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Precise Fit and Torque Minimize Implant-Abutment Interface Microleakage in Two-Stage Internal Hexagon Dental Implants

Avshalom Tam, M.Sc.
Vladislav Dvoyris, D.M.D
Soron Steinberg, D.M.D., Ph.D

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Avshalom Tam, M.Sc. [i],
Vladislav Dvoyris [ii],
Doron Steinberg, D.M.D Ph.D [iii],

[i,iii] Biofilm Research Laboratory, Faculty of Dental Medicine, Hebrew University, Jerusalem.

[ii] Ditron Dental, a division of Ditron Precision LTD / Ashkelon, Israel

הפקולטה לרפואת שיניים
The Faculty of Dental Medicine
בית הספר לרפואת שיניים של אוניברסיטה העברית והדסה מיסודה של אחוות אלפא אומגה
The Hebrew University-Hadassah School of Dental Medicine Founded by the Alpha Omega Fraternity

ABSTRACT

Bacterial leakage and presence of bacterial colonies around and inside the implant-abutment connection is an important factor in the development of peri-implant mucositis and peri- implantitis, severely affecting the long-term survival of implants. The purpose of our study was to test in-vitro whether the internal hexagon implant-abutment connection of the Ditron Dental MPI™ system can provide an effective biological seal against oral microorganisms, preventing them from flowing in or out the implant inner cavity. 20 MPI implants and 20 abutments were divided into 2 groups for a 2-phase experiment with Streptococcus mutans bacteria, testing the ability of the I-A seal to shield the implant from outside bacteria and preventing bacteria present in the implant well from leaking out. The implants and abutments were then separated and scanned with an electron microscope. No outside bacteria were detected in any of the implant wells. No inside bacteria were detected in the nutrient broth. The implant abutment connection is prone to micro movement and micro- gap which could lead to microbial leakage. Inflammation and bone loss are becoming an ever increasing concern in modern day implanttology and thus it is imperative to minimize bacterial presence in and around the implant-abutment junction. The MolecuLock™ internal hexagon connection provides an effective seal against oral microorganisms with regard to a simulated in- vitro bacterial invasion.

INTRODUCTION

Bacterial leakage and presence of bacterial colonies around and inside the implant-abutment connection is an important factor in the development of peri-implant mucositis and peri-implantitis[1], severely affecting the long-term survival of implants. High levels of leakage and micromovements in the implant-abutment interface were observed in the first osseointegrated implants, leading to increased bone loss in the first year of function.[2] Implant manufacturers have since sought to decrease the amount of bacterial microleakage, introducing novel implant systems and diminishing the implant-abutment microgap, thus maximizing peri-implant bone stability[3,4,5,6].

The prevention of bacterial infiltration within the implant- abutment interface is nowadays one of the biggest challenges for modern implant systems manufacturers. The implant-abutment interfaces, located subgingivally, usually include microscopic gaps of up to $49_{\mu m}$, arising from a less-than-perfect implant- abutment fit. These gaps are ideal potential sites for retention of pathogenic bacteria *(ranging in size from 1 to* $10_{\mu m}$*)*. Indeed, anaerobic periopathogenic bacteria such as Fusobacterium nucleatum, Prevotella intermedia, Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans and spirochetes can occupy deep peri-implant pockets 3-6 months after implant placement. [7,8,9,10]

Numerous manufacturers have so far attempted to perfect the implant-abutment interface design by offering different types of implants and abutments and various protocols of surface treatment and coating. According to the manufacturers' recommendation, the abutment screws should be tightened with torque wrenches achieving preload forces of 10-35$_{N/cm}$, however some dentists use handheld screwdrivers, achieving maximum torque values of 12.9$_{N/cm}$.[11]

Fluid passage and bacterial accumulation were shown around the implant-abutment connection regardless of the connection type, in numerous studies. Previous studies have demonstrated that the seal between implants and abutments cannot be maintained even with controlled torque.[12]

The Moleculock™ concept of the Ditron Dental MPI™ implant system aims at ensuring a perfect implant-abutment fit, reducing risks of micromovement and minimizing microgaps to 1$_{\mu m}$. The purpose of our study was to test in-vitro whether the internal hexagon implant-abutment connection of the Ditron Dental MPI™ system can provide an effective biological seal against oral microorganisms, preventing them from flowing in or out the implant inner cavity.

