

Assessing Targeted Proteomics in Inflammatory Skin Diseases with the Tape-stripping Method

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This cross-sectional study aimed to determine the detectability of a panel of 92 inflammatory biomarkers in tape strips from the skin of patients with inflammatory skin diseases, and whether this depended on the number of tape strips taken. Furthermore, the biomarker levels in the patients were compared with those in healthy controls. Eight consecutive uniform tape strips (each 3.8 cm²) were obtained from 2 adjacent skin sites from 8 atopic dermatitis, contact dermatitis, and psoriasis patients, respectively, and 5 controls. Three separate analyses were carried out using the Olink® Target Inflammation panel: (i) all 8 tape strips (1–8) from 1 skin site and from the other skin site, (ii) the first 4 tape strips (1–4), and (iii) the next 4 tape strips (5–8). Biomarkers were above the detection limit for 65.7% of atopic dermatitis, 70.2% of psoriasis, and 45.1% of contact dermatitis samples. There were no significant differences in biomarker levels between the use of 4 or 8 tape strips, or between the first and last 4 tape strips. In general, atopic dermatitis and psoriasis patients were distinguishable from controls, whereas contact dermatitis patients were not. Based on the overall data quality, analysing protein signatures in tape strips with the targeted inflammatory panel is feasible.

Key words: atopic dermatitis; contact dermatitis; proteomics; psoriasis; skin tape.

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Tape-stripping provides epidermal material that can be used to analyse different cutaneous biomarkers, e.g., with proteomics (1). Due to the minimally invasive nature of the method, it is optimal for obtaining repeated samples (2). It holds great potential for following disease course over time (2), determining severity and prognosis (3, 4), or evaluating treatment response (5). Additionally, the method is applicable and preferable to paediatric patient groups (2, 3) and even sensitive areas such as the

SIGNIFICANCE

Examining inflammatory skin diseases with the minimally invasive tape-stripping technique seems advantageous when repeated samples must be collected or when samples are obtained from children or from sensitive areas such as the hands or face. However, obtainment and analysis of tape strip material has some methodological challenges. This study finds that the tape-stripping method, using 4 tape strips combined with a protein analysis technology that has low sample volume requirement, can be used to measure protein signatures in the skin of patients with psoriasis and atopic dermatitis but needs to be further optimized for use in patients with contact dermatitis.

hands and face (6). In contrast, biopsy specimens possess strength in the ability to examine all layers of the skin (7), yet the invasiveness of the method comes with limitations.

The use of tape strips has been investigated in different patient groups such as atopic dermatitis (AD) (4, 8–14) and to a lesser degree psoriasis (9, 10, 15, 16) and hand eczema, including irritant or allergic contact dermatitis (CD) (6, 17–20). Although these studies show promising results, a surprisingly large variation in methodology challenges the practical applicability of the technique. The number of applied tape strips differs from study to study, along with which layers and how many tape strips are pooled before different types of analyses. Choosing an appropriate number of tape strips is pivotal. It requires finding the balance between, on one hand, obtaining enough epidermal material to detect the desired proteins, and on the other hand, limiting the time-consuming steps to make the sampling method favourable in an everyday clinical setting.

Different methods for protein analysis of the tape strip material can be employed. Most previous studies have used Meso Scale Discovery (MSD) or mass spectrometry (21, 22). One of the main challenges with tape strips is the small amount of skin material obtained and, hence, the low protein concentrations. Olink® is a multiplex immunoassay that requires very little material compared with conventional immunoassays (22). Thus, it may be an optimal method for analysing tape strips, yet the technology has traditionally been used on blood speci-

mens. So far, investigation into the combination of tape strips and Olink® is still in its early stages (21–24).

In this study, we investigated the tape-stripping method for examining protein signatures of the skin in patients with AD, CD, and psoriasis using the targeted inflammatory Olink® panel. We evaluated the use of different numbers of tape strips and investigated whether the depth of the tape strips influences the detection of disease-relevant proteins.

