Race to the Surface

Modelling bacterial and human cell growth on dental implant surfaces

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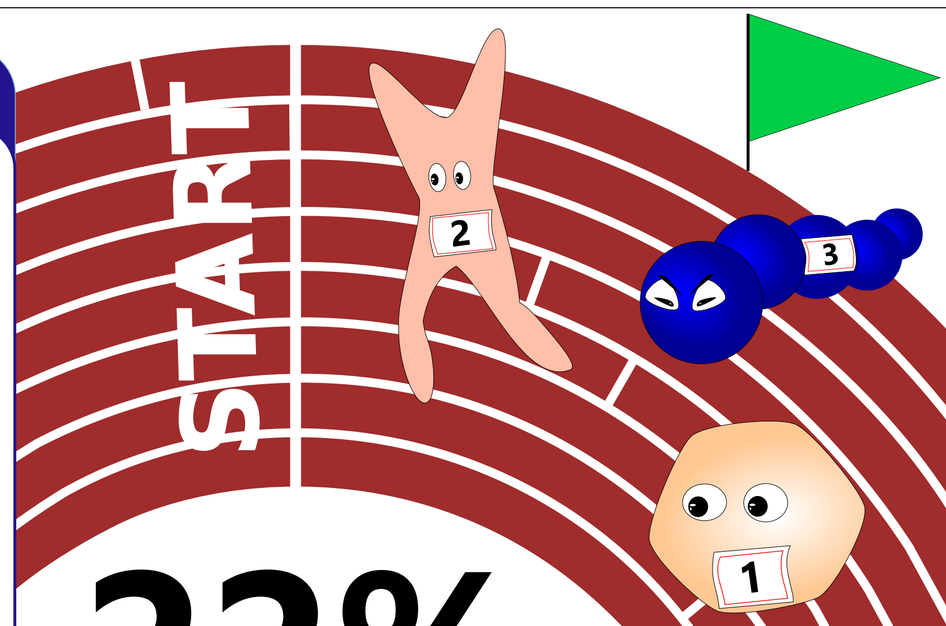
Introduction

Two common issues that can prevent successful dental implant surgery are:

1. the possibility of infection
2. poor compatibility with human cells

Human cells in the mouth are in **competition** with bacteria to occupy an implant surface. If bacteria successfully colonise on the surface, it can result in costly and painful **revision surgery**.

New materials for implants are being developed but are not making it into clinics. To tackle this issue, we are developing and combining models of **oral tissue** with **bacteria** to better understand how cells compete for space on the implant surface.



"...and here we have the starting line up. The human keratinocyte, number 1, is found on the gum tissue surface. If he arrives in first place, he will be able to prevent any bacteria from continuing in the race..."

"...His teammate, the human fibroblast sporting number 2, is found deeper in the gum tissue and is essential for integrating the implant into the jaw..."

22%

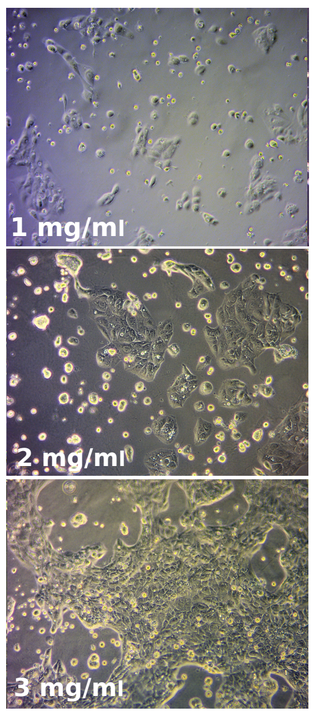
of patients will develop a serious infection within 10 years of dental implant placement[1]

"...Finally, going up against the tissue cells is Streptococcus sanguinis, a bacteria that likes to colonise the implant surface early on, before encouraging more bacteria to join in biofilm formation.

Lets see how this race pans out..."