Materials & Methods

Materials

- 20 MPI™ 5.0mm x 13.0mm Gamma-ray sterilized dental implants.
- 20 abutments *(Ditron Dental / Ashqelon, Israel)* were used in the trial and divided into 2 groups for a two-phase experiment.

Methods

- Phase I was intended to test the ability of the seal to shield the implant well from external bacterial leakage.
- Phase II tested the ability of the seal to prevent bacteria present in the implant well from leaking out.

PHASE I: OUTSIDE-IN BACTERIAL LEAKAGE

10 MPI™ implants *(5X13$_{mm}$, LOT 843/624)* were connected to abutments *(ABT-6040, LOT 928/730)*, using a torque of 25$_{N/cm}$. The abutment screw opening was sealed with silicone *(SILICONE RUBBER, RTV 116Q, 12NWFA012/MOMENTIVE Performance Materials)*. The connected implants and abutments were then autoclaved.

A BHI medium was inoculated with fresh Streptococcus mutans *(ATCC 27351)* bacteria, taken from frozen stock *(-80°C)* and incubated in 37°C in 5% CO2 for 18 hours. The uniformity of bacterial species was then tested and confirmed.

The implant-abutment systems were transferred to four tubes, each containing 12.5_{ml} BHI and 1_{ml} of bacteria, and incubated at 37°C in 5% CO2 for 96 hours with rotary shaking. After 96 hours, the bacteria that grew in the suspension were checked with phase microscope.

The implant-abutment systems were washed in sterilized distilled water. The connection was opened and the parts were transferred to fixative in preparation for scanning electronic microscope *(SEM)* analysis. SEM images of five areas on the abutment and 7 areas on the implant were captured and analyzed for presence of bacteria.

PHASE II: INSIDE-OUT BACTERIAL LEAKAGE

10 MPI™ implants *(5X13mm, LOT 843/624)* and abutments *(ABT-6040, LOT 928/730)* were autoclaved. A fresh BHI medium was inoculated with fresh Streptococcus mutans *(ATCC 27351)* bacteria taken from frozen stock *(-80°C)* and incubated in 37°C in 5% CO2 for 18 hours. The uniformity of bacterial species was then tested and confirmed.

$2_{\mu l}$ of bacteria from the above inoculum were then inserted inside the implant.

The implant and abutment were connected and screw-tightened, using a closing torque of $25_{N/cm}$. The abutment screw opening was then sealed with silicone *(SILICONE RUBBER, RTV 116Q, 12NWFA012/Momentive Performance Materials)*.

After 2 hours, the connected implant-abutment systems were transferred to fresh BHI medium and incubated at 37°C in 5% CO2 for 48 hours with rotary shaking. The implant was then disconnected from the abutment and taken to SEM. SEM images of six areas on the abutment and eight areas on the implant were taken and analyzed for presence of bacteria.

RESULTS

PHASE I: OUTSIDE-IN LEAKAGE TRIAL

SEM images of the abutments *(Fig. 1A-F)* and implants *(Fig. 2A- H)* have not shown any bacterial penetration on the inside part of the abutment or the implant.

