PSYCHODERMATOLOGY: STRESS & ITCH

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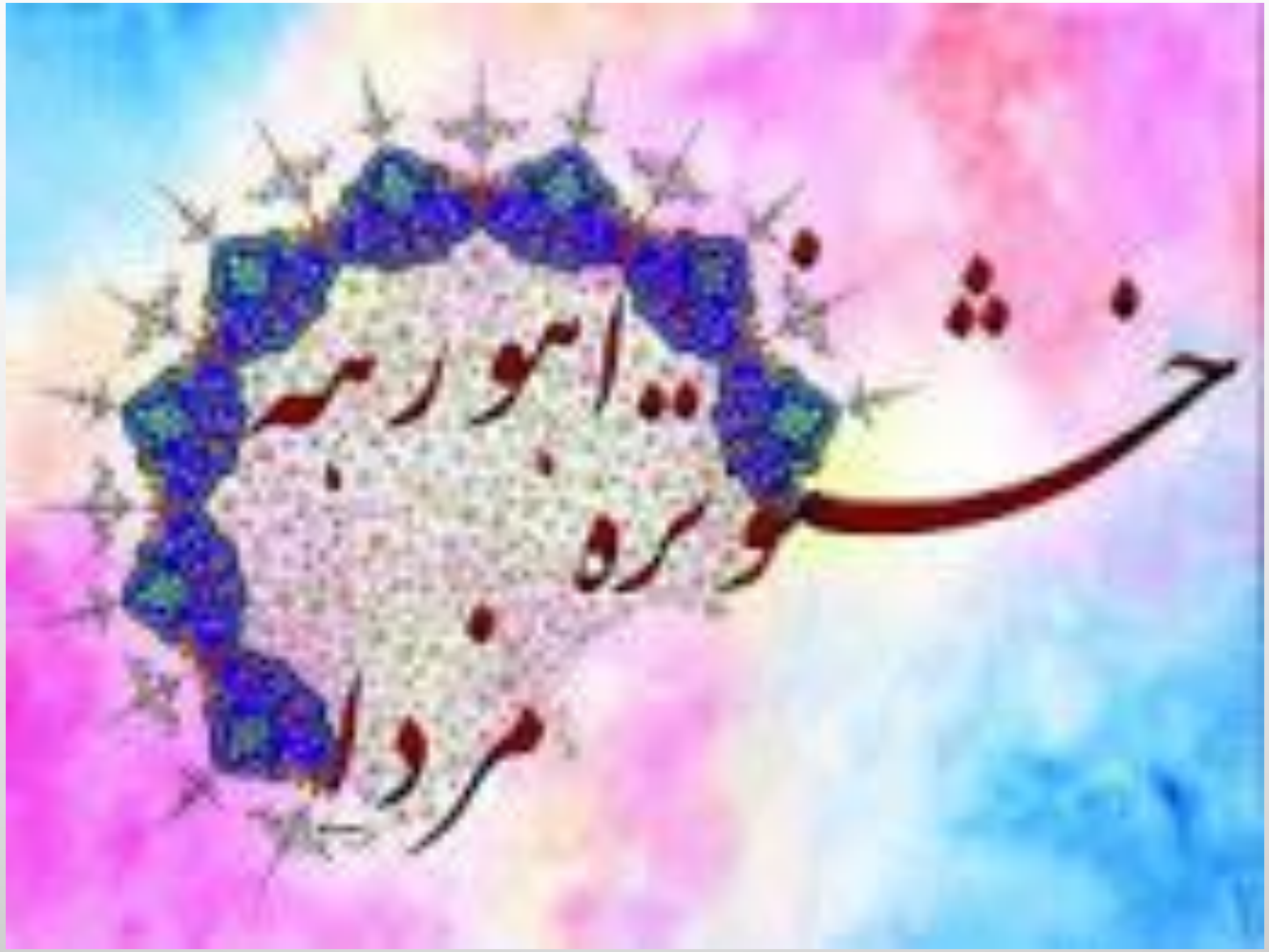
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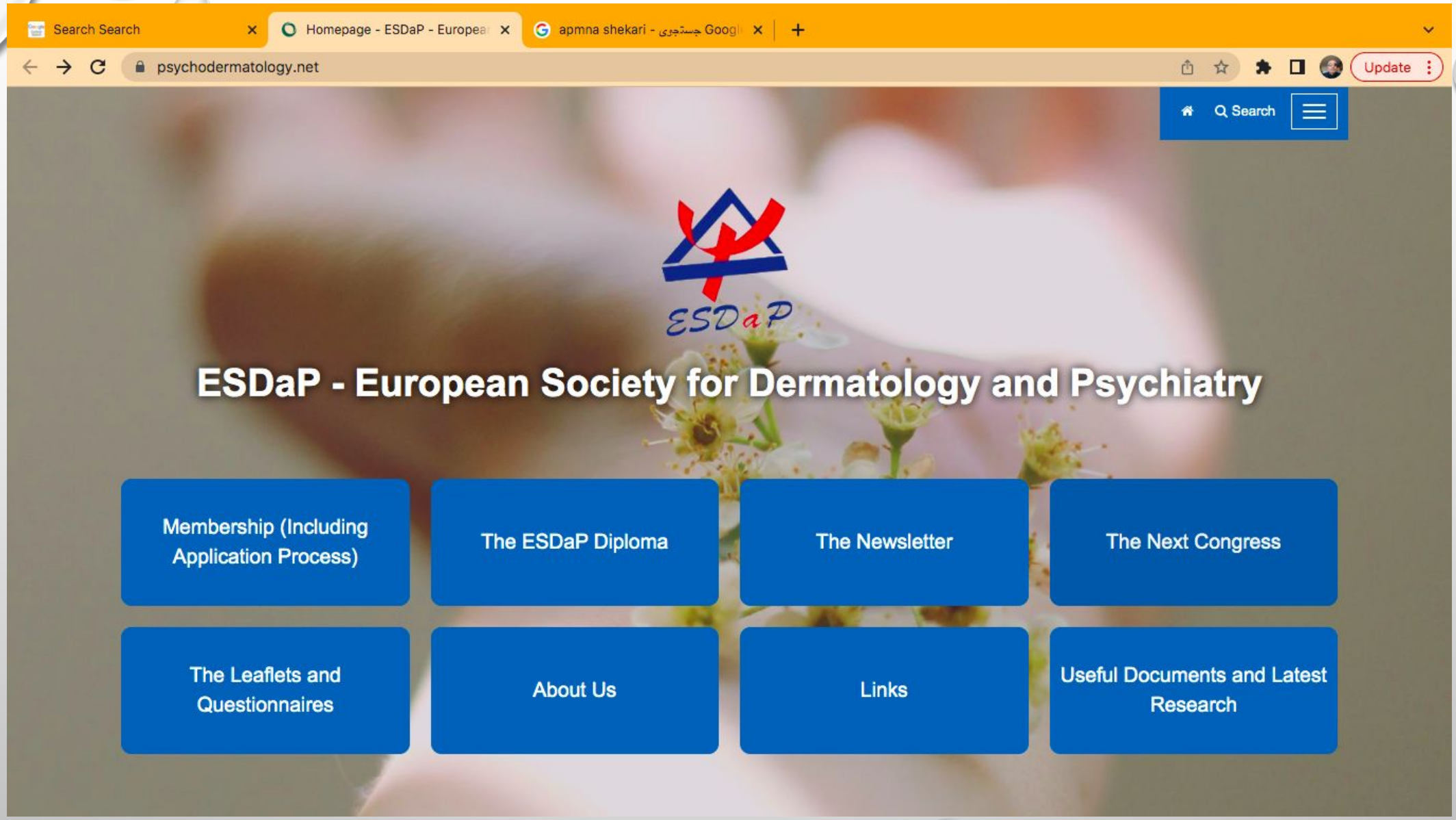
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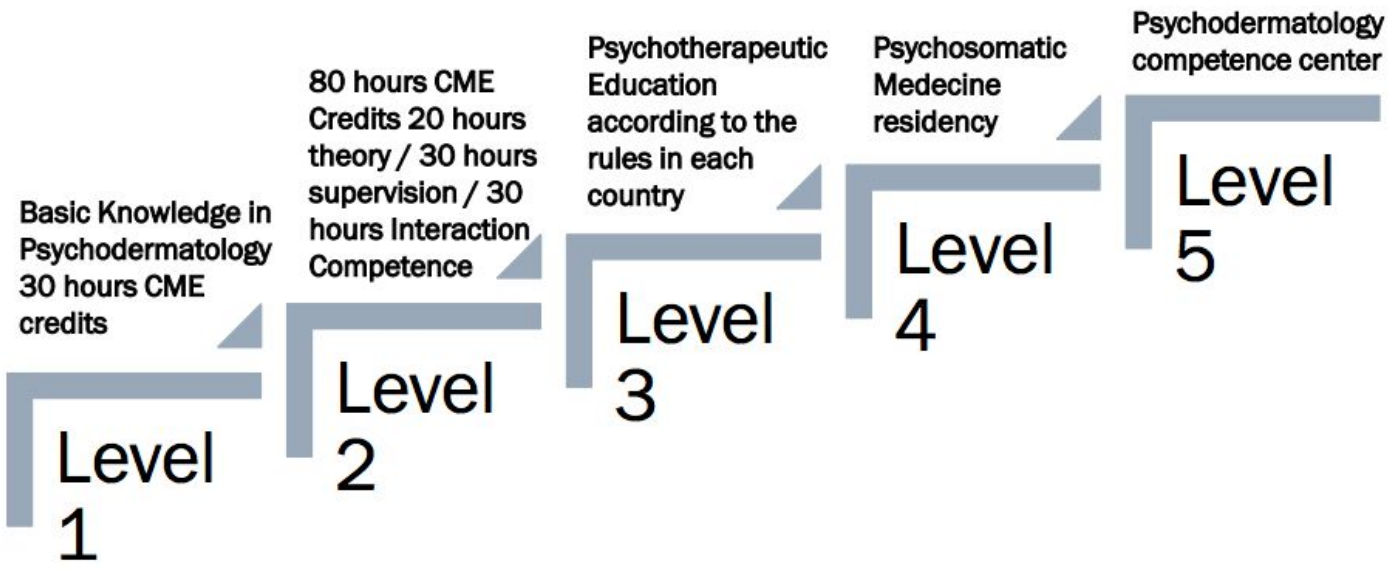

