	unidentified. The presence of bacteria unrelated to periodontitis could instigate inflammation in
268	the peri-implant tissues, highlighting notable distinctions in the microbiota between periodontal
269	and peri-implant regions. Also, a higher prevalence of opportunistic pathogens, such as
270	Staphylococcus and Candida species, characterizes the microbiome associated with peri-
271	implantitis (Iușan et al., 2022). The structure of biofilms in peri-implantitis is more intricate
272	compared to that in periodontitis. Although various bacterial species have been identified as
273	potential pathogens in peri-implantitis, periodontopathogenic bacteria are less prevalent
274	(Koyanagi et al., 2013). In periodontitis, bacteria from the red complex are vital pathogens,
275	whereas they are not prevalent in peri-implant biofilms. There might be a confirmation bias in
276	the dissemination of information regarding their presence (Kotsakis & Olmedo, 2021). Another
277	study revealed no discernible difference in the occurrence of periodontal bacteria around implant
278	sites in patients with peri-implant mucositis compared with patients with gingivitis (Salvi et al.,
279	2022). Host-bacterial interactions shape unique microbiomes in both periodontal and peri-
280	implant environments, indicating differences in microbial composition associated with health



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and disease, both individually and at the core microbiome level. However, diseases can facilitate the migration of periodontal bacteria into peri-implant sulci, or periodontitis can progress to peri-implantitis (*Robitaille et al., 2016*). In this study, individuals diagnosed with peri-implant mucositis had concomitant gingivitis, while gingivitis was detected in most patients presenting with peri-implantitis due to these differences.

Various alterations in microbial populations influence the initiation and advancement of inflammatory reactions surrounding both natural teeth and dental implants. Furthermore, prior occurrences of periodontal disease exert an additional influence on modifying the immune reactions of peri-implant and periodontal tissues in response to the accumulation of biofilms (Dutra et al. 2023). The majority of the cells in both entities tend to be plasma cells and lymphocytes. However, neutrophil granulocytes and macrophages have been reported to be more abundant in patients with peri-implantitis than those with periodontitis (Berglundh et al., 2011). Implant plaque control effectively inhibits the formation of bacterial plaques on titanium abutments. The absence of inflammatory cell infiltrates in the peri-implant mucosa further highlights the ability of the junctional epithelium at titanium surfaces to form a barrier, preventing the formation of a subgingival infection in the absence of supragingival plaque (Berglundh et al, 1991). The expression of mRNA levels for IL-6 and IL-1β is observed to elevate in tissues afflicted by both periodontal disease and peri-implantitis. However, no significant difference was detected in the expression of metalloproteinases and their inhibitors among the studied groups (Figueiredo et al., 2020). Conversely, soft tissues around implants likely trigger more enhanced host immune responses, such as dominant macrophage infiltration, to promote osteoclastogenesis than those in periodontitis in another study (Yuan et al., 2022). In addition, IL-1 and TNF-α serve as sensitive indicators of bone loss adjacent to both natural teeth and dental implants (Machtei, Oved-Peleg & Peled, 2006). Salvi et al. (Salvi et al., 2022) similarly reported that IL-1 β levels were the same in their study, while MMP-8 levels were greater around the peri-implant region. Although peri-implantitis and periodontitis share similarities in clinical presentation and etiology, significant histopathological distinctions differentiate these two conditions (Berglundh et al., 2011). These significant histopathologic differences may affect the incidence of peri-implantitis and periodontitis at the same time. This study supports this situation.



Genetic variations in the Fmlp Receptor (FPR1) gene are strongly linked to a higher
susceptibility to periodontitis and peri-implantitis (Turkmen & Firatli, 2022). The genetic
variation within the IL-17A gene may potentially influence the predisposition to peri-implant
diseases (Talib & Taha, 2024). Also, the alleles 1 and 2 of the IL1A gene and the alleles 1 and 2
of the IL1B gene were statistically associated with the success or no success of the dental
implants (Vaz et al., 2012). Ten genetic polymorphisms of inflammation-related molecules,
including pro-inflammatory cytokines and protease inhibitors, may have substantially influenced
periodontitis. An individual may inherit several relatively common high-risk polymorphisms,
resulting in a cumulative high-susceptibility profile for periodontitis (Kinane, Hart, 2003). To
date, specific genetic variations consistently associated with periodontitis in certain populations
encompass those within ANRIL, COX2, IL1, IL10, and DEFB1 genes. However, many proposed
candidate genes for periodontitis lack robust validation or replication (Loos et al., 2015).
According to a study, individuals carrying the G genotype exhibit an increased susceptibility to
periodontitis, while those with the G/C genotype demonstrate a greater risk of peri-implantitis
(Turkmen, Firatli, 2022). With the effect of the differentiation of the genetic profile in peri-
implantitis and periodontitis, periodontitis may not be seen in every patient with peri-implantitis,
as seen in the study.