PHASE I: OUTSIDE-IN – SEM IMAGES OF ABUTMENT

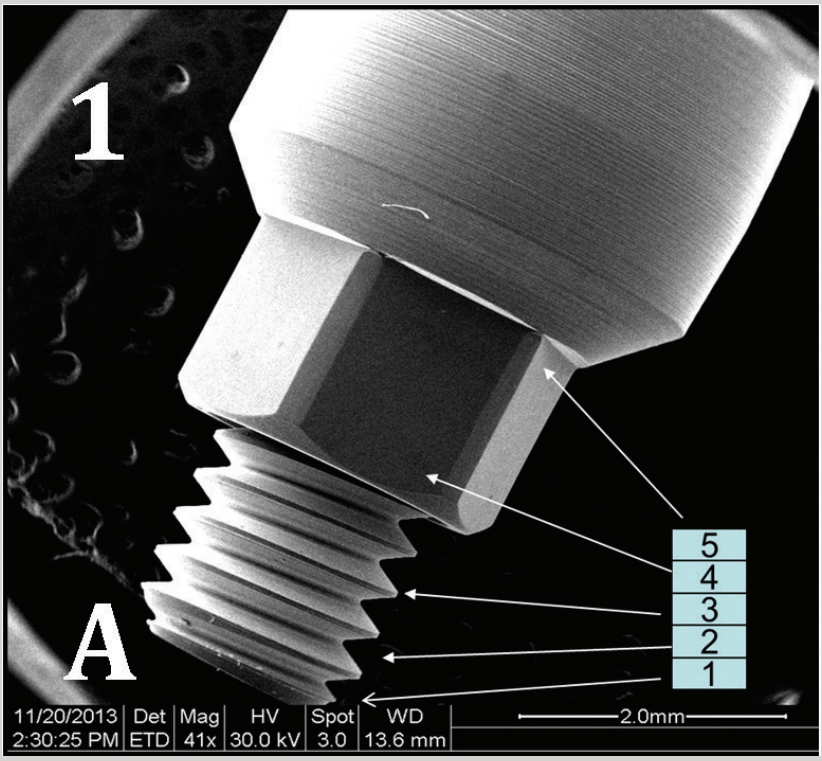

Fig. 1A Scanned Areas of the Abutment

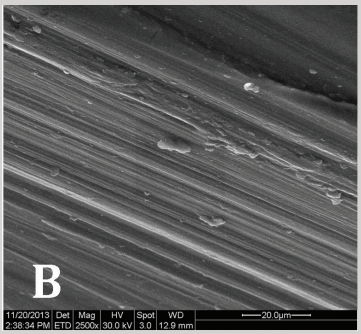

Fig. 1B Area #1 (x2500) –
No Bacteria Detected

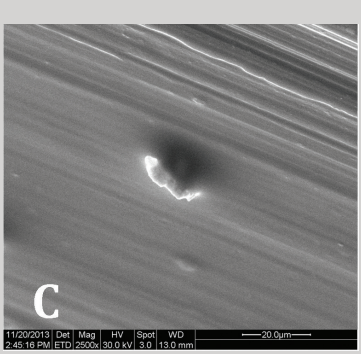

Fig. 1C Area #2 (x2500) –
No Bacteria Detected

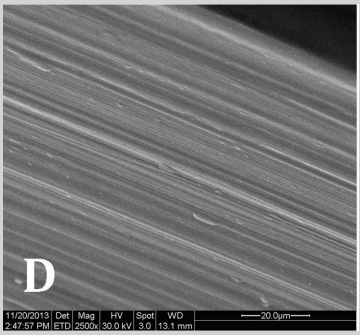

Fig. 1D Area #3 (x2500) –
No Bacteria Detected

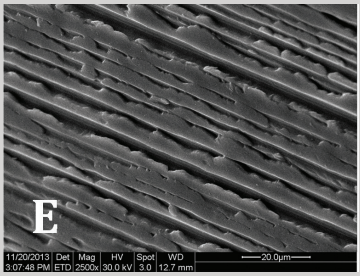

Fig. 1E Area #4 (x2500) –
No Bacteria Detected

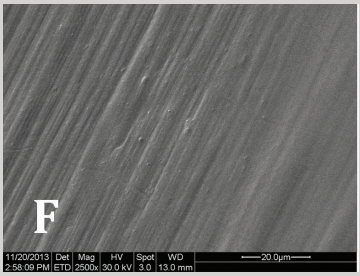

