CHARACTERIZATION OF MICROFLORA IN FELINE ODONTOCLASTIC RESORPTIVE LESION (FORL)

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Introduction
The Feline Odontoclastic Resorptive Lesion (FORL) is a painful feline oral disease that leads to tooth loss for which the aetiology is unknown, but believed to be multifactorial. The aim of this study is to investigate the role of the oral microflora in FORL.

Methods
Aerobic and anaerobic culture coupled with PCR, Sanger sequencing of 16S rDNA and BLAST search and bar-coded Illumina MiSeq high throughput sequencing of the hypervariable V4 region of 16S rDNA (HTP-sequencing) was used to identify bacteria in subgingival plaque of 25 healthy cats and 40 cats with FORL. HTP-sequencing data was processed with QIIME, clustered in Operational Taxonomic Units (OTUs) and assigned to appropriate taxonomy. Data reduction and diversity analyses were performed with Paleontological statistics (PAST).

Results
From 1040 culture isolates, 17 novel species (< 97% similarity) were identified (6, healthy; 11, FORL). Utilizing HTP-sequencing 441 OTUs were identified in cats (353 healthy; 396 FORL). No single species was identified to be exclusively associated with FORL, but Bergeyella, Prophyromonas, Capnocytophaga, Lampropedia, Moraxella, and Treponema were the six most common genera identified in the feline oral cavity. Principle component analysis (PCA), species richness and species diversity indices of the microbiome data did not show a clear difference between the healthy and FORL group. However, two step cluster analysis of the microbiome data identified two sub-groups within the FORL group, one cluster showed a clear separation on PCA and significant lower species richness and species diversity from the other FORL cluster and the healthy cats.

Conclusion
Specific microorganisms associated with FORL were not identified, but a subgroup of cats with FORL with an altered microbiome was observed. Since the oral microflora is polymicrobial in nature, a slight change in this polybiotic synergy could led to dysbiosis, resulting in community virulence and inflammation that exacerbate FORL.