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The diagnostic value of miR-1-3p in atopic dermatitis and its correlation with inflammatory factors

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Abstract

Introduction and Objective. MicroRNAs (miRNAs), such as miR-1-3p, play a crucial role in inflammatory processes, including atopic dermatitis (AD). The aim of the study is to investigate the role of miR-1-3p in AD, and its correlation with inflammation.
Materials and Method. miR-1-3p level was analyzed using qRT-PCR. Its diagnostic value was assessed via ROC curve analysis. Correlations between miR-1-3p and inflammatory factors were assessed by Spearman's rank correlation coefficient. Proliferation of HaCaT cells were detected by CCK-8. The influence of miR-1-3p for inflammatory factors were examined through enzyme-linked immunosorbent assay (ELISA). Target genes of miR-1-3p were predicted using bioinformatics databases.
Results. miR-1-3p was upregulated in AD patients and had a high diagnostic value (area under ROC curve=0.927, sensitivity=0.830, specificity=0.934) for AD. miR-1-3p was negatively correlated with lymphocytes (r=-0.558), and positively correlated with CRP (r=0.570), IL-6 (r=0.511), and IL-22 (r=0.596) levels, respectively. A high miR-1-3p level in HaCaT cells suppressed proliferation and increased inflammatory cytokine levels. Bioinformatics analysis identified 61 ovrerlapping target genes of miR-1-3p, enriched in pathways associated with inflammation and immune response.
Conclusions. miR-1-3p is elevated in AD and associated with inflammation, suggesting a role in AD pathogenesis. Its effects on HaCaT cells and correlation with inflammatory markers indicate a mechanistic involvement in AD.

Key words

inflammation, miR-1-3p, atopic dermatitis, HaCaT cell

INTRODUCTION AND OBJECTIVE

Atopic dermatitis (AD) is a persistent inflammatory skin condition [1]. This condition is marked by inflammatory rashes that may appear on different areas of the body, and it is generally observed in children starting around five years old, occasionally continuing into adulthood [2]. The development of AD is influenced by multiple factors, including intricate interactions among genetic predispositions, environmental elements, immune system irregularities, and compromised skin barrier function [3]. Disruption of the skin barrier in individuals with AD results in increased transepidermal water loss and a greater entry of allergens, which triggers an inflammatory reaction [4]. Inflammation plays an important role in AD, including lymphocytes, macrophages, eosinophils, and mast cells accumulating within lesions [5–7]. Proinflammatory cytokines, comprising CRP [8], IL-6 [9], and IL-22 [10], are elevated in AD patients and contribute to the pathogenesis of the disease. These factors contribute to the recruitment and activation of immune cells, promoting Th2 cell differentiation, and exacerbating skin inflammation [11].

MicroRNAs (miRNAs), including miR-1-3p, have been implicated in regulating inflammatory diseases. In particular, miR-1-3p modulates immune responses [12]. The status and importance of miR-1-3p in inflammatory diseases are becoming increasingly recognized, as it can influence the polarization of macrophages and the activation of signalling pathways, such as the STAT6 pathway, which is crucial in the context of inflammation and immune regulation [13, 14]. An miRNA expression profile performed by Luo et al. found that miR-1-3p was upregulated in AD patients [15]. miR-1-3p may drive AD pathogenesis and contribute to skin diseases. The function of miR-1-3p in skin pathology is an area of active investigation, with potential implications for skin inflammation and fibrosis.

The aim of the study is to investigate the function of miR-1-3p in AD, and its correlation with inflammation. The results obtained could potentially indicate future therapeutic strategies for AD management.

MATERIALS AND METHOD

Subject selection. The sample size was evaluated by GPower 3.1, the effect size was set at 0.5, α – 0.05, power – 0.9, and sample

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size – 90. AD patients (n=106) were randomly selected from The Second Hospital of Hebei Medical University between July 2021 – June 2024. The patients were diagnosed based on Williams's diagnostic criteria [16]. Age and gender-matched healthy individuals (n=137) without any skin conditions were designated as the control group. All participants were over 18 years of age. Exclusion criteria included: (1) severe liver, heart, or kidney diseases, or had received organ transplants; (2) pregnant or breastfeeding women; (3) use of immunosuppressive agents or corticosteroids in the two months before the study; (4) history of allergies to the study drugs. Using the SCORAD score [17], AD cases were categorized into mild, moderate, and severe groups, with cutoff values set at <25, 25–50, and >50.