Fig. 1F Area #5 (x2500) –
No Bacteria Detected

PHASE I: OUTSIDE-IN – SEM IMAGES OF IMPLANT

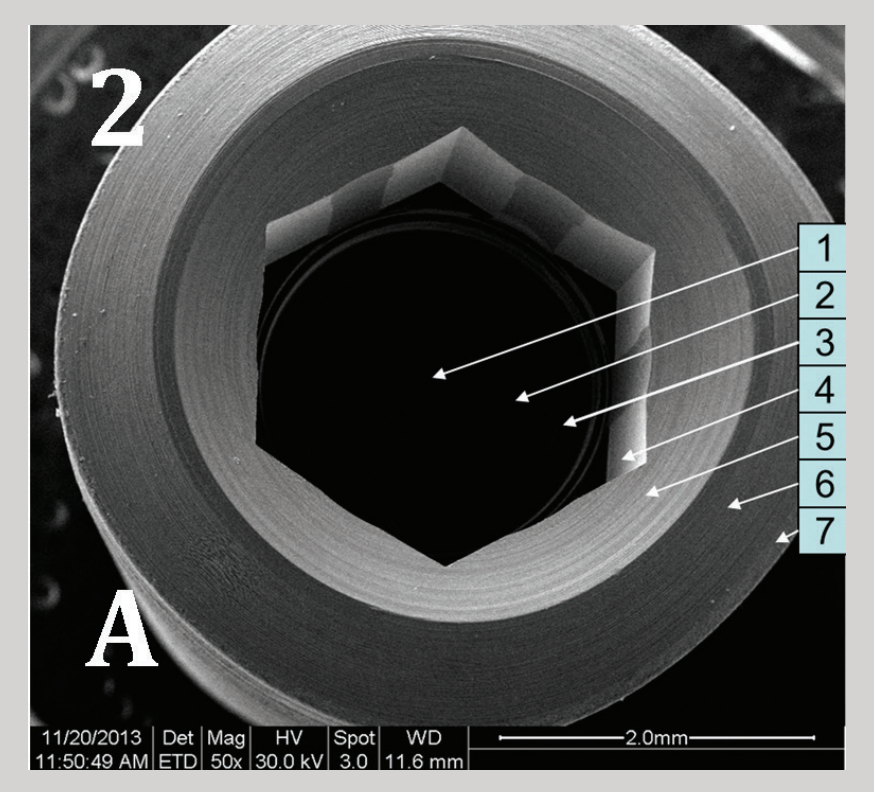

Fig. 2A Scanned areas of the Implant

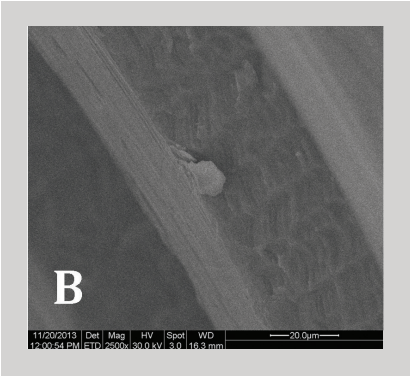

Fig. 2B Area #1 (x2500) –
No Bacteria Detected

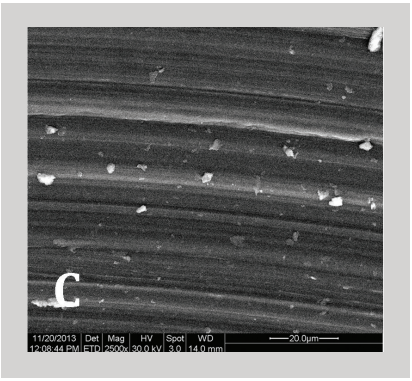

Fig. 2C Area #2 (x2500) –
No Bacteria Detected

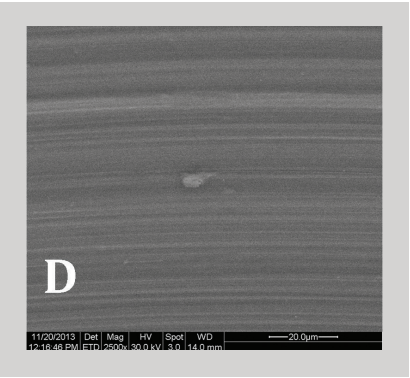

Fig. 2D Area #3 (x2500) –
No Bacteria Detected

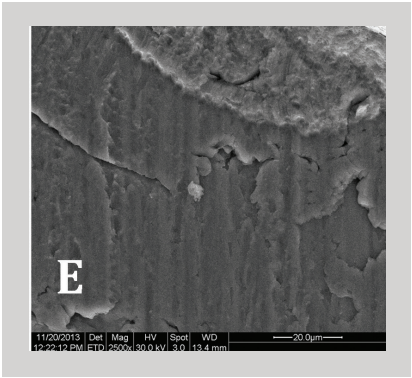

Fig. 2E Area #4 (x2500) –
No Bacteria Detected