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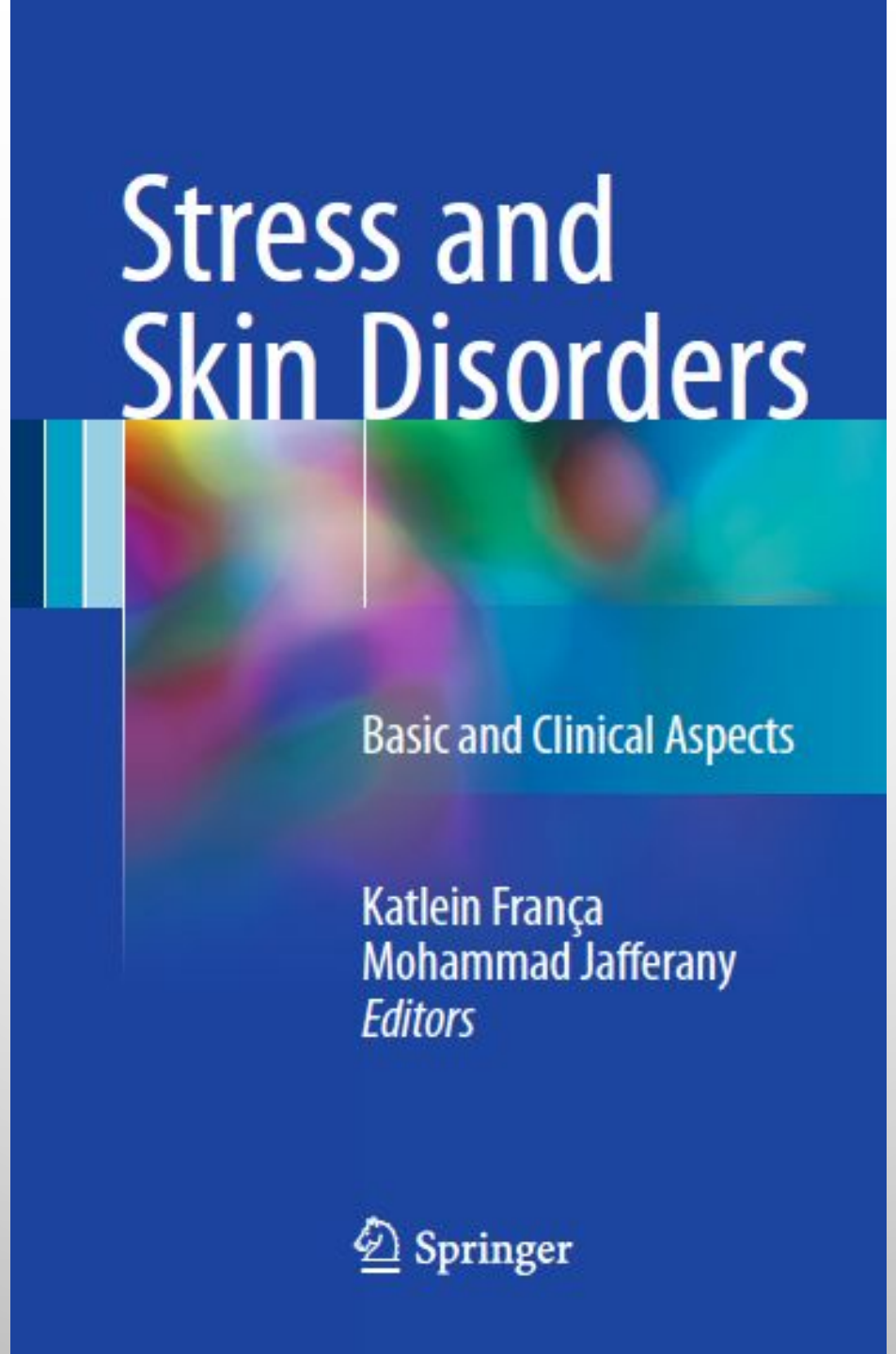
Stress and
Skin Disorders
Basic and Clinical Aspects
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PSYCHONEUROIMMULONGY