Informed consent was obtained from each participant. The study was approved by the Institutional Review Board of the 2nd Hospital of Hebei Medical University. The experiment protocol followed the Declaration of Helsinki.

Cell culture and transfection. Human immortalized epidermal cells (HaCaT, BNCC339817) were cultivated in DMEM-H medium with 10% foetal bovine serum (FBS) at 37 °C with 5% CO_2. Negative control (NC) (50 nM), miR-1-3p mimic (50 nM), or miR-1-3p inhibitor (50 nM) were transduced into HaCaT cells by Lipofectamine 2000 (Thermo Fisher).

Cell proliferation. A total of 100 µL transfected cells (2×10^4 cells/mL) was sown into 96-wells and cultivated at 37° C. After incubation periods of 0, 24, 48, and 72 h, respectively, 10 µL CCK-8 solution was added to every well. After a 4 h incubation, readings were taken at 450 nm.

ELISA. Inflammatory factors, including CRP (ab260058), TNF-α (ab181421), IL-1β (ab214025), IL-6 (ab178013), IL-8 (ab214030), IL-22 (ab216170), and IgE (ab317776), were quantified in the serum samples and supernatant of cultured cells using ELISA kits. The result was detected at 450 nm.

qRT-PCR. Total RNA was gathered from serum and cell samples utilizing the miRNA extraction Kit (Absin, Shanghai). FastKing One Step RT-PCR kit (Tiangen, Beijing) was used to synthesize cDNA. RNU6B was used as endogenous control. Primer sequences for miR-1-3p were 5'-CAGTGCGTGTCGTGG AGT-3' (forward) and 5'-GGCCTGGAATGTAAAGAAGT-3' (reverse); for RNU6B were 5'-CTCGCTTCGGCAGCACA-3' (forward) and 5'-AACGCTTCACGAATTTGCGT-3' (reverse). The $2^{-\Delta\Delta Ct}$ method was employed to measure miR-1-3p levels.

Functional enrichment analysis for target genes. miR-1-3p targets were selected from TargetScan, miRWalk, and ENCORI databases. Overlap target genes were obtained by the Venn diagram. Functional enrichment analysis for overlapping genes was assessed using Gene Ontology (GO). Pathway enrichment annotations were performed by the Kyoto Encyclopedia of Genes and Genomes (KEGG). The figures were plotted by an online bioinformatics tool (http://bioinformatics.com.cn/?keywords=pathway).

Statistical analysis. The mean ± SD was used to express normally distributed continuous data, which was then evaluated using a t-test. Those not normally distributed were presented by the median and quartiles, and assessed by nonparametric test. The normality of continuous variables was assessed using the Shapiro-Wilk test, and the χ^2 test was used to analyze the qualitative variables. ROC curve determined the value of miR-1-3p for AD diagnosis. Correlation of miR-1-3p with inflammatory factors was explored using Spearman's rank correlation coefficient analysis. Correlation strength was interpreted as: weak (r = 0.1–0.3), moderate (r = 0.3–0.5), strong (r > 0.5). The threshold for significance was set to 0.05.

RESULTS

Clinical features of atopic dermatitis patients. Age and gender distribution were not significant between AD and controls (P>0.05) (Tab. 1). Sixty-six patients had a history of allergic rhinitis, 62 had food allergies, and 52 patients had asthma. These histories of allergic or atopic conditions were more frequently discovered in AD patients (P<0.001). Based on the SCORAD score, AD patients were divided into three grades: 27 were mild grade, 42 were moderate, and 37 were severe grade. WBC, lymphocytes, monocytes, neutrophils, CRP, TNF-α, IL-1β, IL-6, IL-8, IL-22, and Immunoglobulin E (IgE) were also more obviously elevated in AD cases than in controls (P<0.001) (Tab. 1).