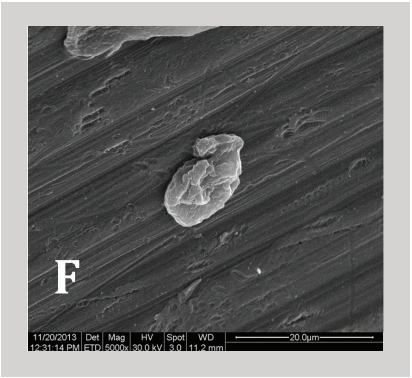

Fig. 2F Area #5 (x5000) –
No Bacteria Detected

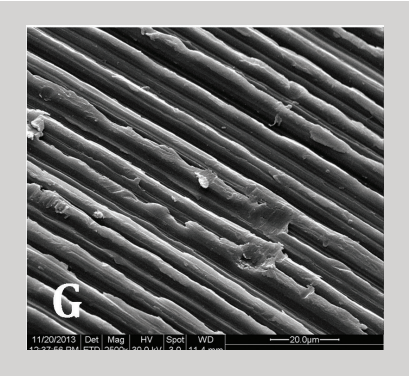

Fig. 2G Area #6 (x2500) –
No Bacteria Detected

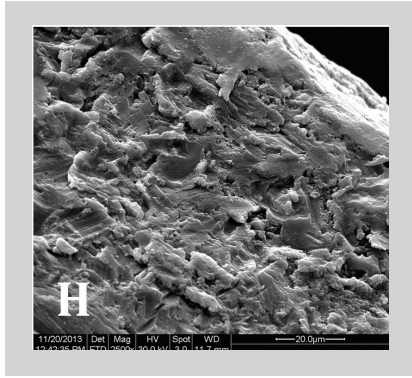

Fig. 2H Area #7 (x2500) –
No Bacteria Detected

PHASE II: INSIDE-OUT BACTERIAL LEAKAGE

SEM images of the abutments (*Fig. 3A-I)* have shown some bacterial penetration from the implant space *(Area #1)* up to the coronal part of the hexagon *(Area #4)*. No bacteria have, however, penetrated further *(Areas #5-6)*.