Fig. 1.2 A simple representation of psychoneuro-immunological response to a stressor

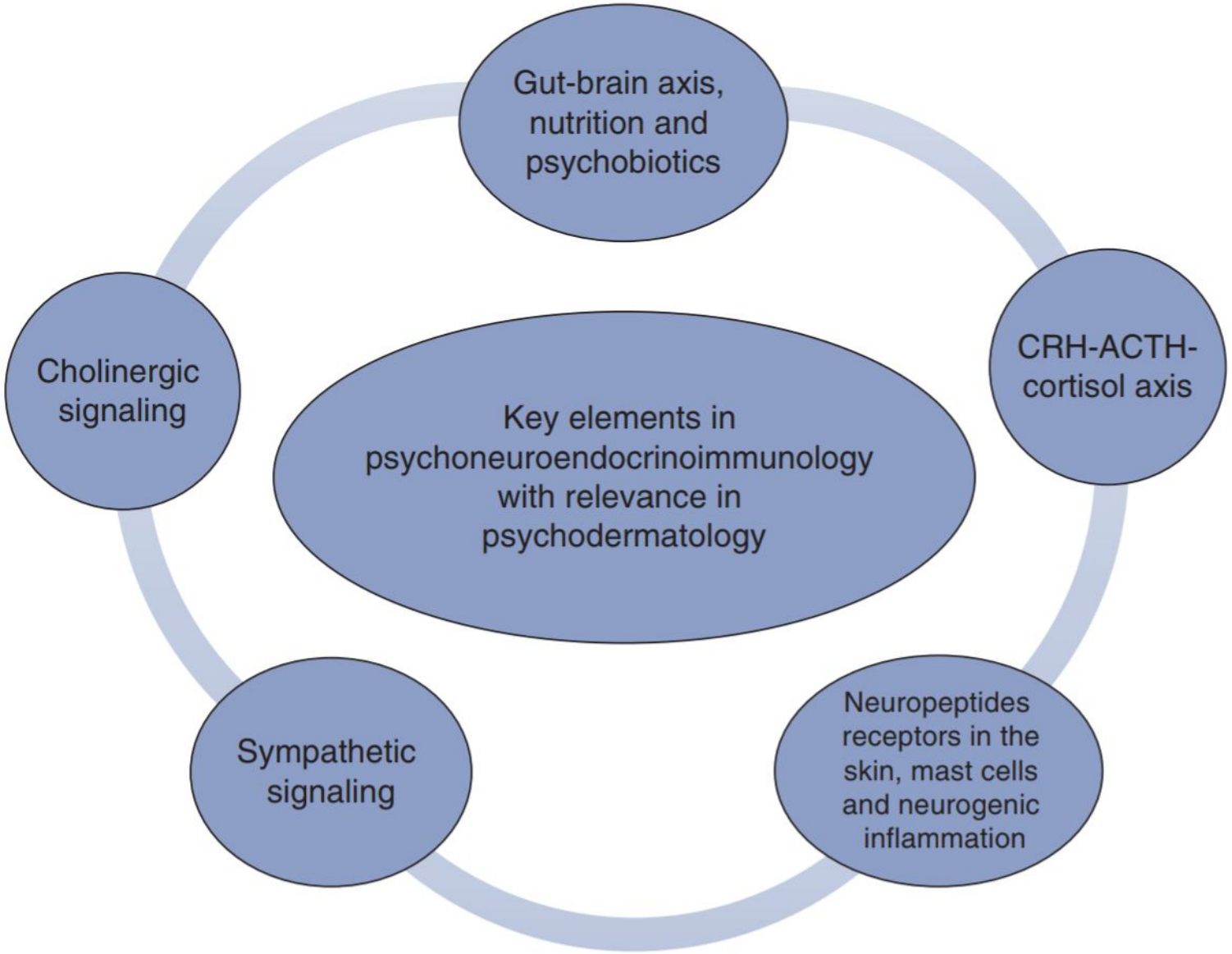

Fig. 2.1 Key elements involved in the connection between the mind and the skin and the physiopathology of the psychophysiological dermatoses, elaborated by Ferreira, Jafferany & Patel