Table 1. Clinical characteristics of the atopic dermatitis patients

Characteristics	AD n=106 (%)	Controls n=137 (%)	*P*
Age	30.15±6.52	29.23±6.50	0.273
Gender			0.959
Male	60 (56.60)	78 (56.93)	
Female	46 (43.40)	59 (43.07)	
History of allergic/atopic conditions			
Allergic rhinitis	66 (62.26)	45 (32.85)	<0.001
Food allergy	62 (58.49)	27 (19.71)	<0.001
Asthma	52 (49.06)	18 (13.14)	<0.001
Severity			
Mild	27 (25.47)		
Moderate	42 (39.62)		
Severe	37 (34.91)		
WBC (10^9/L)	6.95 (6.25, 7.60)	9.30 (6.88, 11.60)	<0.001
Lymphocytes (10^9/L)	2.50 (1.60, 3.13)	5.30 (4.00, 6.80)	<0.001
Monocytes (10^9/L)	0.45 (0.28, 0.61)	0.67 (0.38, 0.89)	<0.001
Neutrophils (10^9/L)	4.20 (3.20, 5.10)	3.30 (1.90, 4.50)	<0.001
CRP (mg/L)	11.92 (10.08, 13.75)	6.00 (3.00, 8.00)	<0.001
TNF-α (pg/mL)	8.73±2.62	6.97±3.17	<0.001
IL-1β (pg/mL)	1.02 (0.67, 1.49)	0.60 (0.41, 0.80)	<0.001
IL-6 (pg/mL)	5.03 (3.84, 6.38)	2.37 (1.80, 2.79)	<0.001
IL-8 (pg/mL)	23.99 ±10.54	18.32±9.40	<0.001
IL-22 (pg/mL)	1329.35 (1143.40, 1567.83)	529.20 (412.89, 632.16)	<0.001
IgE (IU/ml)	119.00 (74.50, 164.50)	195.89 (124.41, 255.12)	<0.001

Notes: WBC – white blood cell count; SCORAD – Scoring Atopic Dermatitis; CRP – C-reactive protein; TNF-α, tumour necrosis factor-α; IL – interleukin; IgE – Immunoglobulin E.
Data are presented as mean±SD for normally distributed variables and as median (IQR) for non-normally distributed variables, as determined by the Shapiro-Wilk test

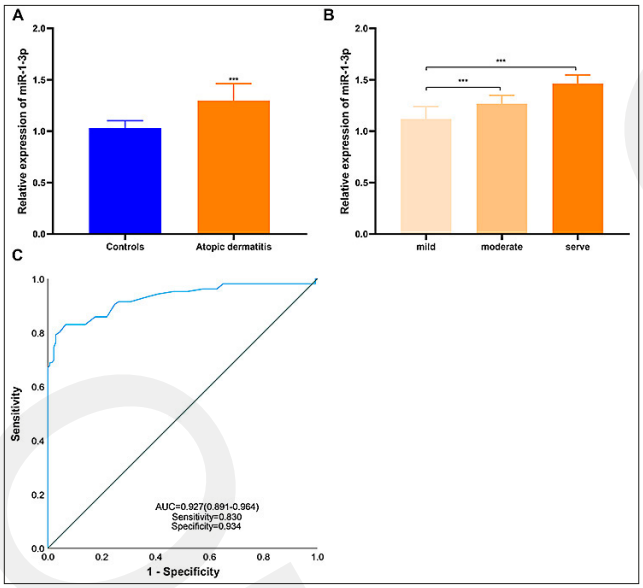

Figure 1. Relative expression of miR-1-3p. (A) miR-1-3p was elevated in AD patients. (B) Difference of miR-1-3p in different AD severity. (C) Diagnostic value of miR-1-3p for AD patients.
***, *P*<0.001

miR-1-3p was elevated in AD patients. qRT-PCR found miR-1-3p overexpression in AD patients than in healthy controls (1.28 [1.14, 1.39] vs. 1.00 [0.93, 1.06]) (Fig. 1A). Additionally, the relative miR-1-3p level was increased with the degree of AD severity; it was significantly higher in severe grade (P<0.001) (Fig. 1B).

Diagnostic efficacy of miR-1-3p was assessed using ROC curve, which revealed an AUC of 0.927 (0.891–0.964). At the threshold of 1.105, the sensitivity and specificity were 0.830 and 0.934 (Fig. 1C). This demonstrated that high miR-1-3p could diagnose AD patients from healthy controls.

miR-1-3p correlated with inflammatory factors in AD patients. Slight correlation presented between miR-1-3p with WBC (r=-0.343), monocytes (r=-0.273), neutrophils (r=0.278), TNF-α (r=0.208), IL-1β (r=0.322), and IL-8 (r=0.187), respectively (P<0.001) (Tab. 2). Besides, miR-1-3p was negatively related to lymphocytes (r=-0.558), and positively related to CRP (r=0.570), IL-6 (r=0.511), and IL-22 (r=0.596) levels, respectively (P<0.001) (Tab. 2),