PHASE II: INSIDE-OUT – SEM IMAGES OF ABUTMENT

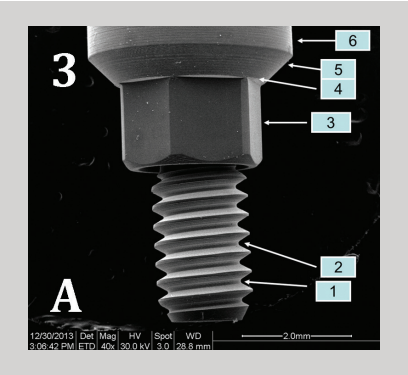

Fig. 3A Scanned Areas of the Abutment

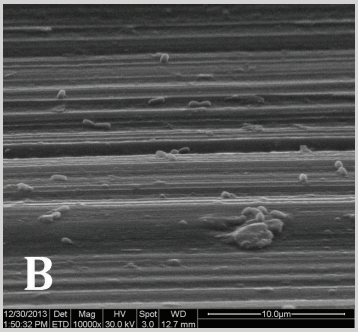

Fig. 3B Area #1 (x10000) – Bacterial Strains Detected

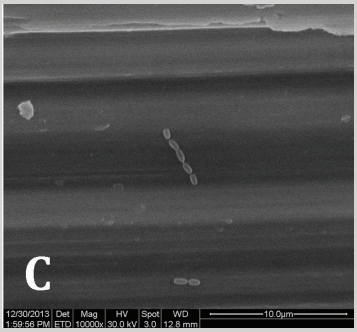

Fig. 3C Area #2 (x10000) – Bacterial Strains Detected

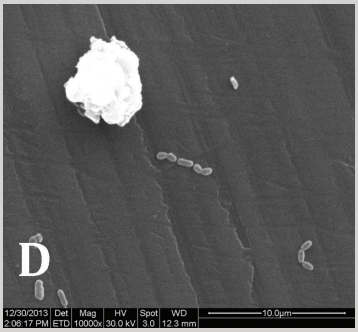

Fig. 3D Area #3 (x10000) – Bacterial Strains Detected

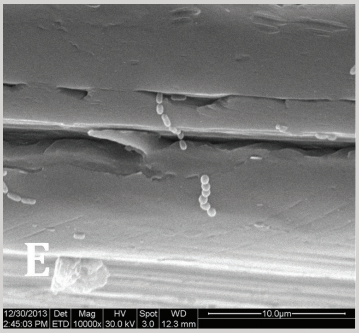

Fig. 3E Area #4 (x10000) – Bacterial Strains Detected

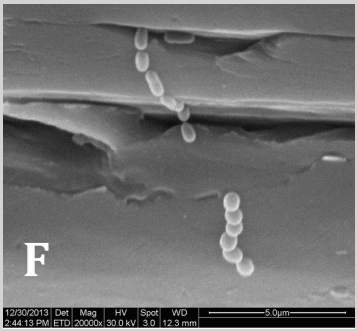

Fig. 3F Area #4 (x20000) – Magnification of Bacterial Strains

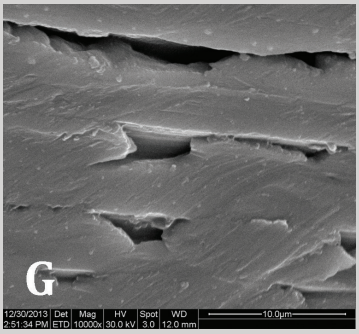

Fig. 3G Area #5 (x10000) – No Bacteria Detected

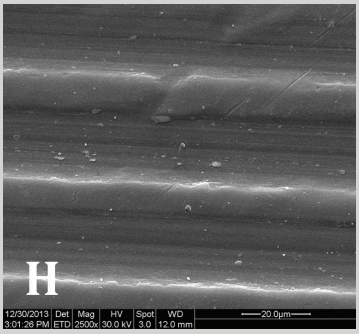

Fig. 3H Area #6 (x2500) – No Bacteria Detected

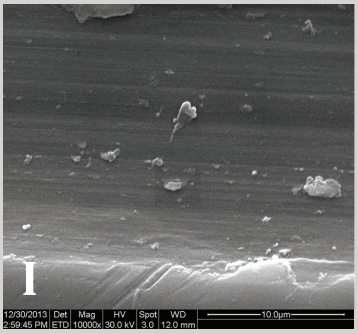

Fig. 3I Area #6 (x10000) – No Bacteria Detected

Similar scans of the implants *(Fig. 4A-H)* have shown bacterial penetration from the implant space *(Area #6)* to the coronal part of the hexagon *(Area #2a)*, without further penetration outside the implant-abutment connection. Some turbidity was observed in the suspension outside the implants, however it did not seem to be of bacterial origin.