Substantial evidence suggests an elevated risk of peri-implantitis among individuals with a previous history of chronic periodontitis, inadequate plaque control proficiency, or lack of consistent post-implant therapy maintenance (Schwarz et al., 2018). Additionally, robust evidence indicates that periodontitis amplifies the probability of implant loss. Moreover, there exists moderate evidence suggesting that individuals affected by periodontitis exhibit elevated rates of implant-bone loss, thus establishing this condition as a predisposing factor for peri-implantitis (Shiba T et al., 2021). Although the presence of periodontitis is a serious risk factor for peri-implantitis, this is not always the case, as found in this study.

Furthermore, it is imperative to consider immunological and histopathological distinctions when devising treatment strategies for peri-implantitis and periodontitis (*Berglundh et al., 2011*). Following non-surgical interventions, the microbial makeup of periodontal and peri-implant sites is observed to undergo comparable alterations, transitioning from an abundance of periodontal pathogens to a composition akin to healthy sites (*Shiba T et al., 2021*). Notably, in implants



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341	featuring rough surfaces, a previous history of periodontal disease detrimentally affects survival
342	rates, despite undergoing scaling and root planing procedures (Young et al., 2021). There were
343	also reports of disease progression or recurrence, as well as implant loss despite treatment (Heitz-
344	Mayfield, Mombelli, 2014). Microbial, genetic, and immunologic differences in peri-implantitis
345	and periodontitis are reflected in treating these diseases. The study's results support these
346	differences, and a personalized approach is considered more appropriate in treating peri-
347	implantitis.
348	This study has several limitations and strengths worth noting. A key limitation is the
349	potential selection bias, as the sample was drawn from a single university clinic and may not
350	represent the broader population. Although random sampling and strict inclusion and exclusion
351	criteria were employed, some bias might still exist. Additionally, the cross-sectional design
352	limits the ability to establish causal relationships between peri-implant and periodontal
353	conditions. Despite these limitations, the study has notable strengths, including the use of well-
354	established diagnostic criteria from the 2017 AAP/EFP World Workshop on Classification of
355	Periodontal and Peri-implant Diseases and Conditions, ensuring consistent and reliable
356	assessments. The comprehensive data collection by a single clinician, which included various
357	indices such as plaque index, gingival index, probing depth, bleeding on probing, clinical
358	attachment loss (CAL), and gingival recession, provides a thorough evaluation of the conditions.
359	Importantly, the study contributes valuable insights into the co-occurrence of peri-implant and
360	periodontal conditions, particularly highlighting that peri-implantitis is often absent in patients
361	with periodontitis, suggesting a complex relationship between implant treatment and periodontal
362	health that warrants further investigation.
363	Conclusions
364	This study tested whether peri-implant and periodontal conditions occur simultaneously and
365	found that most patients with peri-implantitis did not have concomitant periodontitis. Clinically,
366	these results indicate that peri-implant and periodontal conditions should be evaluated and
367	treated independently, emphasizing preventive care, regular monitoring, and patient education on
368	rigorous oral hygiene practices, while future research should explore the underlying mechanisms

differentiating peri-implantitis from periodontitis to develop targeted therapies and improved

management strategies for patients with dental implants.



- 371 Decleratation
- 372 Ethics approval and consent to participate
- 373 Bolu Abant İzzet Baysal University Clinical Researches Ethics Committee approved the study
- 374 (2022/163-28/06/2022). Participants were informed verbally and in writing about the design of
- 375 the study. The study was conducted with respect to the Helsinki Declaration. The informed
- 376 consent form was obtained from all participants.
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Table 1(on next page)

Demographic characteristics of participants



		f	%
Gender	Male	70	57.1
	Female	53	42.9
	Elementary School	27	21.4
Education	Middle School	ddle School 14	
	High School	33	25.7
	University	49	40.0
	Working	85	68.5
Work status	Nonworker	9	7.1
	Retired	29	22.9
Age	=	Mean±S.D	Min-Max
nge		51.37±10.64	26-72

 Table 1: Demographic characteristics of participants



Table 2(on next page)