Table 2. Correlation analysis between miR-1-3p and inflammatory factors

Inflammatory factors	r	P
WBC	-0.343	<0.001
Lymphocytes	-0.558	<0.001
Monocytes	-0.273	<0.001
Neutrophils	0.278	<0.001
CRP	0.570	<0.001
TNF-α	0.208	0.001
IL-1β	0.322	<0.001
IL-6	0.511	<0.001
IL-8	0.187	0.003
IL-22	0.596	<0.001
IgE	0.465	<0.001

Notes: WBC – White blood cell count; CRP – C-reactive protein; TNF-α – tumour necrosis factor-α; IL – interleukin; IgE – Immunoglobulin E.
Correlation strength was interpreted as: weak (r = 0.1–0.3), moderate (r = 0.3–0.5), strong (r > 0.5)

Effects of miR-1-3p on proliferative activity and inflammation in HaCaT cells. Supernatant miR-1-3p expression level was significantly elevated in miR-1-3p mimic cells and reduced in miR-1-3p inhibitor cells (Fig. 2A). CCK8 results revealed that enhancing miR-1-3p levels hindered the proliferative activity of HaCaT cells, whereas a reduced level of miR-1-3p fostered cell growth (Fig. 2B). In addition, TNF-α (Fig. 2C), IL-1β (Fig. 2D), IL-6 (Fig. 2E), IL-8 (Fig. 2F), and IL-22 (Fig. 2G) concentrations were distinctly increased by upregulated miR-1-3p and suppressed by miR-1-3p repression.

Bioinformatics analysis. Selection of miR-1-3p target genes was conducted using TargetScan, miRWalk, and ENCORI databases. A total of 61 overlap genes were obtained by the Venn diagram (Fig. 3A). KEGG analysis found that there were 12 significant pathways, and Figure 3B presented the top 10 pathways. The enrichment analysis of GO functions examined terms related to biological processes (BP), cellular

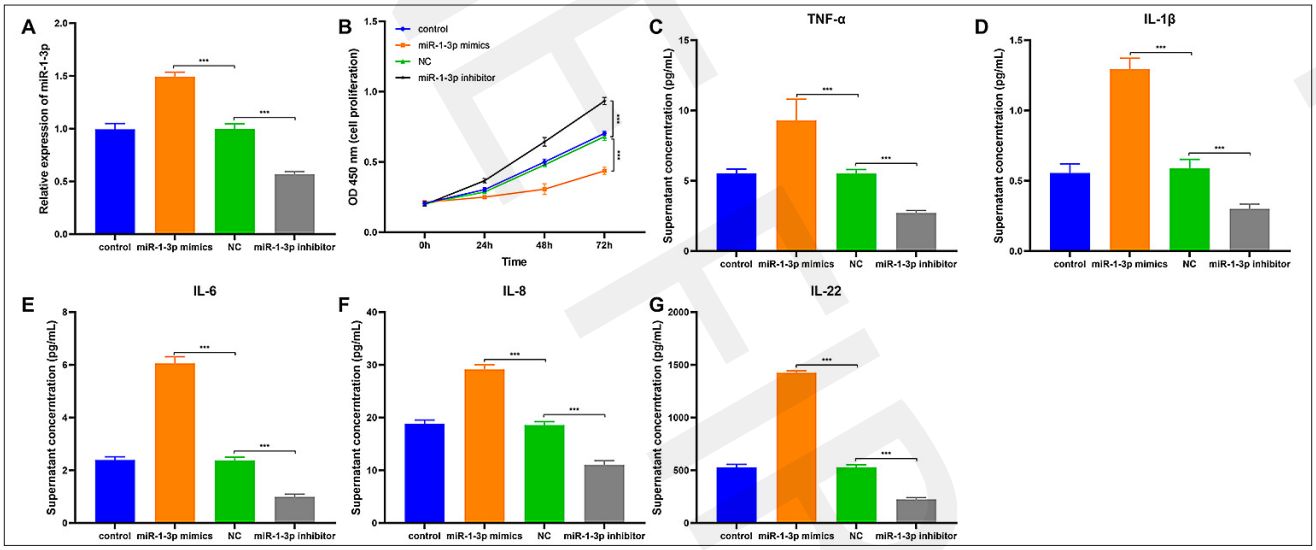

Figure 2. Effects of miR-1-3p on proliferation and inflammation in HaCaT cells. (A) Relative expression of miR-1-3p in transfected cells. (B) Effects of miR-1-3p on proliferation. (C) Influence of miR-1-3p on TNFα. (D) Influence of miR-1-3p on IL1β. (E) Influence of miR-1-3p on IL6. (F) Influence of miR-1-3p on IL8. (G) Influence of miR-1-3p on IL22.
***, *P*<0.001