MATERIALS AND METHODS

This method study was conducted as a part of the BIOSKIN cohort study that has been described in detail (25). The study is approved by the Scientific Ethical Committee of the Capital Region (H-21032986) and the Danish Data Protection Agency (P-2021-435). An overview of the study design is presented in Fig. 1A. Patients with AD ($n=8$), CD ($n=8$), psoriasis ($n=8$), and healthy controls

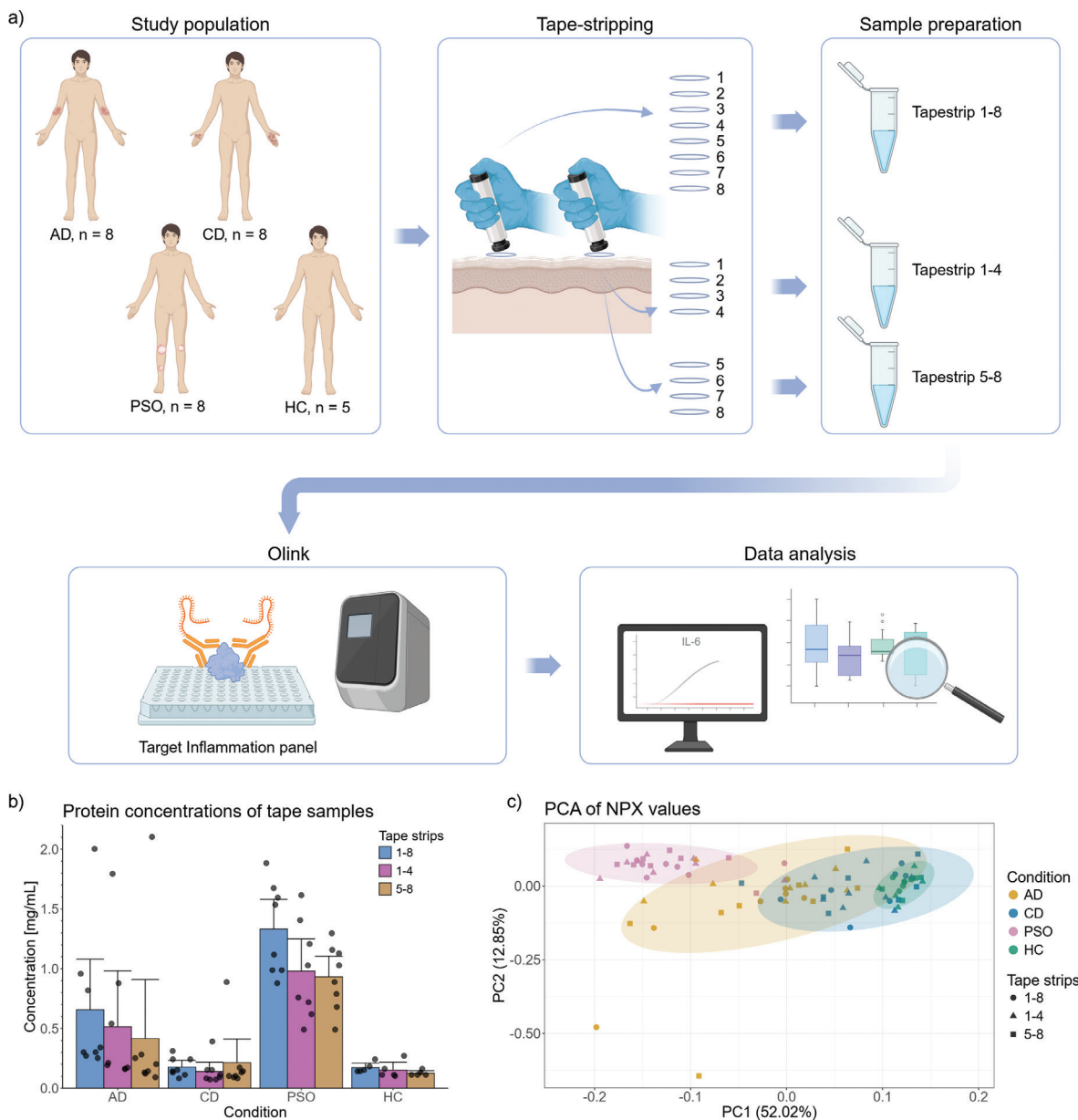


Fig. 1. Study design, protein concentrations, and principal component analysis. (A) Overview of study design. Patients with atopic dermatitis (AD), contact dermatitis (CD) and psoriasis were enrolled ($n=8$ in each group) along with healthy controls (HC, $n=5$). Two sets of 8 tape strips were obtained from each study participant – lesional samples for disease groups and samples from healthy skin for controls. In one set, all 8 tape strips were pooled before analysis, in the other, number 1–4 and 5–8 were pooled separately. Samples were analysed with the Target Inflammation panel from Olink® including 92 protein assays. (B) Bar plot of protein concentrations for samples from patients with AD ($n=8$), CD ($n=8$), psoriasis ($n=8$), and healthy controls ($n=5$). Protein concentrations from tape strips number 1–8, 1–4 and 5–8, respectively, are depicted for each disease group, with individual values as dots and error bars showing the bootstrapped mean standard errors. (C) Principal component analysis (PCA) of relative protein concentrations for patients with AD, CD, and psoriasis and healthy controls, with the percentage of variance explained by the first and second principal components (PC1 and PC2) in parentheses. Fig. 1A was created with BioRender.com. HC: healthy controls; NPX: Normalized Protein eXpression; PC: principal component; PSO: psoriasis.

($n=5$) were recruited from November 2023 to January 2024. Inclusion criteria were participants ≥ 18 years of age and able to read and understand Danish. Lesional sample sites were assessed with a local severity score (Table I) (25). Each lesion had to be homogeneous based on the local severity score and large enough to contain 2 sets of tape strips. Exclusion criteria were pregnancy, breastfeeding, and deprivation of liberty.

Tape-stripping

