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BOOK OF ABSTRACTS

lymphocytes, key modulators of antitumor immune responses. Specifically, we assessed the frequency of regulatory $CD4^+$ $CD25^+$ $CD127^{neg}$ $Foxp3^+$ T cells (Tregs), including those co-expressing CD39 ($CD39^+$ Tregs), as potential biomarkers to inform clinical outcomes.

Methods
Patients with advanced CSCC treated with cemiplimab were prospectively included. Patients were classified as having 'clinical benefit' (CB) if they achieved a RECISTv1.1-defined complete response (CR), partial response (PR), or stable disease (SD). Patients with progressive disease (PD) were classified as 'no clinical benefit' (NO-CB). Peripheral blood samples were analyzed at baseline (T0), after the first cemiplimab infusion (T1), and at a later on-treatment timepoint (T2). Circulating total Tregs and $CD39^+$ Tregs were quantified by multiparametric flow cytometry using monoclonal antibodies against CD45, CD3, CD4, CD127, CD25, CD39, PD-1, Foxp3.

Results
Among 23 included patients, 17 achieved CB, while 6 had NO-CB. Consistent with previous reports, anti–PD-1 therapy hampered PD-1 expression on peripheral blood cells using commercially available antibodies; accordingly, all patients showed a significant reduction in detectable PD-1 expression on total $CD3^+$ T cells both after the first treatment cycle and at later on-treatment timepoints (T0 vs T1, p=0.0001; T0 vs T2, p=0.0001).
At baseline, patients with NO-CB displayed a significantly higher frequency of circulating $CD39^+$Tregs compared with CB patients (T0, p= 0.0126). Notably, this difference persisted during treatment, with NO-CB patients showing consistently higher levels of $CD39^+$ T regs at both early and later timepoints (T1, p=0.0197; T2, p=0.0063).

Conclusions
These results suggest that higher levels of circulating $CD39^+$ Tregs, assessed at baseline and during treatment, may be used as a predictive biomarker of resistance to anti-PD-1 therapy in advanced CSCC. Larger studies including detailed quantitative and functional characterization of circulating CD39+ Treg cells before and after ICIs are needed.

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Development and Preliminary Evaluation of Postbiotic-Based Chitosan and Carbopol Spray Formulations for Supportive Skin Care in Oncology-Associated Cutaneous Complications

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Background
Cutaneous toxicities induced by cancer therapies such as radiotherapy, chemotherapy, and targeted treatments commonly result in skin barrier disruption, pH imbalance, and increased susceptibility to microbial colonization, particularly in immunocompromised patients. Chitosan, a naturally derived biopolymer with film-forming, adhesive, antimicrobial, and barrier-supportive properties, and carbopol, a polymer providing formulation stability and sprayability, are suitable for oncology-related dermal applications. In parallel, postbiotic solutions derived from *Lacticaseibacillus casei* offer antimicrobial activity without the safety concerns associated with live microorganisms; however, data on postbiotic dose selection and incorporation into topical spray systems for therapy-compromised skin remain limited.

Methods
A cell-free postbiotic solution was produced by controlled fermentation of *L. casei*, followed by filtration and sterilization. Antimicrobial and antibiofilm activities of the postbiotic solution was evaluated in vitro against selected skin-associated microorganisms. Based on these findings, an appropriate dose was determined and incorporated into two topical spray formulations: a chitosan-based and a carbopol-based system. Physicochemical characterization included pH determination to assess skin compatibility.

Results
The *L. casei*–derived postbiotic solution showed clear antimicrobial activity, supporting its role in microbial load control and guiding dose selection. *The* postbiotics exhibited low to moderate inhibition zones against *B. subtilis*, *E. faecalis*, *P.s aeruginosa*, and *Candida* spp. in the agar diffusion assay, indicating a broad-spectrum antimicrobial activity. MIC results demonstrated that the postbiotics exerted inhibitory effects particularly against Gram-positive bacteria and yeast species, with MIC values ranging between 2500 and 5000 µg/mL. At a concentration of 10 mg/mL, *L. casei*–derived postbiotics inhibited biofilm formation by 51.94% in *Staphylococcus aureus* and 42.35% in *Pseudomonas aeruginosa*. Both spray formulations showed skin-compatible pH values for oncology-related compromised skin, and incorporation of the postbiotic solution did not affect formulation homogeneity or spray performance; antimicrobial testing of the final formulations is ongoing.

Conclusions

This study reports the development of postbiotic-based topical sprays for supportive skin care in oncology-associated cutaneous complications. Antimicrobial and antibiofilm activities of the *L. casei*–derived postbiotic solution supported dose selection, while chitosan and carbopol provided suitable functions for compromised skin. The cell-free nature of the formulation may offer a safety advantage for immunocompromised patients and supports further in vitro microbial and pharmaceutical evaluation for oncology-related dermal care.

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Dissecting the shift in YAP/TAZ signalling modalities with disease progression in cancer-associated fibroblasts of cutaneous squamous cell carcinoma.

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Background

Cancer-associated fibroblasts (CAFs) can promote or supress tumour growth. YAP/TAZ signalling is often dysregulated in cancers, including cutaneous squamous cell carcinoma (cSCC), and YAP-TEAD inhibitors are already in clinical trials for cancers. Little is known about YAP/TAZ signalling in CAFs during cancer progression and how inhibition affects CAF phenotype. Using isogenic fibroblast lines isolated in our lab from normal skin tissue, primary tumour, and recurrent tumour, we aim to dissect the distinct YAP/TAZ signalling modalities in CAFs. We hypothesise that YAP/TAZ signals through different co-factors in cancer cells and CAFs and targeted inhibition will affect these cell types differently during cSCC progression.

Methods

Changes in YAP/TAZ signalling with disease progression were assessed using ATAC/RNAseq datasets, 2D and 3D skin co-culture models, YAP/TAZ transient knockdowns, and YAP-TEAD inhibitor treatment as a discovery tool.

Results

Immunofluorescent staining of cSCC tissue shows increased stromal YAP/TAZ expression, and RNAseq and RT-qPCR data confirmed YAP/TAZ signalling in CAFs increases with disease progression. While YAP/TAZ signal through TEADs in cSCC keratinocytes, analysis of ATAC/RNAseq data indicates a shift in association of YAP/TAZ between TEAD and AP-1 in our CAF progression series. Accessibility of AP-1 binding motifs peaks in primary CAFs, and TEAD motif accessibility peaks in recurrent CAFs. While normal fibroblasts respond strongly to TEAD-specific inhibition, impacting proliferation, contraction, and ECM remodelling, CAFs become less responsive with disease progression, providing further evidence of a signalling switch. We are currently investigating how this switch influences CAF-cancer cell crosstalk by modulating YAP-TEAD and AP-1 signalling in 3D organotypic co-cultures.

Conclusions

These results suggest YAP/TAZ signalling is altered in CAFs and evolves with disease progression. A switch in association of YAP/TAZ with downstream transcription factors could affect efficacy of TEAD inhibitors in clinical development at different stages of cSCC.

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Efficacy and safety of tirbanibulin 1% ointment in elderly patients with actinic keratosis: a pooled analysis of Phase 3 clinical trials

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Background

Actinic keratosis (AK) is more common in patients with cumulative sun exposure, and therefore elderly patients are at a higher risk of developing the disease. These patients often present multiple comorbidities and polypharmacy, which must be considered when selecting a treatment. This post-hoc analysis aims to evaluate the efficacy and safety of tirbanibulin in very elderly patients (≥80 years),

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This is to certify that

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