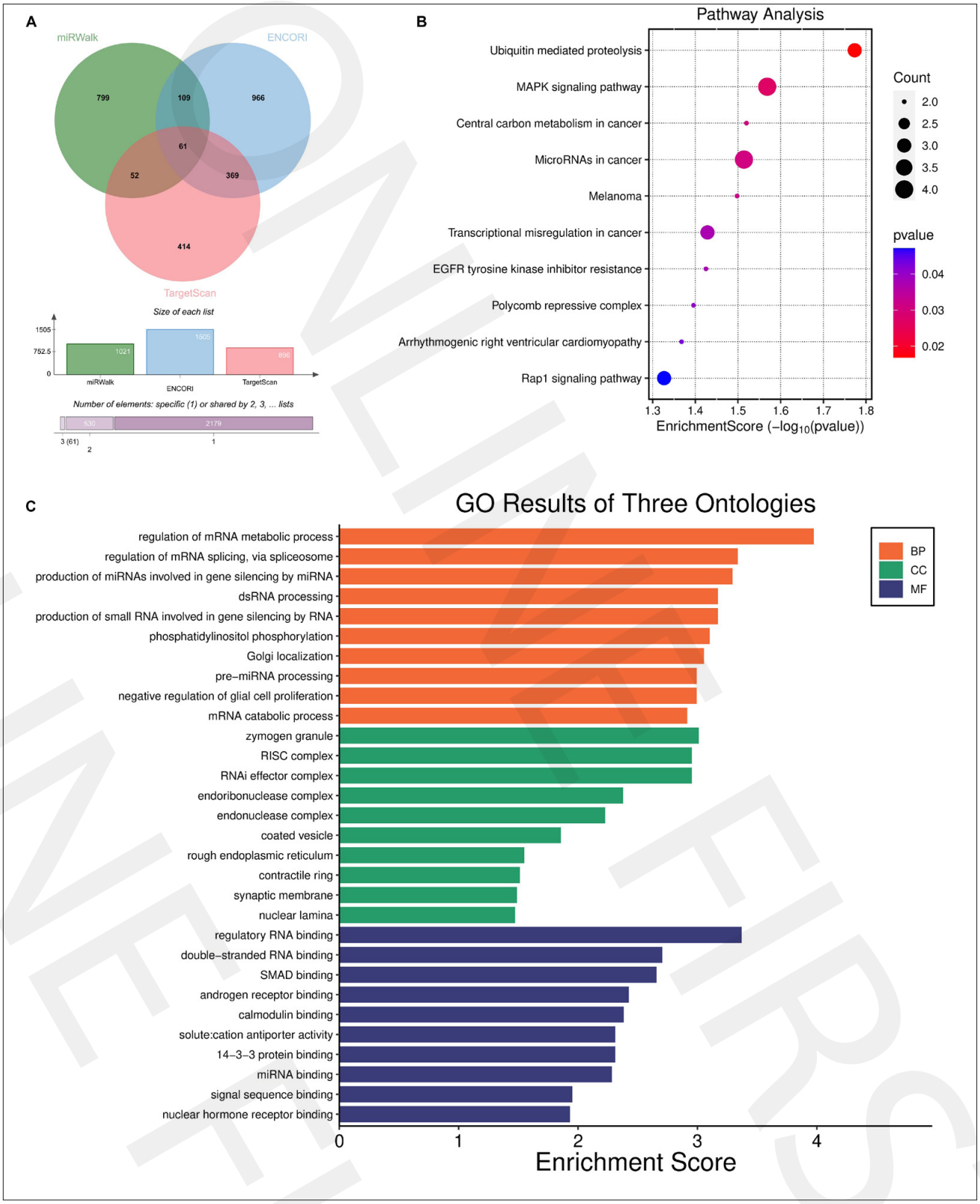

Figure 3. Bioinformatics analysis for the target genes of miR-1-3p. (A) Venn plot for overlap target genes of miR-1-3p. (B) KEGG results for the target gene of miR-1-3p. (C) GO analysis for target gene of miR-1-3p

components (CC), and molecular functions (MF). Ultimately, 231 BP terms, 21 CC terms, and 42 MF terms proved to be significant. The foremost ten terms are shown in Figure 3C.