Circular 22 mm tape strips (D-Squame, CuDerm, Dallas, TX, USA) were applied with sterile tweezers to lesional skin for patient groups and healthy skin for controls. Eight consecutive tape strips from two homogeneous sample sites were obtained. The sampling site was marked with a skin marker on the periphery of the tape strip to ensure that the following 7 tape strips were obtained from the same area. Application of each tape strip was followed by 10 s of standardized pressure (225 g cm^{-2}) using a pressure applicator (D500, D-Squame, CuDerm) (Fig. S1A). Following removal with the tweezers (Fig. S1B), each tape strip was immediately placed in an empty 2 mL microtube (SARSTEDT, Nümbrecht, Germany) with the adhesive side facing towards the lumen. All tubes were stored at -80°C until analysis.

Protein extraction

All tape strips were pooled before protein analysis as either all 8 tape strips (1 to 8) or tape strip numbers 1 to 4 and 5 to

8. The pooled tapes were immersed in 800 μL lysis buffer (0.2% Triton X100 in PBS + cOmplete™ Mini Protease Inhibitor Cocktail [Cat # 11836153001, Roche, Basel, Switzerland]) and stirred overnight at room temperature. The lysates were then transferred to clean tubes, and 1 mL syringes (Cat # 2933.1101, Hounisen, Skanderborg, Denmark) were used to pass the samples through a 0.2 μm filter (Cat # 83.1826.001, SARSTEDT, Nümbrecht, Germany). The following protein extract was used for Olink® analysis (1 μL). Protein concentration was measured using the Pierce BCA protein assay (Cat # 23227, Thermo Fisher, Waltham, MA, USA). Samples were stored at -80°C until analysis.

Olink® analysis

For protein analysis, the Target Inflammation panel (96-well plate) from Olink® (Olink Proteomics AB, Uppsala, Sweden) including 92 protein assays was used. Detailed information can be found in Appendix S1. Samples with a total protein concentration higher than 1 mg/mL were normalized to 1 mg/mL. Remaining samples were not further normalized. Each sample either passed the quality control (QC) step, or they were marked with a "warning" based on a deviation larger than a pre-determined value (± 0.3) from the median value of all samples on the plate. For each assay, a limit of detection (LOD) was calculated based on negative controls plus 3 standard deviations (SD) according to the manufacturer's guidelines.