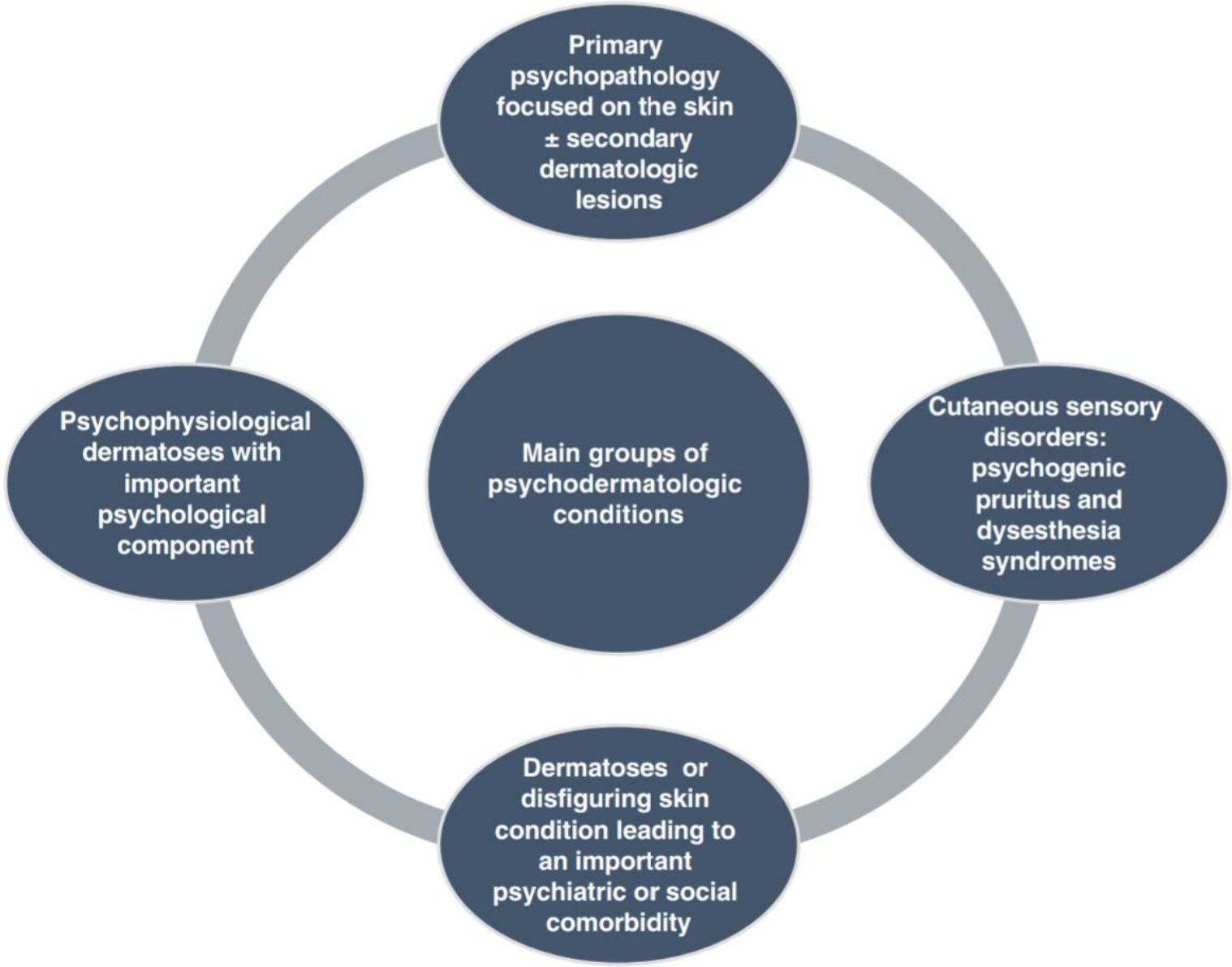

Fig. 5.1 Classification in psychodermatology: four main groups, elaborated by Ferreira, Jafferany & Patel

