Recent Therapeutic Modalities In Atopic Dermatitis

Essay

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رياله ي العظنيم

سورة العلق آية ١- ٥

إهداء

أهدى عملى ومجهودى هذا إلى روح أبى الحبيب ومعلمى الأول، راجيةً من الله أن يجعله في ميزان حسناته.

هبة الله عبدالمنعم عودة

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LIST OF ABBREVIATIONS

8-MOP : 8- methoxypsoralen

AAAAI : American Academy of Allergy, Asthma and

Immunology

ACAAI : American College of Allergy, Asthma and

Immunology

AD : Atopic dermatitis

APCs : Antigen presenting cells

BMV : Betamethasone valerate

cAMP : Cyclic AMP

CAM : Complementary alternative medicine

CD : Cluster of differentiation

CE : Cornified envelope

Chromosome: The 31st band of the long arm of chromosome

5q31 number5

CLA : Cutaneous lymphocyte-associated antigen

CTACK : Cutaneous T-cell-attracting chemokine

CTCL : Cutaneous T-cell lymphoma

DBPCFC: Double-blind, placebo-controlled oral food

challenge

DC : Dendritic cells

EASI : Eczema area and severity index

ECP : Eosinophil cationic protein

ECP : Extracorporeal photopheresis

EDC : Epidermal differentiation complex

EDN : Eosinophil-derived neurotoxin

EPX : Eosinophil protein X

FceR1 : the β chain of the high affinity IgE receptor

FDA : Food and Drug Adminstration

FKBP : FK binding protein

FLG : filaggrin gene ; filament-aggregating protein

gene

FTU : Fingertip unit

Glu : Glutamine amino acid

GM-CSF : Granulocyte-macrophage colony-stimulating

factor

GRA : Glycyrrhetinic acidHβD : Human beta defensins

ICAM-1 : Intercellular adhesion molecule -1

IDEC : Inflammatory dendritic epidermal cells

IFN-γ : Interferon-γ

IgE : Immunoglobulin E

IL : Interleukin

IL-4R : Interleukin-4 receptor

IP3 : Inositol triphosphateIV : Icthyosis vulgaris

KC : Keratinocytes

LC : Langerhans cells

LEP : Late envelope proteins

LPR : Late phase reaction

LPS : Lipopolysaccarides

LTC 4 : Leukotriene C4

LTRAs : Leukotriene receptor antagonists

LysLysine amino acidMessenger RNA

MBP : Major basic protein

MC : Mast cells

MCP : Monocyte chemotactic protein

mDC : Myeloid Dendritic cells

MDC : Macrophage-derived chemokine
 MHC : Major histocompatibility complex
 MIP : Macrophage inflammatory protein

MMP : Matrix metalloproteinase

Mo : Monocytes

NFATc : Nuclear factors of activated T cells cytoplsmic

NFATp : Nuclear factors of activated T cells

phosphorylated

NK : Natural killer cell

NMF
 Natural moisturizing factor
 PAD
 Peptidyl arginine deaminase
 PAF
 Platelet-activating factor

PBMC: Peripheral blood mononuclear cells

pDC : Plasmacytoid Dendritic cells

PDEIs : Phosphodiestrase enzyme inhibitors

PGE2 : Prostaglandin E2

PPARs : Peroxisome proliferator-activated receptors

PPAR- γ : γ subtype of Peroxisome proliferator-activated

receptors

PUVA : Psoralen + ultraviolet A

RANTES: Regulated upon activation normal T-cell

expressed and secreated

S.aureus: Staphylococcus aureus

SCIT : Subcutaneous specific immunotherapy

SCORAD : Scoring Atopic Dermatitis

SEB : Staphylococcal enterotoxin B

SIT : Specific immunotherapy

SLIT : Sublingual immunotherapy

SPINK5 : serine protease inhibitors, Kazal type 5

SPRs : Small proline-rich proteins

STAT : signal transducer and activator of transcription

T reg cells : T regulatory cells

TARC: Thymus and activation- regulated chemokine

Tc cells : T cytotoxic cells

TCIs : Topical calcineurin inhibitorsTCM : Traditional Chinese medicine

TCR : T cell receptor

tel : telmestine

TGF-β : Transforming growth factor- β

Th : T helper cells

Th0 : T helper type zero cells

TIMs : Topical immunomodulators

TLRs : Toll-like receptors

TNF: Tumor necrosis factor

UV : Ultraviolet

VCAM-1 : Vascular cell adhesion molecule-1

VLA-4 : Very late antigen 4
Vv : Vitis vinifera extract
WAPs : Written action plans

Introduction

Atopic dermatitis (AD) is a chronic inflammatory skin disorder usually presenting with severe pruritus and flaring eczematous lesions in varying localizations depending on the age of the patient. It is considered the most common chronic skin disease of young children (**Boguniewicz**, 2005).

The disease is based on both, a disturbance of the epidermal barrier and increased tendency to immunoglobulin E (IgE) production, partly on a genetic predisposition, upon being triggered by various environmental factors (**Bieber**, **2008**).

Skin lesions are assumed to result from complex interactions between IgE-bearing antigen-presenting cells, T cell activation, mast cell degranulation, keratinocytes, eosinophils, and the combination of immediate and cellular immune responses (Novak and Bieber, 2005), fostered by defects of the physical barrier, such as stratum corneum lipid-protein disturbances resulting, among others, from filaggrin gene mutations (Weidinger et al., 2008).

Treatments for AD are aimed at controlling inflammation with emollients, topical corticosteroids, and topical immune-