Table I. Baseline characteristics

	Total $n=29$	AD $n=8$	CD $n=8$	PSO $n=8$	HC $n=5$	p -values
Demographics						
Age, median (IQR)	52.0 (29.5)	47.5 (26.3)	54.5 (30.5)	53.0 (27.5)	35.0 (29.5)	0.674
Sex, females, n (%)	13 (44.8)	2 (25.0)	6 (75.0)	3 (37.5)	2 (40.0)	0.218
Disease relevant characteristics						
Topical treatment ^a , n (%)	8 (33.3)	2 (25.0)	5 (62.5)	1 (12.5)	N/A	
Systemic treatment, n (%)	11 (45.8)	4 (50.0)	4 (50.0)	3 (37.5)	N/A	
Disease-specific severity score, median (IQR)						
EASI Range 0–72	N/A	6.3 (10.7)	N/A	N/A	N/A	
HECSI Range 0–360	N/A	N/A	69.0 (42.0) ^b	N/A	N/A	
PASI Range 0–72	N/A	N/A	N/A	8.95 (6.9)	N/A	
Disease-specific sample site local severity score, median (IQR)						
AD ^c Range 0–12	N/A	4.5 (3.3)	N/A	N/A	N/A	
CD ^d Range 0–18	N/A	N/A	6.5 (1.8)	N/A	N/A	
Psoriasis ^e Range 0–12	N/A	N/A	N/A	6.0 (1.0)	N/A	
Sample site, n (%)						
Chest	1 (3.4)	1 (12.5)	–	–	–	
Back	8 (27.6)	3 (37.5)	–	3 (37.5)	2 (40.0)	
Arm	5 (17.4)	2 (25.0)	–	2 (25.0)	1 (20.0)	
Palm	6 (20.7)	–	5 (62.5)	1 (12.5)	–	
Dorsal hand	2 (6.9)	–	2 (25.0)	–	–	
Buttocks	1 (3.4)	–	–	1 (12.5)	–	
Leg	5 (17.2)	2 (25.0)	–	1 (12.5)	2 (40.0)	
Foot	1 (3.4)	–	1 (12.5)	–	–	

p -values were calculated for demographic variables using ANOVA for age and χ^2 -test for sex.

^aTopical treatment on the sample site within the last week. ^bOne missing value. ^cSample site local severity score for atopic dermatitis (AD) patients was calculated based on an evaluation scale from 0–3 for erythema, oedema/papulation, excoriation, and lichenification, resulting in a score from 0 to 12. ^dSample site local severity score for contact dermatitis (CD) patients was calculated based on an evaluation from 0–3 for erythema, infiltration, vesicles, fissures, scaling, and oedema, resulting in a score from 0 to 18. ^eSample site local severity score for psoriasis patients was calculated based on an evaluation scale from 0–4 for erythema, induration, and scaling, resulting in a score from 0 to 12.

EASI: Eczema Area and Severity Index; HC: healthy controls; HECSI: Hand Eczema Severity Index; IQR: interquartile range; N/A: not available; PASI: Psoriasis Area and Severity Index; PSO: psoriasis.

Statistical analysis

All statistical analyses were carried out in R version 4.3.0 (R Foundation for Statistical Computing, Vienna, Austria) (26). Statistical tests were conducted using a significance level of 0.05. Differences between using 8 tape strips and 4 tape strips for each protein assay (1 to 8 vs 1 to 4 and 1 to 8 vs 5 to 8) and differences between using the superficial or deeper layers (1 to 4 vs 5 to 8) were calculated for each patient group using Wilcoxon matched-pairs signed rank test, as implemented in the *rstatix* library (27).

Additionally, a Kruskal–Wallis test was performed for each protein assay on data from tape strips 1 to 8 to find protein assays with different NPX values in any of the disease groups compared with the controls. The test was performed using the *kruskal.test* function in R, and post-hoc analysis was performed for selected contrasts using a Dunn's test as implemented in the *dunn.test* library (28). The resulting *p*-values were adjusted according to the Benjamini–Hochberg method. The non-parametric Kruskal–Wallis test was selected on the basis that the data did not follow a normal distribution when examined with the Shapiro–Wilk test.

Principal component analysis was performed on the scaled and centered NPX values using R's *prcomp* function. The first two principal components were plotted using *ggplot2* (29).

RESULTS

To assess whether the inflammatory Olink® panel could be used to measure proteins obtained from tape strips, we first collected samples from patients with AD, CD (ICD *n* = 3, ACD *n* = 2, mixed ACD/ICD *n* = 2, unknown aetiology *n* = 1), psoriasis, and healthy controls (Fig. 1A). Baseline characteristics are shown in Table I.

Protein concentrations were highest in tapes obtained from patients with psoriasis, and lowest in patients with CD and healthy controls (Fig. 1B, Table SI). A principal component analysis (PCA) revealed that the groups with AD and psoriasis could be distinguished from the healthy

controls, while this was not the case for the group with CD (Fig. 1C).