PHASE II: INSIDE-OUT – SEM IMAGES OF IMPLANT

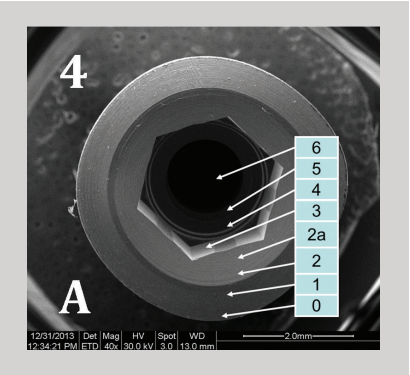

Fig. 4A Scanned Areas of the Implant

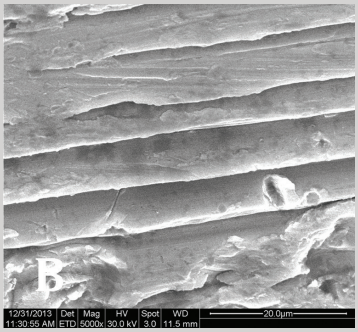

Fig. 4B Area#0(x5000) – No Bacteria Detected

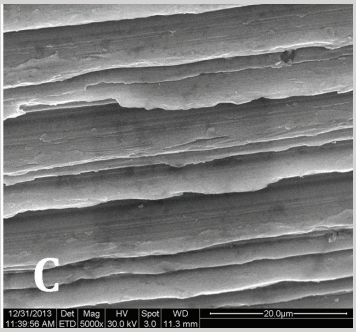

Fig. 4C Area #1 (x5000) – No Bacteria Detected

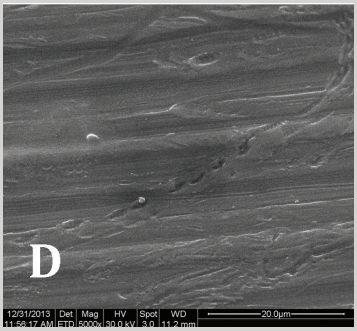

Fig. 4D Area #2 (x5000) – No Bacteria Detected

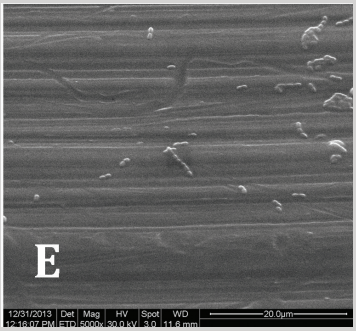

Fig. 4E Area #2a (x5000) – Bacterial Strains Detected

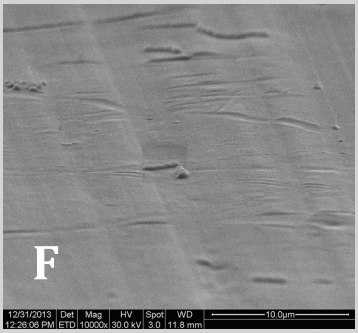

Fig. 4F Area #3 (x10000) – Bacterial Strains Detected

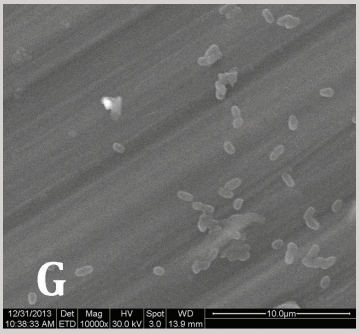

Fig. 4G Area #4 (x10000) – Bacterial Strains Detected

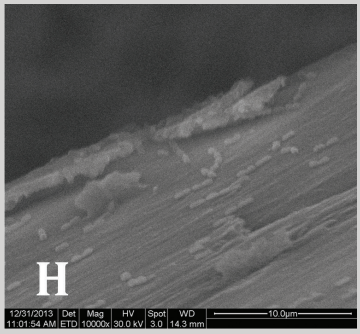

Fig. 4H Area #5 (x10000) – Bacterial Strains Detected

DISCUSSION

Microbial and biomechanical[1] factors are the 2 main reasons for implant failure. These failures can be further divided into early and late failures[2]. Early implant failure has been associated with inappropriate surgical technique such as surgical trauma and overheating, premature loading, compromised bone quality, and infection[3]. Late failures, which occur after implant restoration, have been associated with bacterial infection and biomechanical failure modes. Bacterial infection has been suggested as a leading factor of long term implant failure. The micro-gap caused by the misfit between the implant and the prosthetic component facilitates the infiltration of fluids and macromolecules from tissue fluids and saliva, harboring bacterial invasion and proliferation[3]. Bacterial permeability in the prosthetic abutment/implant connection, which has been studied by several researchers, allows the exchange of fluids and bacteria between the inner part of the implant and the oral environment[4].

In vitro studies have suggested that bacterial contamination through the prosthetic implant/abutment connections may be eventually correlated with gap sizes or misfits. The level of contamination varies or depends not only on the precision of fit, but also on the degree of the applied forces and torque. The incidence of loads and unscrewing of the prosthetic abutment can increase infiltration, whereas optimal adaptation, minimal micromovement and exceptional prosthetic and occlusal planning are factors that can prevent or minimize microleakage[5].