Demographic characteristics of participants



Table 2: Distribution of examination groups

		f	%
Peri-implant	Peri-implantitis	41	33.3
group	Peri-implant mucositis	41	33.3
	Peri-implant health	41	33.3
Periodontal group	Gingivitis	55	44.7
	Periodontitis	18	14.6
	Periodontal health	50	40.7
	Total	123	100.0



Table 3(on next page)

Comparison of peri-implant and periodontal groups



1 *Table 3:* Comparison of peri-implant and periodontal groups

		Peri-	_			
		Peri- implantitis	Peri- implant mucositis	Peri- implant health	Total	р
	Gingivitis	17 41.5% ^a	21 51.2% ^a	17 41.5% ^a	55 44.7%	0.003*
Periodontal Group	Periodontitis	13 31.7% ^a	2 4.9% ^b	3 7.3% ^b	18 14.6%	-
-	Periodontal	11	18	21	50	_
	health	26.8% a	43.9% ^b	51.2% b	40.7%	_
Total		41	41	41	123	
		100.0%	100.0%	100.0%	100.0%	



Table 4(on next page)

Comparison of index values in implant and periodontal evaluation



1 Table 4: Comparison of index values in implant and periodontal evaluation

		İmplant index		Periodontal index		
	_	Mean±S.D.	95% CI (L-U)	Mean±S.D.	95% CI (L-U)	p
	Peri-implantitis	0.13 ± 0.31	0.03-0.23	0.36 ± 0.48	0.21-0.52	0.001*
Plaque	Peri-implant mucositis	0.09 ± 0.24	0.01-0.16	0.21 ± 0.22	0.15-0.28	0.002*
index	Peri-implant health	0.06 ± 0.14	0.02-0.10	0.16 ± 0.19	0.10-0.22	0.025*
	Total	0.09 ± 0.24	0.05-0.13	0.24 ± 0.33	0.19-0.30	0.001*
	Peri-implantitis	0.42 ± 1.09	0.07-0.76	$0.11\pm0,11$	0.07-0.14	0.001*
Gingival	Peri-implant mucositis	0.26 ± 0.50	0.10-0.42	$0.14\pm0,17$	0.08-0.19	0.023*
index	Peri-implant health	0.16 ± 0.42	0.02-0.29	0.09 ± 0.16	0.04-0.15	0.412
	Total	0.28 ± 0.74	0.15-0.41	0.11 ± 0.15	0.09-0.14	0.001*
•	Peri-implantitis	3.91±2.14	3.24-4.59	2.17±0.26	2.09-2,25	0.001*
Probing on	Peri-implant mucositis	2.14 ± 0.44	2.00-2.28	1.98 ± 0.88	1.70-2.26	0.165
depth	Peri-implant health	1.97 ± 0.48	1.82-2.12	2.06 ± 0.25	1.98-2.14	0.837
	Total	2.67 ± 1.55	2.40-2,95	2.07 ± 0.55	1.97-2.17	0.006*
	Peri-implantitis	0.66 ± 0.37	0.54-0.78	0.23 ± 0.25	0.15-0.31	0.001*
Bleeding	Peri-implant mucositis	0.62 ± 0.29	0.53-0.71	0.18 ± 0.15	0.13-0.23	0.001*
on probing	Peri-implant health	0.00 ± 0.02	0.00-0.01	0.14 ± 0.18	0.09-0.20	0.001*
	Total	0.43 ± 0.40	0.36-0.50	0.18 ± 0.20	0.15-0.22	0.001*
	Peri-implantitis	$0.72\pm1,41$	0.27-1.17	0.19 ± 0.38	0.06-0.31	0.001*
Gingival	Peri-implant mucositis	0.25 ± 0.66	0.04-0.46	0.12 ± 0.20	0.05-0.18	0.014*
recession	Peri-implant health	0.16 ± 0.42	0.02-0.29	0.23 ± 0.47	0.08-0.38	0.410
	Total	0.38 ± 0.96	0.20-0.55	0.18 ± 0.37	0.11-0.24	0.001*
	Peri-implantitis	3.97±2 . 21	3.28-4.67	2.28±0.36	2.16-2.39	0.001*
Clinical	Peri-implant mucositis	2.09 ± 0.55	1.92-2.26	1.98 ± 1.02	1.66-2.30	0.869
attachment level	Peri-implant health	1.98±0 . 61	1.79-2.17	2.02 ± 0.68	1.80-2.23	0.971
	Total	2.68 ± 1.63	2.39-2.97	2.09±0.74	1.96-2.22	0.001*

2 95% Confidence Interval (Lower Bound-Upper Bound)

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