Quality of Olink® data and comparison of number and depth of tape strips

Quality assessment of the Olink® results is presented in **Table II**. Overall, 88.5% of samples passed quality control without a warning. This was highest for patients with psoriasis (95.8%) and healthy controls (93.3%), and lowest for patients with AD or CD (83.3% in both groups). Only 187 (2.3% of all samples) resulted in "No data". Of all NPX values 54.7% were above LOD, and thus our results were above the recommended exclusion limits of the assay. Detectability was highest in patients with psoriasis and AD (70.2% and 65.7% of all samples were above LOD, respectively) and lowest in patients with CD and controls (45.1% and 27.7% of all samples were above LOD, respectively). For more detailed information regarding which protein assays were detectable for each group, please refer to Tables SII–SV. LOD for all assays and the related mean NPX values for all disease groups and healthy controls are presented in Table SVI.

When analysing all proteins and all groups with regard to using either 4 tape strips or all 8 tape strips and using deeper or superficial tape strips, we found no statistically significant differences (Table SVII).

Comparison between disease groups and healthy controls

When we compared the expression level of all measured proteins from each disease group with healthy controls, we found that patients with psoriasis were the most distinguishable from healthy controls (Table SVIII and Fig. S2). Of the differentially expressed proteins, 58 were upregulated in patients with psoriasis and 39 in patients with AD compared with controls, with an overlap of 35 upregulated proteins in common (Fig. S3). Only IL-1 α was significantly downregulated – this was consistent across all disease groups. Sixteen relevant proteins from the Target Inflammation panel were selected (4 interleukins, 4 chemokines, 4 cytokines, and 4 miscellaneous

Table II. Quality of Olink® data

	Total <i>n</i> = 29	AD <i>n</i> = 8	CD <i>n</i> = 8	PSO <i>n</i> = 8	HC <i>n</i> = 5
Samples for all protein assays, <i>n</i>	8,004	2,208	2,208	2,208	1,380
NPX values above LOD (of samples for all protein assays) ^a , <i>n</i> (%)					
Overall	4,275 (54.7)	1,389 (65.7)	954 (45.1)	1,550 (70.2)	382 (27.7)
1 to 8	1,372 (55.3)	439 (68.5)	289 (44.9)	511 (69.4)	133 (28.9)
1 to 4	1,465 (54.9)	464 (63.0)	350 (47.6)	519 (70.5)	132 (28.7)
5 to 8	1,438 (53.9)	486 (66.0)	315 (42.8)	520 (70.7)	117 (25.4)
Passed quality control (of samples for all protein assays), <i>n</i> (%)					
Overall	7,084 (88.5)	1,840 (83.3)	1,840 (83.3)	2,116 (95.8)	1,288 (93.3)
1 to 8	2,208 (82.8)	552 (75.0)	552 (75.0)	644 (87.5)	460 (100.0)
1 to 4	2,484 (93.1)	644 (87.5)	644 (87.5)	736 (100.0)	460 (100.0)
5 to 8	2,392 (89.7)	644 (87.5)	644 (87.5)	736 (100.0)	368 (80.0)

^a Samples with "No data" were excluded.

AD: atopic dermatitis; CD: contact dermatitis; HC: healthy controls; LOD: limit of detection; NPX: Normalized Protein eXpression; PSO: psoriasis.

proteins) based on previously published literature and high expression in our data set. Relative concentrations for all disease groups and healthy controls of the selected proteins are presented as boxplots in **Fig. 2**.

All cytokines and most of the interleukins were significantly higher in patients with psoriasis compared with healthy controls including IL-17, IL-6, IL-8, IFN- γ , and TNF, which are all well known for psoriasis (see Table