Everyday forces and functional loads are also prone to reduce implant-abutment stability and in turn instigate bacterial infiltration into internal spaces of the implant. Consequently, fluids can migrate between the implants and external environments and thereby increase the concentration of bacterial metabolites in the peri-implant region[6]. In this sense, it may be assumed that the role of the abutment/implant connection, with regard to the accurate fit between components and mechanical stability, is of considerable importance for long-term success.

While the occlusal factor may be controlled with careful prosthetic planning, the microbial factor is more elusive. The presence of a micro-gap in some submerged implant systems has prompted researchers to speculate that the initial bone loss typically observed in the first 12 month after implant restoration is the result of bacterial presence at the implant-abutment interface.[7] Recent studies have shown that because of the physical space created by the gap, fluids containing bacteria, bacterial byproducts and nutrients could pass through the interface gap into the implant well, contributing to malodor and peri-implantitis.[3,4,5,12,16,18] In implants where a micro-gap is present, microbial leakage and persistent bacteria at this peri-implant location could lead to inflammation. This sustained activation of inflammatory cells has been shown to

promote osteoclast formation and activation, which can result in alveolar bone loss[3]. Therefore, the importance of minimal bacterial presence is more and more apparent in or around the implant-abutment junction.

In a study where 3 different bacterial sizes were used: small *(A. actinomycetemcomitans)*, medium *(S. oralis)*, and medium-large *(F. nucleatum)*, it was evident that if a small microorganism such as A. actinomycetemcomitans could not penetrate the implant- abutment interface, then any more sizable microorganisms, such as E coli, which is 1.1-1.5 µm wide and 2.0-6.0 µm long, would not. A. actinomycetemcomitans, S. oralis, and F. nucleatum were chosen because these bacteria are a common finding in the oral cavity.[4]

In a recent systematic review, 21 studies of microleakage in various implant-abutment systems were analyzed.[8] Significant I-A gaps of 1-49$_{\mu m}$ were found in all studies, resulting in both inside- out and outside-in inoculation of most specimens, even with rather small, facultative bacteria. The reviewers have concluded that proper use of manufacturer recommended torque preload can minimize the gap, and hence the leakage. Another study has, in turn, shown that changing the screw torque value from 20 to 35$_{N/cm}$ did not significantly change the amount of leakage. Thus, previous research has demonstrated that bacteria will accumulate at the implant-abutment interface, regardless of the abutment screw torque value or the material used for the abutment.[9]

In this study, the bacterial seal provided by the MPI Moleculock™ concept was tested. Apparently, the metal to metal seal between the implant and the abutment was hermetic or too narrow for bacterial penetration since most bacteria are larger than 0.5$_{\mu m}$ in diameter. Hence, the gap in the tested implant-abutment systems is probably less than 0.5$_{\mu m}$.

However, in implants where inside-out leakage was tested, some degree of bacteria penetration was observed. The degree of leakage could be dependent on the closing torque – there was an inverse correlation between the degree of closing torque and leakage severity, and the higher the torque intensity was, the less leakage was observed[10].

Under these experimental conditions, and with the limitations related to a small sample size, there was little leakage between the inside of the implant and the outside environment in both phases of the study. The precise fit between the implant and abutment has reduced the microgap to a level which prevented both outside-in and inside-out microbial leakage of S. mutans between the implant well and the outside environment. This, in turn, may reduce the risk of pert-implant inflammation and infection.

Our preliminary findings suggest that the MolecuLock™ internal hexagon connection provides an effective seal against oral microorganisms in an in-vitro model simulating bacterial contamination with oral bacteria, S. mutans. Further clinical studies with larger samples, analyzing more bacterial species and especially common periopathogenic bacteria, should be performed to confirm conclusions drawn from the present investigation.

Moreover, it remains necessary to perform a cross- examination with other techniques *(color, microbial)* and on implant abutment connection that were subjected to cycle loads simulating day to day function of the implants and abutments. Implant type, position and abutment screw seal may also be factors affecting the extent of bacterial leakage, and should be further investigated. Additional verification on the nature of turbidity in the supernatant fluid is also required.

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