DISCUSSION

Although many miRNAs have been investigated in the development of AD [18], the the presented study was conducted to elucidate the role of miR-1-3p in AD, and its

relationship with inflammatory responses. Evaluated miR-1-3p in AD cases was found to be significant, correlating with disease severity. Additionally, a link was discovered between miR-1-3p and several inflammatory markers, indicating its possible role in inflammation associated with AD. This study also investigated how miR-1-3p affects both proliferation and inflammatory processes in HaCaT cells, offering valuable insights into its mechanistic function in AD pathogenesis.

Current data indicates increased miR-1-3p in AD patients, which aligns with a prior study reporting miR-1-3p overexpression in AD patients [15]. Li et al. identified elevated miR-1-3p in multiple sclerosis patients [19]. However, it is worth noting that miR-1-3p may not be uniquely associated with AD, as its expression patterns can also reflect broader inflammatory pathways. miR-1-3p has been shown to influence the polarization of macrophages and the activation of signalling pathways like STAT6, which is crucial in immune regulation [13]. This indicates that miR-1-3p upregulation in AD may reflect the overall inflammatory state rather than disease-specific changes. Despite this, the significant value of miR-1-3p in AD diagnosis, as indicated by a high AUC of 0.927, suggests a reliable biomarker for AD. Elevated miR-1-3p in AD patients suggests a possible role in the inflammatory response to the pathogenesis of the disease.

Correlation coefficients of serum miR-1-3p with inflammatory factors provide evidence of its involvement in the inflammatory response. It was found that miR-1-3p was negatively related to lymphocytes and positively related to CRP, IL-6 and IL-22. This indicated that miR-1-3p contributes the regulation of immune cell activity and the release of proinflammatory cytokines. The observed correlations are statistically significant, although mostly moderate, and should therefore be interpreted with caution, as they suggest an association, not a cause-and-effect relationship.

The present results are consistent with previous studies. Li et al. indicated that an enhanced miR-1-3p level could facilitate the differentiation of Th17 cells, and was significantly correlated with CRP and IL-17A [19]. Bajestan et al. indicated that miR-1-3p could regulate IL-6 in patients with inflammatory bowel disease [20]. Previous studies found that IL-22 was upregulated in the skin and correlated with the response to fezakinumab in AD patients [21]. Present findings demonstrated that miR-1-3p exerts a crucial function in inflammation.

The impacts of miR-1-3p on HaCaT cell proliferative activity and inflammation further support its role in AD, as changes in miR-1-3p levels influenced the inflammatory factors. Proliferation of HaCaT cells was restrained by miR-1-3p. The TNF-α, IL-1β, IL-6, and IL-22 levels were facilitated in transfected HaCaT cells. Proliferation of human keloid fibroblasts was restrained by miR-1-3p [22]. Pathway and functional annotation identified that 61 overlapping miR-1-3p target genes are mainly enriched in ubiquitin-mediated proteolysis, MAPK signaling pathway, and so on. These pathways are associated with inflammation progression [23–25], suggesting that miR-1-3p may contribute to AD pathogenesis through these mechanisms.

Limitations of the study. Several limitations of this study should be acknowledged. First, this was a single-centre, cross-sectional study, which limited the generalizability of the findings and precluded causal inferences. In the future, multi-centre, large sample, prospective cohort studies should be performed to certify results obtained and the causality. Second, although bioinformatics analysis of potential target genes was performed, experimental validation of these targets was not conducted, and future studies should include experimental validation of key predicted targets. Third, the use of HaCaT cells, while informative for epidermal responses, may not fully recapitulate the complexity of AD pathophysiology *in vivo*. Fourth, the absence of a control group of patients with psoriasis or other inflammatory skin conditions, limits the diagnostic specificity of miR-1-3p in AD. Finally, the confounding effects of coexisting atopic diseases may have confounded the inflammatory conditions and should be taken into consideration when interpreting the results.

CONCLUSIONS

In summary, miR-1-3p is upregulated in AD and is associated with disease severity and inflammation. These findings suggested a potential involvement of miR-1-3p in AD- related information. Although its role may not be specific to AD, miR-1-3p could serve as a potential therapeutic target pending more comprehensive mechanistic and clinical studies.

Future research should focus on elucidating exact mechanisms by which miR-1-3p contributes to AD pathogenesis. This could involve in-depth studies into miR-1-3p target genes and their roles in immune cell function and skin barrier integrity. Additionally, miR-1-3p should be explored through interventional studies, assessing how modulating its levels affects AD severity and inflammation. It would also be beneficial to conduct multicentre studies to validate the diagnostic capability of miR-1-3p in diverse populations. Other inflammatory skin disease and coexisting atopic diseases also should be considered in future studies to certify the specificity of miR-1-3p in AD.

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