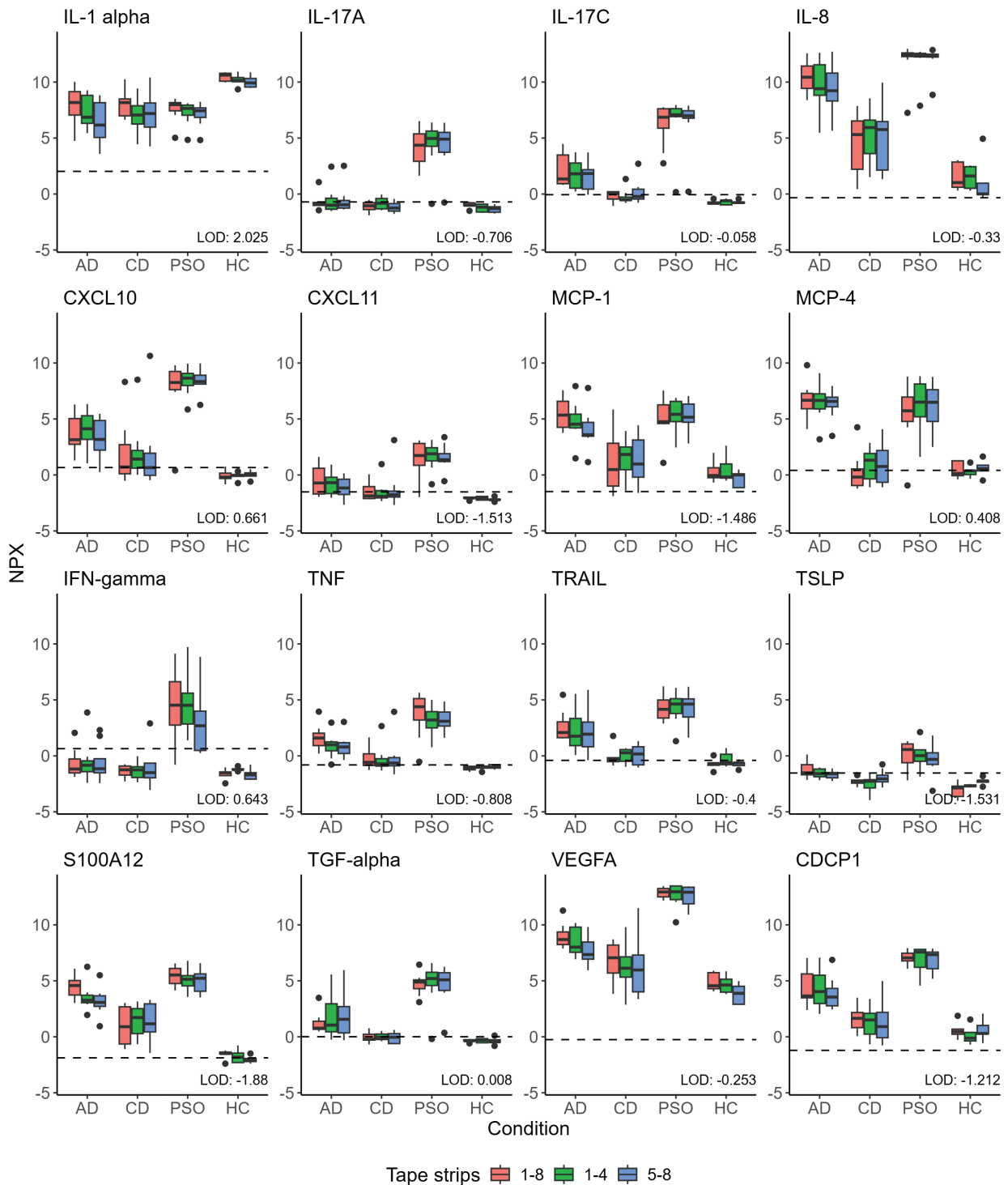


Fig. 2. Boxplots of relative protein concentrations for selected proteins from the Olink® Target Inflammation panel. Boxplots depict Normalized Protein eXpression (NPX) for samples from patients with atopic dermatitis (AD, $n=8$), contact dermatitis (CD, $n=8$), and psoriasis (PSO, $n=8$) and healthy controls (HC, $n=5$). For each group, relative protein concentrations (NPX) from tape strips numbers 1–8, 1–4, and 5–8, respectively, are presented. The dotted line depicts the limit of detection (LOD). AD: atopic dermatitis; CD: contact dermatitis; CXCL: chemokine (C-X-C motif) ligand; HC: healthy controls; IFN: interferon; IL: interleukin; LOD: limit of detection; MCP: monocyte chemoattractant protein; NPX: Normalized Protein eXpression; PSO: psoriasis; TNF: tumour necrosis factor; TRAIL: TNF-related apoptosis-inducing ligand receptor 1; TSLP: thymic stromal lymphopoietin; VEGFA: vascular endothelial growth factor A.

