



# Molecular Cartography of 'Healthy' and 'Diseased' Gingival Tissues

## BACKGROUND INFO

- Periodontitis: the most common non-communicable disease in humans
- Caused by a destructive interaction between dysbiotic biofilm and dysregulated host immune-inflammatory response, leads to tooth loss and associated with pre-mature mortality
- Pathophysiological mechanisms only incompletely understood

We **aim** to identify the spatial transcriptomic signatures that

- (i) define the transition from 'healthy' to 'diseased' status,
- (ii) help explain the potential differences of grade B ('moderate' progression) to grade C ('fast progression') periodontitis, and
- (iii) can be linked to specific bacterial-host interactions

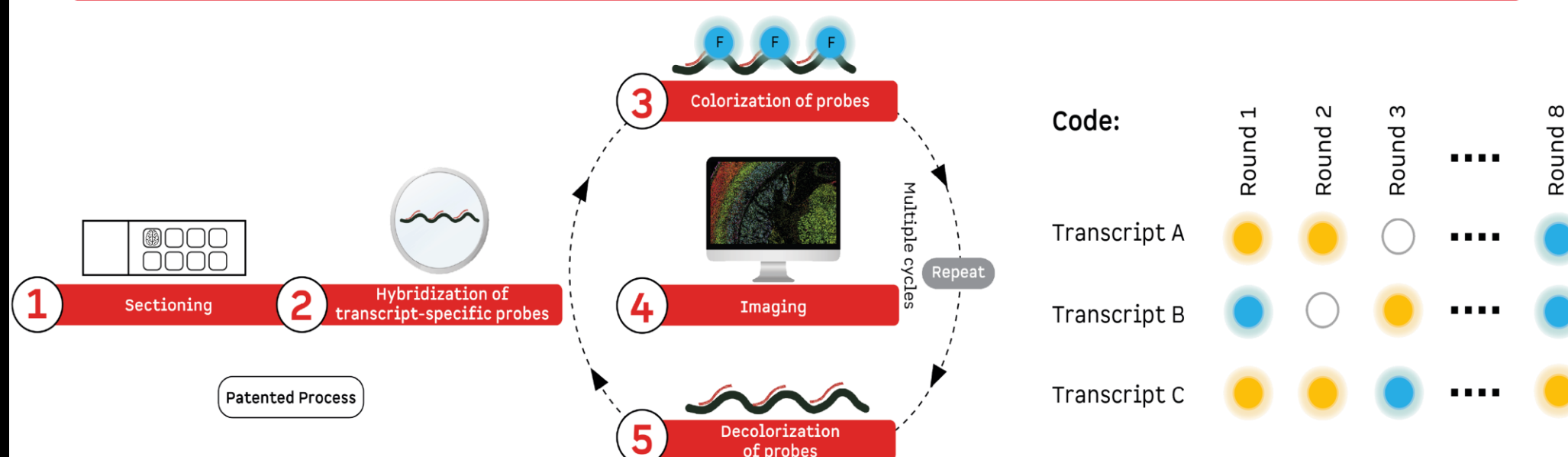


**Figure 1:** Sample Harvesting (right) and Intra-operative Situation (left)

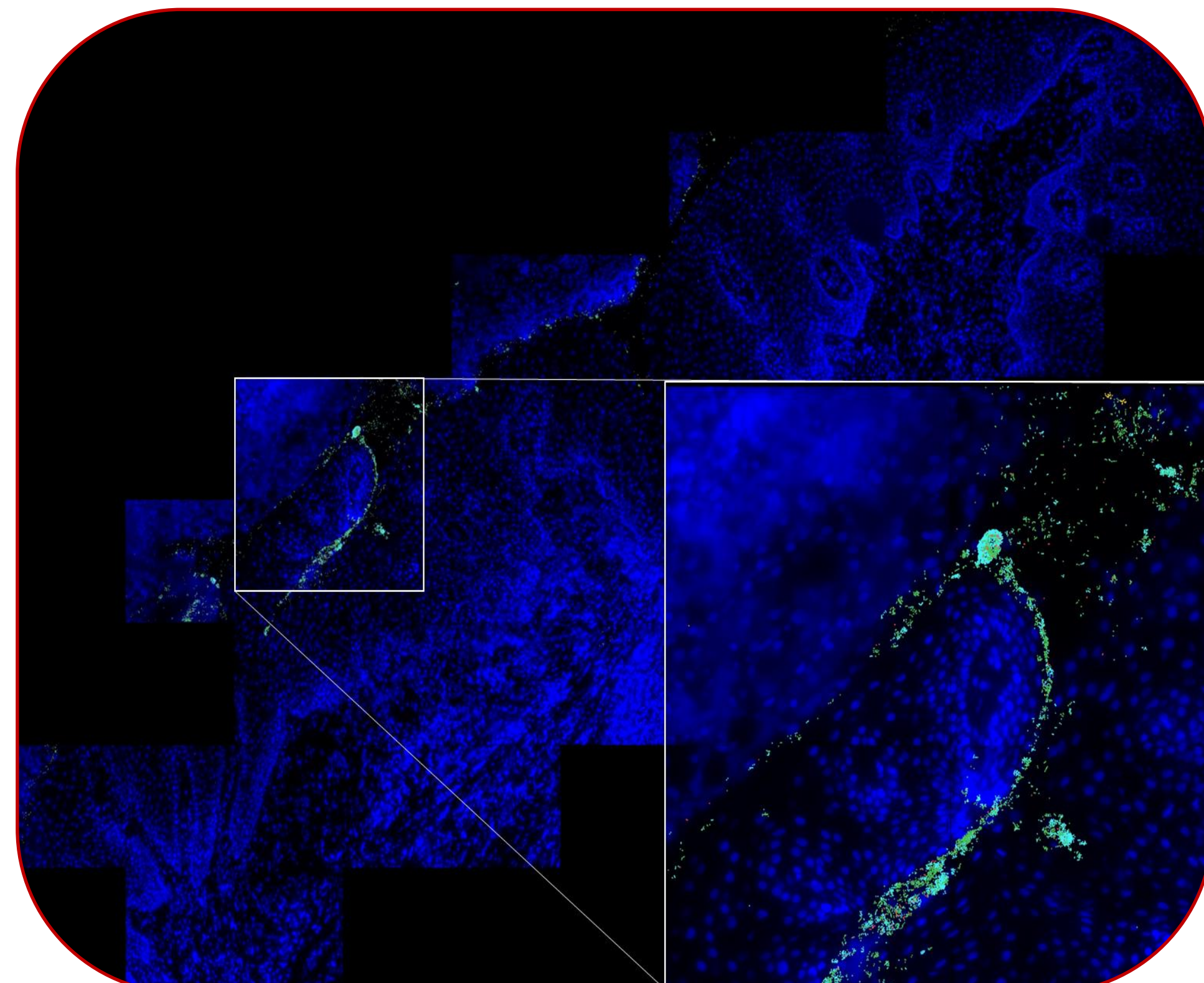


## METHODS

- Assessment of gingival tissue samples using Resolve Molecular Cartography technology (**Figure 2**) coupled to meta-genomic sequencing of associated subgingival biofilms using Illumina technology
- Systemically healthy non-smokers contributed (**Figure 1**)
  - (i) 'diseased' gingival papilla (bleeding-on-probing, probing depth  $\geq 4\text{mm}$ , clinical attachment loss  $\geq 3\text{mm}$ )
  - (ii) 'healthy' papilla (no bleeding-on-probing, probing depth  $\leq 4\text{mm}$ , and clinical attachment loss  $\leq 4\text{mm}$ )
- Mixed model regression utilized for assessment of specific differences between disease groups, clusters of characteristic signatures identified using mixture model-based clustering whilst accounting for covariates



**Figure 2:** Workflow Diagram of the Resolve Molecular Cartography Technology



**Figure 3:** Bacterial Signals (green) in Diseased Tissue Sample

## RESULTS

- Specific spatial transcriptomic signatures exist, that
  - (i) help differentiate 'healthy' and 'diseased' status
  - (ii) different progression rates
- These signatures can be contributed to defined cell populations and locations within tissues
- Driven by specific microbial complexes (**Figure 3**)

## CONCLUSION

These findings could help to improve early diagnostics of progressive disease, as well as inform targeted therapeutic approaches.

## REFERENCES

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