Methods & Results

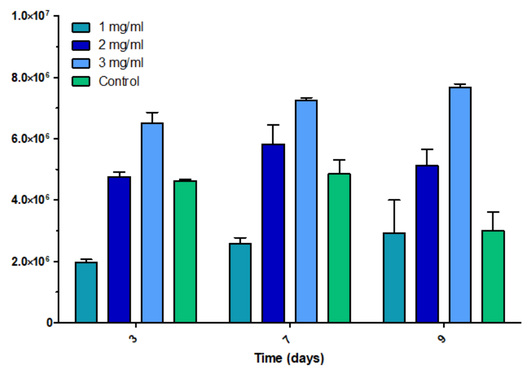
Developing a tissue model



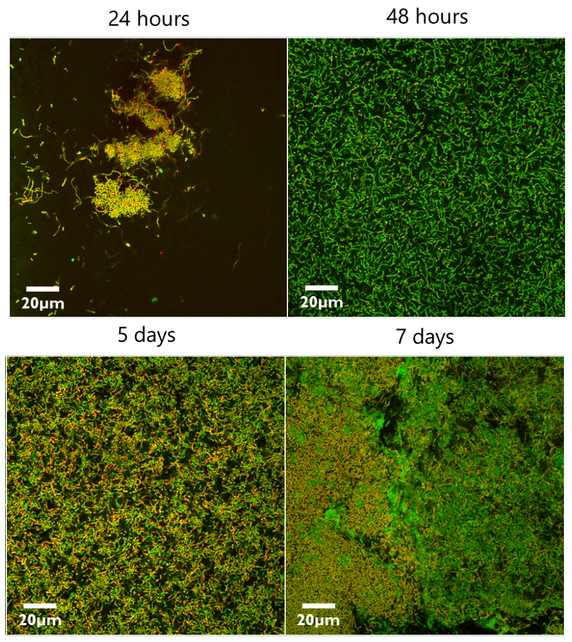
Currently, there are no gum tissue models that contain multiple cell types and mimic the mechanical properties of human tissue. We used **collagen**, an abundant protein found in oral tissue, to create a 3D gel resembling gum tissue. **Keratinocytes** were seeded upon various concentrations of collagen gels to determine which gel best supported cell growth.

Left - Human keratinocytes 24 hours after seeding on the surface of collagen gels at varying concentrations.

Right - Number of keratinocytes on the surface of collagen gels over 9 days. Higher concentrations of collagen induced higher cell numbers. Controls were grown on an empty well plate.



Characterising bacterial biofilms



Above - Confocal micrographs of *S. sanguinis* biofilms. Green and red cells represent live and dead bacteria respectively.

The mouth is colonised by millions of bacteria, living in communities called **biofilms**.

We grew biofilms of *S. sanguinis*, a bacteria that often initialises biofilm formation, on glass slides and imaged them using confocal microscopy (left). We were able to quantify this growth by counting the number of live and dead bacteria using computational image analysis software.

This has enabled us to gain an understanding of how biofilms form in the absence of tissue cells.

Factors affecting implant design and development:

Our **ageing population** requires implants to last much longer.

Instances of **peri-implantitis**, a chronic infection, are high and require costly restorative surgeries.

Antimicrobial resistance has significant implications for the success of dental implants.

New implant materials need testing in physiological environments. Currently, the best methods are **animal models**, which are costly and bring ethical challenges.

Conclusions

1. There are many threats to dental implant success. It is essential that we advance our understanding of how cells interact with implant surfaces.
2. Improved methods are needed to enhance the design of new implant materials and understand why implants can fail.

We have optimised current models of oral tissue and bacterial biofilms. The next step is to combine these models into one system to assess how these cells interact and observe the '**race to the surface**'.

FINISH

References

1) AlGhamdi, J. (2017) "Prevalence of peri-implant diseases among patients received dental implants at Riyadh city, KSA". International Journal of Applied Research, 3(7): 792-797.

Acknowledgements

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