SVIII). Most cytokines and chemokines were higher in patients with AD compared with healthy controls. In the protein panel, especially IL-4, IL-10, IL-13, IL-17C, IL-22RA1, IL-33, S100A12, and TSLP are relevant for AD. Except for IL-4, IL-10, and IL-13, we found all upregulated (see Table SVIII). The patient group with the least detectability in the selected proteins was CD. Nonetheless, patients with CD demonstrated over 50% of samples above LOD for the proteins IL-1 α , IL-8, MCP-1, CXCL10, TNF, TRAIL, CDCP1, S100A12, and VEGFA (see Fig. 2 and Table SIII).

DISCUSSION

This study shows that the tape-stripping method, using only 4 tapes combined with the Olink[®] technology, can be used to measure protein signatures in the skin of patients with psoriasis and AD but needs to be further optimized for use in CD. In general, patients with psoriasis or AD could be distinguished from healthy controls based on the applied method, whereas this was not the case for patients with CD. We found no significant difference in the comparison of 4 or 8 tape strips across all disease groups and healthy controls. Similarly, no significant difference was found between tapes from the superficial or deeper layers. Inflammatory biomarkers that end up in the stratum corneum are expressed by living resident skin cells and recruited immune cells in dermis and deeper epidermis (8). Clausen et al. hypothesized that biomarkers are transferred to the extracellular space with lipids secreted from deeper epidermis, which might explain why they found no concentration gradient across the stratum corneum (8). Our results support this view of stratum corneum as one compartment with a consistent molecular milieu.

The method applied in this study enabled detectability of many key inflammatory biomarkers in psoriasis including IFN- γ , TNF, IL-6, IL-8, IL-12B, IL-17A, IL-17C, and CXCL10 (10, 31) and concentrations for these proteins were all significantly higher in lesional psoriasis skin compared with healthy skin. A significant increase was observed for TGF- α only in patients with psoriasis. The proliferative effect of TGF- α on keratinocytes and its overexpression in psoriatic lesional epidermis are well known (32), hence our results confirm previous findings and underscore that Olink[®] captures meaningful inflammatory signals. Most of the previous tape-strip studies of lesional skin in patients with psoriasis have looked at RNA and not protein expression (9, 10, 16). He et al. investigated the correlation between gene expression levels in tape strips vs biopsies in psoriasis compared with healthy controls (10). Notably, tape strips revealed a larger differentiation than biopsies regarding CXCL9/10 in psoriasis skin compared with healthy skin. Our results showed a significant upregulation of CXCL9/10 in psoriasis compared with healthy skin. As we did not directly

compare tape strips and biopsies, it remains unclear whether CXCL9/10 levels would be higher in tape strips.

According to two reviews summarizing proteomic studies regarding AD (21, 22), only a single study has examined tape strips from AD patients with Olink[®]: He et al. compared lesional tape strips before and after treatment with dupilumab and demonstrated significant decreases in e.g., IL-6, IL-8, IL-17C, CCL3, CXCL1, MCP-1, MCP-4, TNF, S100A12, MMP-1, and MMP-10 in lesional skin following treatment (23). In our study, these proteins were significantly higher in patients with AD compared with healthy individuals.

One of the key inflammatory biomarkers in AD, IL-4 (33), was not detectable in our study. Other studies have previously demonstrated how this cytokine is difficult to detect when analysing only the superficial layers of the skin with tape strips (34, 35). This is supported by Andersson et al. analysing the cytokine profile in tape strips from paediatric AD patients using MSD (3). They found that biopsies showed a type 2 skewed response whereas tape strips revealed increased biomarkers related to innate immune response. This key finding suggests that the protein signature of AD may vary based on the applied skin sampling method. Similar to our study, Andersson et al. found high expression of IL-8 in tape strips from lesional skin in patients with AD.

Patients with CD exhibited lowest detectability and were overall not distinguishable from healthy controls. The samples were taken mainly from the hands, where the expression of inflammatory biomarkers is typically lower than for other skin sites and where the physical barrier differs too (36, 37). The results might have been more favourable if the control group had included samples from the hand. However, it was possible to detect some of the proteins that were upregulated in recent hand eczema studies using proteomics and transcriptomics, such as IL-6, S100A12, CXCL9, CXCL10, TNF, TNFRSF9, TNFSF14, CSF-1, and OSM (6, 20, 36). In line with our results, a recent study demonstrated overall low detectability in CD and hand eczema patients with the inflammatory panel from Olink[®] compared with two other multiplex immunoassays (24).