PSYCHONEUROIMMUNOLOGY OF STRESS AND PSYCHODERMATOLOGIC DISORDERS

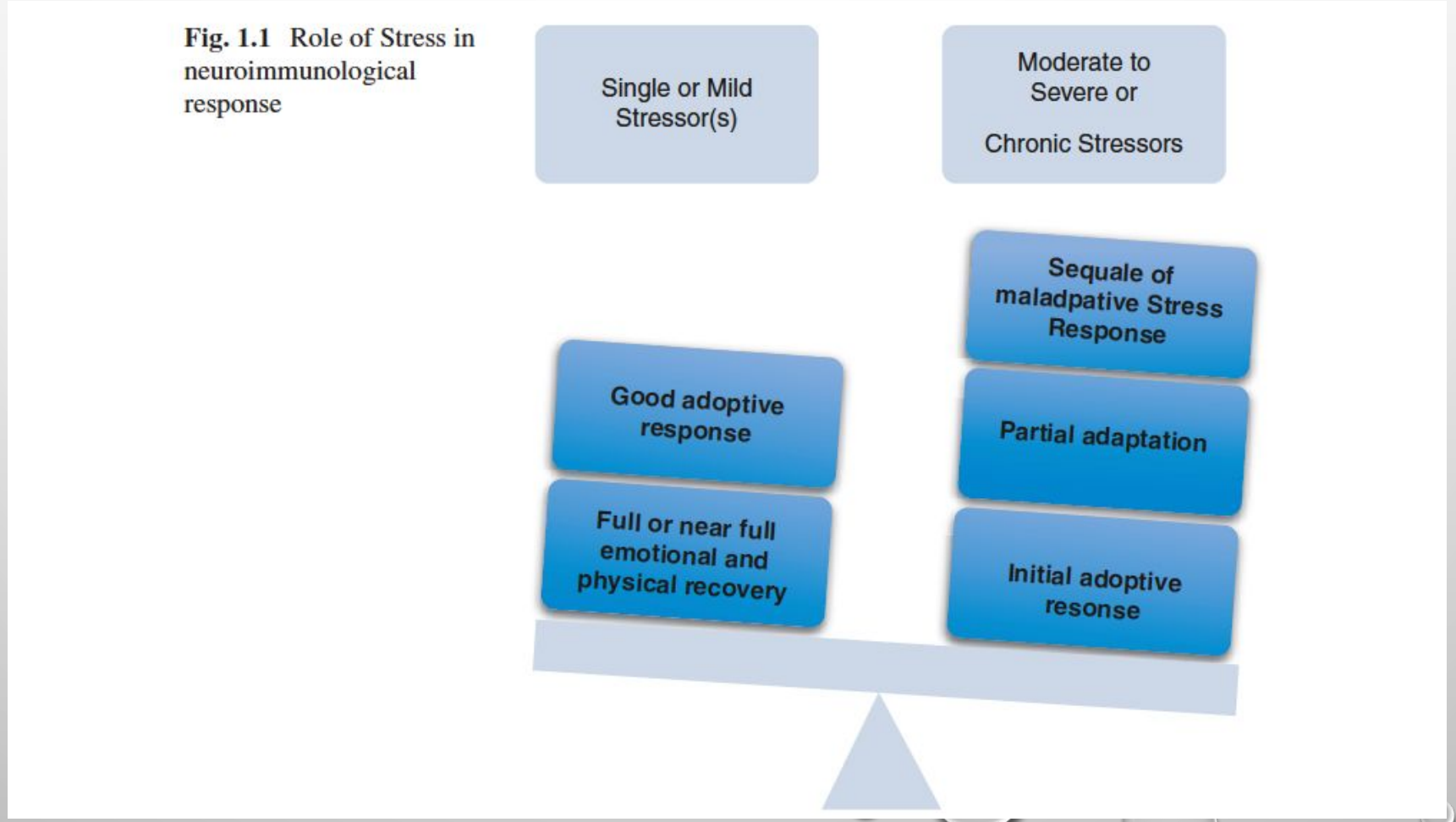

Fig. 1.1 Role of Stress in neuroimmunological response

PSYCHONEUROIMMULONGY

- **SKIN** BEING THE **MOST OUTER, BIGGEST AND MOST INNERVATED** ORGAN CAN SHOW THE BURDEN OF STRESS IN MOST OBVIOUS WAY.
- **SKIN** AND **BRAIN** BOTH SHARE AN EMBRYOLOGICAL ORIGIN FROM THE SINGLE LAYER OF GERMINAL CELLS; THE **ECTODERM**.
- A SPECIALIZED POPULATION OF MULTI-POTENT CELLS KNOWN AS **NEURAL CREST** CELLS EMERGE FROM THE JUNCTION OF NEURAL AND SURFACE ECTODERM; THESE CELLS CAN DEVELOP IN DIFFERENT TYPES OF CELLS LIKE **EPIDERMIS, SYMPATHETIC NERVOUS SYSTEM, PERIPHERAL SENSORY NEURONS, AND MELANOCYTES**.

NERVOUS SYSTEM RESPONSE TO STRESS

- CENTRAL NERVOUS SYSTEM (**CNS**) MODULATES THE IMMUNE RESPONSE TO STRESS VIA THREE DISTINCT MECHANISMS; ACTIVATION OF HYPOTHALAMIC-PITUITARY-ADRENAL AXIS (**HPA** AXIS) AND AUTONOMIC NERVOUS SYSTEM (**ANS**) AND MODULATION OF **MICROGLIA** ON LOCAL LEVEL.
- INITIATION OF BOTH **HPA & ANS** ALSO LEADS TO PRODUCTION OF BIOLOGICALLY ACTIVE **MOLECULES** THAT IN TURN CAN **INTERACT WITH IMMUNE CELLS** DIRECTLY AND CAN FURTHER MODULATE THE STRESS IMMUNE RESPONSE.

NERVOUS SYSTEM RESPONSE TO STRESS

- **ANS** ACTIVATION STARTS THE RELEASES OF **NOREPINEPHRINE** FROM THE ADRENAL CORTEX.
- MOST OF THE ORGANS E.G. **LYMPHOID** ARE INNERVATED WITH **NORADRENERGIC** POSTGANGLIONIC NERVE FIBERS.
- THE SYMPATHETIC INNERVATIONS GENERALLY FOLLOW A SIMILAR PATTERN IN DIFFERENT TISSUES.
- THESE NERVE FIBERS MAKES **NEUROEFFECTOR JUNCTIONS** WITH LYMPHOID CELLS LIKE **MACROPHAGES** TO EXERT EFFECT ON IMMUNE SYSTEMS.
- THE NORADRENERGIC INNERVATED AREAS WITHIN THE **LYMPHOID CELLS** ARE ALSO RICH WITH **NEUROMODULATORY NEUROPEPTIDES** LIKE SOMATOSTATIN, SUBSTANCE P, NEUROPEPTIDE Y, CALCITONIN GENE-RELATED PEPTIDE, OPIATE PEPTIDES, AND VASOACTIVE INTESTINAL PEPTIDE.

NERVOUS SYSTEM RESPONSE TO STRESS

- **NOREPINEPHRINE** HAS SHOWN TO MODULATE IMMUNE RESPONSE BY MODULATING **THYMOCYTE MITOGENESIS, LYMPHOCYTE PROLIFERATION** IN SOME LYMPH NODES, **CELL EXPRESSION OF ANTIGENS, ANTIBODY RESPONSE, DETERS COMPLEMENT ACTIVATION, AND INHIBITS MACROPHAGES MEDIATED LYSIS OF CERTAIN CANCEROUS OR INFECTIOUS CELLS.**
- **LYMPHOID** TISSUES HAVE **CATECHOLAMINE** AND VARIOUS NEUROPEPTIDE-SPECIFIC **RECEPTORS.** CATECHOLAMINES AND OTHER NEUROTRANSMITTERS RELEASED FROM NERVE FIBERS CAN ACTIVATE THESE RECEPTORS AND CAN MODULATE THE IMMUNE RESPONSE VIA INTRACELLULAR SIGNALS **INFLUENCING A PARTICULAR CELL LINE PROLIFERATION, ANTIBODY AND CYTOTOXIN PRODUCTION** ETC. THIS CAN LEAD TO **VASODILATION AND ADHESIONS OF LEUKOCYTES** WHICH CAN FURTHER MODULATE LOCAL INFLAMMATORY RESPONSE IN RESPONSE TO STRESS.

MICROGLIA RESPONSE TO STRESS

- THE THIRD PATHWAY THE CNS MEDIATED STRESS RESPONSE IS THROUGH MICROGLIA THAT ARE INACTIVE OR RESTING MACROPHAGES FOUND ALL OVER THE BRAIN AND SPINAL CORD.
- IN RESPONSE TO A STRESS STIMULI, THESE RESTING MICROGLIA CAN BECOME ACTIVE. **ACTIVATION OF MICROGLIA** CAN LEAS TO **EXPRESSION** OF CELL-SURFACE MARKERS SUCH AS **MHC, COMPLEMENT RECEPTORS, AND CD4 CELLS**.
- MICROGLIA THEN MORPHOLOGICALLY CHANGE TO BECOME ACTIVE PHAGOCYTES.
- THESE MICROGLIA/MACROPHAGES ARE WEAK PHAGOCYTE WHEN COMPARED TO THE PERIPHERAL MACROPHAGES. HOWEVER, WHEN OVER ACTIVATED THEY **RELEASE CERTAIN PRO-INFLAMMATORY CYTOKINES SUCH AS PLATELET- ACTIVATING FACTOR, REACTIVE OXYGEN MOLECULES, AND NITRIC OXIDE THAT CAN LEAD TO NEURONAL INJURY.**