Significant downregulation of IL-1 α compared with controls was consistent across all disease groups. The proinflammatory cytokine is constitutively expressed in basal keratinocytes, playing a role in maintaining skin barrier homeostasis (30, 38). As increased expression of IL-1 α has been identified as a driver of cutaneous inflammation, one might expect it to be upregulated across the investigated inflammatory skin diseases in this study (38). The observed downregulation could be due to the presence of IL-1 α in stratum basale (30) and the fact that expression levels in superficial tape strips do not necessarily align with those in biopsies (36). Nevertheless, our findings were consistent with previous studies regarding both tape strips and biopsies (8, 30, 39).

Tape-stripping is a promising method with an inarguable advantage over biopsy specimens due to its minimally invasiveness, which is particularly favourable in the case of children or sensitive areas such as the hands (2). Furthermore – despite the cross-sectional nature of this study – we believe that one of the undeniable strengths of tape-stripping is its applicability in longitudinal studies or in monitoring patient progress over time in routine clinical practice. However, it is important to keep in mind that the skin material obtained and the immunologic signatures differ in tape-stripping and biopsies (3, 11, 14, 40) and the two methods cannot be used interchangeably. Instead, they offer different advantages and limitations. Another important consideration regarding tape-stripping is the lack of methodological consistency (4, 6, 8–13, 15–19). In order for tape-stripping to be implemented in clinical practice, the method must be streamlined to a greater extent in future studies. In addition, immunologic biomarkers vary according to anatomical sample site – in particular the palm differs from other sample sites (36). This too must be considered when evaluating analysis of tape strips.

Limitations

This study has some limitations that must be considered. Eight patients were enrolled in each disease group and 5 healthy controls participated. The size of the study population could be part of the explanation as to why we found no significant differences between the numbers and depth of the tape strips. Our results do, however, align with the study by Clausen et al. (8). Total protein concentration varied substantially between the disease groups. To avoid overlooking a biological variation between diseases, the NPX values were not adjusted for absolute protein concentration. This is, however, a methodological limitation that must be considered when interpreting the results. We did not investigate whether there was a difference in other numbers of tape strips and, hence, it remains unclear whether, e.g., 1 or 2 tape strips contain enough skin material to detect different protein signatures. Due to the low protein concentrations in the patient group with CD, it would probably not be advantageous to obtain fewer tape strips, unless other methodological aspects were optimized, e.g., changing the lysis buffer volume and thus the protein concentration. Lack of significance in protein expression levels from patients with CD could be explained by the heterogeneity of the patients' aetiological subtypes, low total protein concentration due to technical circumstances in the tape-stripping procedure of this disease group, and/or by the lack of site-matched samples in the control group. Another limitation of the study was that we used only one method to detect proteins, and the different available methods to detect small amounts of proteins need to be compared. The Target Inflammation panel used from Olink® consists of 92 protein assays, thus many other

proteins that could be relevant to the diseases have not been part of the current study.

Conclusion

The tape-stripping method applied in this study is suitable for investigating protein signatures in inflammatory skin diseases. Of the 3 patient groups, patients with psoriasis demonstrated the highest protein concentrations and highest detectability of the inflammatory biomarkers in question, and they were the most distinguishable from healthy controls. Across all groups, there were no significant differences in the number of tape strips or depth of tape strip material. Analysing cutaneous inflammatory biomarkers in tape strips with Olink® appears to be favourable in patients with psoriasis or AD.

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Data availability statement: The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

IRB approval status: This study has been approved by the Danish National Committee on Health Research Ethics (H-21032986). The patients provided their written informed consent prior to inclusion in the study.

Conflict of interest disclosures: LS has been a paid speaker for AbbVie, Eli Lilly, Takeda, Sanofi, Pfizer, and LEO Pharma, and has been a consultant or has served on Advisory Boards with AbbVie, Eli Lilly, Novartis, Pfizer, LEO Pharma, Janssen, UCB, Almirall, Galderma, Bristol-Myers Squibb, Stada, Takeda, and Sanofi and has received research and educational grants from Bristol-Myers Squibb, Janssen, Sanofi, UCB, Almirall, the Danish National Psoriasis Foundation, and the LEO Foundation. The remaining authors have no conflicts of interest to declare.

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