ENDOCRINE RESPONSE TO STRESS

- HPA AXIS HAVE A CAUSE AND EFFECT RELATIONSHIP WITH MANY SKIN DISEASES. WHEN HPA AXIS IS LOOSES ITS ABILITY TO MAINTAIN BASAL AND STRESS RELATED HOMEOSTASIS IT CAN RESULTS IN DISEASE EXPRESSION ESPECIALLY IN SKIN DISEASES.
- **CRH RECEPTORS** HAS A WIDE DISTRIBUTION OF IN VARIOUS NEURAL CIRCUITS LIKE **LIMBIC** SYSTEM, AND **SYMPATHETIC** AROUSAL SYSTEM BOTH IN THE BRAIN AND SPINAL CORD. ONCE THESE RECEPTORS ARE **STIMULATED** IT LEADS TO A WELL-COORDINATED CHAIN OF EVENTS INCLUDING **PHYSIOLOGIC, BEHAVIORAL** CHANGES LIKE CHANGES IN **APPETITE, AROUSAL, SEXUAL** AND **ACTIVITY** LEVELS.

ENDOCRINE RESPONSE TO STRESS

- **ACTH** EXERTS ITS ACTION BY BINDING WITH MELANOCORTIN RECEPTORS 2 (**MC2**) FOUND IN ALL THREE LAYERS OF ADRENAL CORTEX, AND ACTIVATES DOWNSTREAM ENZYME PATHWAYS IN **STEROIDOGENESIS**.
- THIS IN TURN IS RESPONSIBLE FOR INITIATING THE **NEGATIVE FEEDBACK LOOP** TO PUT A BRAKE TO THE STRESS RESPONSE AT THE LEVEL OF **SUPRAHYPOTHALAMIC** CENTERS, **HYPOTHALAMUS**, AND **PITUITARY** GLAND.
- THIS SELF-REGULATORY STRESS RESPONSE CYCLE PREVENTS ADVERSE CONSEQUENCES OF **PROLONGED** ADAPTIVE CHANGES SUCH AS **CATABOLISM** AND **IMMUNOSUPPRESSION**.
- WHEN STRESS IS **CHRONIC** AND UNRELENTING THE SELF REGULATORY NEGATIVE FEEDBACK DOES NOT OCCUR LEADING TO CONTINUAL **HYPERSECRETION OF CRH**, PERPETUATING A CONSTANT **ACTIVATION** OF **HPA-AXIS**.
- THE CONSTANT EXCITATION OF **HPA-AXIS** RESULT IS A SYNDROMAL STATE CHARACTERIZED BY BEHAVIORAL DISTURBANCES SUCH AS **DEPRESSION**, **ANXIETY** DISORDERS, **EATING** DISORDERS AS WELL AS MANY SYSTEMIC SEQUELAE THAT INCLUDE CENTRAL **OBESITY**, **HYPERTHYROIDISM**, **DIABETES** MELLITUS, **METABOLIC** SYNDROME, **OSTEOPOROSIS**, **ATHEROSCLEROSIS**, **IMMUNOSUPPRESSION**, AND INCREASED **SUSCEPTIBILITY**.

ENDOCRINE RESPONSE TO STRESS

- **LEUKOCYTES** THAT PLAYS THE FIRST LINE DEFENSE IN STRESS RESPONSE HAVE SPECIFIC NEUROENDOCRINE **RECEPTORS, SUCH AS RECEPTORS FOR GH, B -ENDORPHIN, THYROID HORMONE, LHRH, AND SOMATOSTATIN**.
- **DEFICIENCY** OF **GH** CAN LEAD TO **ATTENUATE** PRODUCTION OF **ANTIBODIES,** ACTIVITY OF **NATURAL KILLER CELLS** AS WELL AS **T-CELL LYMPHOCYTES.**
- ON THE OTHER HAND **PROLACTIN** CAN **INHIBIT CELLULAR AND ANTIBODY** RESPONSE PREDISPOSING TO CERTAIN **INFECTIONS**.
- AN **INTERRUPTION** IN THIS **NEUROENDOCRINE** PATHWAY CAN LEAD TO **SUPPRESSION OF CELLULAR RESPONSE FROM SPLEEN AND THYMUS AND ANTIBODY PRODUCTION** AS WELL AS **DIMINISHED NATURAL KILLER CELLS** RESPONSE.

DERMATOLOGIC RESPONSE TO STRESS

- SKIN IS RICHLY INNERVATED AN HAS AN EXTENSIVE IMMUNO-NEURO-ENDOCRINE NETWORK THAT CAN BE COMPROMISED BY STRESS.
- ANY TYPE OF STRESS RESULTS IN WHAT IS CALLED AS ALLOSTATIC OVERLOAD, CAUSING VARIOUS LEVELS OF DYSREGULATION RANGING FROM **INFLAMMATION** TO **IMMUNOSUPPRESSION.**
- SKIN **MAST** CELLS ARE CRUCIAL IN MAINTAINING AN ALLOSTATIC BALANCE AND THEY ARE LABELED AS "**CENTRAL SWITCHBOARD**" OF SKIN-STRESS-RESPONSE.
- SKIN MAST CELLS ARE **ACTIVATED** BY STRESS MEDIATORS SUCH AS **CRH, ACTH, NGF, SP AND STEM-CELL FACTOR WHILE GLUCOCORTICOIDS AND CATECHOLAMINE CAN INHIBIT SKIN MAST CELL ACTIVITY.**

DERMATOLOGIC RESPONSE TO STRESS

- SEVERAL DERMATOLOGICAL CONDITIONS REPRESENT A CLASSIC MODEL OF THE STRESS AND EXPRESSION OF DISEASE PARADIGM E.G. ATOPIC DERMATITIS, PSORIASIS, HAIR DISORDERS, URTICARIA, ANGIOEDEMA, AND SKIN INFECTIONS.
- SKIN **MAST CELLS** HAS SPECIFIC NEUROPEPTIDES RECEPTORS ON THEIR SURFACE MAKING THEM A **CENTRAL PLAYER** IN THE PSYCHO-IMMUNO-NEURO-ENDOCIRNE AXIS.
- THEY ALSO PRODUCE VARIOUS **PRO-INFLAMMATORY** SUBSTANCES LEADING TO THE LOCAL EFFECTS OF INFLAMMATION WITHIN THE SKIN INITIATING THE CLASSIC **ITCH-SCRATCH** CYCLE.

KOO AND LEBWOHL CLASSIFICATION OF PSYCHODERMATOSES

- **PSYCHOPHYSIOLOGICAL** DISORDERS: DERMATOLOGIC DISEASES WITH A WELL ESTABLISHED **ETIOLOGIC ROLE OF PSYCHOLOGICAL STRESS**. THEY INCLUDE PSORIASIS, ALOPECIA AREATA, ATOPIC DERMATITIS, ACNE VULGARIS, AND OTHERS.
- **PRIMARY PSYCHIATRIC** DISORDERS: PSYCHIATRIC DISORDER WITH MANIFESTATION OF DERMATOLOGIC SYMPTOMS LIKE DERMATITIS ARTEFACTA, DELUSIONAL INFESTATION, TRICHOTILLOMANIA, NEUROTIC EXCORIATIONS, AND OTHERS.
- **SECONDARY PSYCHIATRIC** DISORDERS: A DERMATOLOGIC CONDITION DUE TO THE **DISFIGURING** LESIONS LEADING TO THE DEVELOPMENT OF A PSYCHIATRIC CONDITION LIKE CYSTIC ACNE, VITILIGO, ALOPECIA AREATA, ICHTHYOSIS AND OTHERS SKIN CONDITIONS LEADING TO STIGMA AND PSYCHOLOGICAL STRESS.

Table 2.1 Psychosocial stressors and the stress- reactive dermatologic disorders

Psychosocial stressor	Predisposing factors	Precipitating factors	Perpetuating factors
Dermatologic disease – related stress, i.e., stress and daily hassles from impact of skin disorder upon the quality of life. Children and adolescents may experience bullying. Important factor in cosmetically disfiguring disorders		Onset/exacerbation of stress- reactive dermatoses that tend to be cosmetically disfiguring e.g., acne, psoriasis, atopic dermatitis	Stress and hassles from having to live with a chronic and usually cosmetically disfiguring dermatologic condition can be a perpetuating factor
Major stressful life events – e.g., loss of job, marital stress, death of spouse		Onset/exacerbation of a wide range of stress-reactive dermatoses	Unresolved stressors may lead to perpetuation of dermatologic disorder
Traumatic life events i.e., events that overwhelm the patient's coping capacity e.g., history of severe neglect, sexual abuse, trauma of war etc. May affect patient years after the initial event, as patients may get triggered by a person or event that reminds them of the trauma. May be associated with autonomic nervous system (ANS) dysregulation	Autonomic dysregulation and hyperarousal may predispose to exacerbations stress-reactive and self-induced dermatoses	Onset/exacerbation of a wide range of stress-reactive dermatoses, especially disorders associated with autonomic hyperarousal e.g, urticaria. May precipitate self-induced dermatoses. Also onset of other stress-reactive dermatoses e.g., psoriasis	Perpetuation of a wide range of stress-reactive dermatoses, especially disorders associated with autonomic hyperarousal. Factor in chronic idiopathic urticaria and chronic self-induced dermatoses e.g., acne excoriee, dermatitis artefacta

STRESS AND IMMUNE FUNCTION OF THE SKIN

- (I) AN IMMUNE ORGAN AND METABOLICALLY ACTIVE **INTERFACE** BETWEEN THE INDIVIDUAL AND THE OUTSIDE WORLD DURING SLEEP AND WAKEFULNESS,
- (II) AN ORGAN OF **COMMUNICATION** THROUGHOUT THE LIFE SPAN- AT NEUROBIOLOGICAL, PSYCHOLOGICAL AND SOCIAL LEVELS.
- A CRITICAL FUNCTION OF THE EPIDERMIS IS **PERMEABILITY BARRIER HOMEOSTASIS**, AND ACUTE PSYCHOLOGICAL STRESS CAN PREVENT SKIN BARRIER FUNCTION RECOVERY IN HUMANS WHICH CAN LEAD TO EXACERBATIONS OF CONDITIONS LIKE ATOPIC DERMATITIS, PSORIASIS AND CONTACT DERMATITIS.
- **ENHANCED IMMUNO-PROTECTION** (E.G., INCREASED EFFICACY OF IMMUNIZATION AND WOUND HEALING) THAT MAY BE ASSOCIATED WITH ACUTE (TYPICALLY LASTING MINUTES TO HOURS) PSYCHOLOGICAL STRESS, CAN ALSO EXACERBATE IMMUNE-MEDIATED DERMATOLOGIC DISORDERS SUCH AS PSORIASIS AND ATOPIC DERMATITIS.

THE SKIN AS AN ORGAN OF COMMUNICATION

- RIGHT AFTER BIRTH S**KIN-TO-SKIN CONTACT** BETWEEN THE NEONATE AND THE MOTHER IS KNOWN TO HAVE A SIGNIFICANT BENEFICIAL IMPACT ON THE INFANT'S CAPACITY FOR **AUTONOMIC REGULATION AND SOCIALIZATION** IN LATER LIFE.
- THE **PSYCHOSOCIAL DEVELOPMENT** OF AN INFANT WITH DERMATOLOGIC DISEASE MAY BE ADVERSELY AFFECTED IF THE CAREGIVER IS **RELUCTANT TO SUFFICIENTLY TOUCH OR HOLD** THE INFANT. IN LATER LIFE, A COSMETICALLY **DISFIGURING** DERMATOLOGIC DISORDER, AFFECTING THE **'EMOTIONALLY CHARGED'** BODY REGIONS SUCH AS THE GENITAL REGION AND THE **EASILY VISIBLE BODY REGIONS**, ESPECIALLY THE FACE, CAN LEAD TO SIGNIFICANT **STRESS DUE TO FEELINGS OF STIGMATIZATION AND SOCIAL EXCLUSION**.

THE SKIN AS AN ORGAN OF COMMUNICATION

- THE **OVERALL APPEARANCE OF THE SKIN**, EVEN WHEN MINIMALLY FLAWED, CAN HAVE A PROFOUND EFFECT ON THE **BODY IMAGE ESPECIALLY DURING ADOLESCENCE** AND YOUNG ADULTHOOD WHEN THE INDIVIDUAL IS ESPECIALLY VULNERABLE TO PEER DISAPPROVAL AND SOCIAL EXCLUSION INCLUDING BULLYING. THE SKIN, ESPECIALLY FACIAL SKIN, IS ONE OF THE MOST EASILY VISIBLE INDICATORS OF CHRONOLOGICAL AGE.
- **CUTANEOUS BODY IMAGE DISSATISFACTION AND RESULTANT INTERPERSONAL SENSITIVITY AND FEELINGS OF SOCIAL ALIENATION HAVE BEEN ASSOCIATED WITH INCREASED SUICIDE RISK.**

CULTURAL AND ETHNIC FACTORS

- PATIENT'S CULTURAL AND ETHNIC BACKGROUND MAY HAVE AN IMPORTANT EFFECT ON HOW THEIR SKIN DISORDER AFFECTS THEIR QUALITY OF LIFE AND RESULTANT DISEASE-RELATED STRESS. THERE ARE ALSO CULTURAL DIFFERENCES IN THE PHYSIOLOGY OF THE STRATUM CORNEUM BARRIER WHICH PLAYS AN IMPORTANT ROLE IN STRESS REACTIVE DERMATOSES. STUDIES HAVE SHOWN THAT **A LIGHTER SKIN TONE IS PREFERRED BY BOTH INDIVIDUALS OF EUROPEAN CAUCASIAN DESCENT AND CULTURES AND ETHNIC GROUPS WITH A DARKER SKIN COLOR.**
- IN MANY CULTURES THE PREFERENCE FOR FAIR OR LIGHTER COLORED SKIN IS QUITE PERVASIVE, AS **LIGHTER SKIN IS ASSOCIATED WITH SEVERAL PERCEIVED BENEFITS INCLUDING JOB, BEAUTY AND MARRIAGE OPPORTUNITY AND THE PERCEPTION THAT AN INDIVIDUAL'S SKIN IS NOT 'FAIR'** ENOUGH CAN BE A SOURCE OF SOCIAL STIGMATIZATION AND STRESS FOR THE INDIVIDUAL.

A BIOPSYCHOSOCIAL APPROACH

- A BIOPSYCHOSOCIAL APPROACH THAT TAKES INTO CONSIDERATION THE PATIENT'S SOCIAL, PSYCHOLOGICAL/PSYCHIATRIC AND GENERAL MEDICAL STATUS IN ADDITION TO DERMATOLOGIC SYMPTOMS, IS SUGGESTED WHEN ASSESSING THE ROLE OF STRESS IN SKIN DISEASE. THE PATIENT SHOULD BE ASSESSED WITHIN THE CONTEXT OF THEIR DEVELOPMENTAL STAGE AND CULTURAL BACKGROUND. **A COSMETICALLY DISFIGURING CONDITION IN AN ADOLESCENT PATIENT MAY BE A LOT MORE STRESSFUL FOR THE PATIENT, AND THE PERCEIVED STRESS MAY BE GROSSLY OUT OF PROPORTION TO THE DERMATOLOGIC SEVERITY OF THE SKIN DISORDER**. THE PATIENT SHOULD BE **SCREENED** FOR **MEDICAL** (E.G., METABOLIC SYNDROME) AND **PSYCHIATRIC** (E.G., BODY DYSMORPHIC DISORDER, MAJOR DEPRESSIVE DISORDER, POSTTRAUMATIC STRESS DISORDER) INCLUDING SUBSTANCE USE DISORDERS, THAT ARE LIKELY TO ENHANCE '**ALLOSTATIC LOAD**' AND DISEASE-RELATED STRESS. IT IS IMPORTANT TO RECOGNIZE THAT **SLEEP AND CIRCADIAN RHYTHM DISRUPTION PLAY AN IMPORTANT MEDIATING ROLE IN STRESS ASSOCIATED FLARE-UPS OF SKIN DISORDERS**; THEY CAN ALSO **EXACERBATE** OTHER **COMORBIDITIES** SUCH AS METABOLIC SYNDROME AND MAJOR DEPRESSION.

FACTORS THAT CAN MODERATE THE RELATION BETWEEN STRESS AND SKIN DISEASE

- 1. **DEMOGRAPHIC**
- **AGE** – DEVELOPMENTAL STAGE OF PATIENT E.G., AN ADOLESCENT PATIENT'S CONCERN ABOUT THE EFFECT OF A COSMETICALLY DISFIGURING DISORDER UPON THEIR APPEARANCE CAN BE GROSSLY OUT OF PROPORTION TO THE CLINICAL SEVERITY OF THE DISORDER
- **CULTURE AND ETHNICITY** – INDIVIDUAL FACTORS E.G., PERCEIVED SOCIAL BENEFITS OF LIGHTER SKIN IN MANY CULTURES
- **GENDER** – MEN AND WOMEN USUALLY EQUALLY AFFECTED; EARLIER LITERATURE REPORTED GREATER IMPACT ON QUALITY OF LIFE OF WOMEN VERSUS MEN
- **SOCIOECONOMIC FACTORS** – HOMELESSNESS HAS BEEN ASSOCIATED WITH INCREASED DERMATOLOGIC MORBIDITY; PROPER NUTRITION

2. General physical

Sleep – sleep restriction, and sleep and circadian rhythm disruption (e.g., related to rotating shift-work) can lead to a heightened pro-inflammatory state and autonomic dysregulation which can exacerbate many inflammatory skin diseases and decrease pruritus threshold

Body mass index – obesity is associated with higher risk of a range of skin disorders some of which e.g., psoriasis are known to be reactive to stress

Lesions in genital and other 'emotionally charged' regions – lesions in such regions know to be associated with greater stress and greater impact on quality of life; patients may not volunteer information about lesions in these regions for fear of stigmatization

Other symptoms of skin disorder – ask patient 'What bothers you the most about your skin condition?' Patients may be most stressed by symptoms that are not considered to be important from a clinical dermatologic perspective and hence may get over-looked

Physical skin-to-skin contact – in the case of infants and children, ask parents/caregivers if they are reluctant to touch or hold their child because of the skin disorder e.g., for fear of adversely affecting the disorder. Counsel parents/caregivers regarding the importance of skin-to-skin contact and nurturance of the child by touch

3. Psychiatric factors

Suicide risk – the psychosocial impact of disorders such as acne and psoriasis has been associated with increased suicide risk

Substance use – tobacco smoking, alcohol use, other substance use disorders

Psychiatric comorbidity – may be present in up to one-third of dermatology patients, and can affect the patient's perception of stress and ability to manage stress- comorbidities include major depressive disorder, obsessive-compulsive and related disorders, social anxiety disorder, posttraumatic stress disorder, body dysmorphic disorder and dissociative disorders

4. Medical comorbidities

Obesity – obesity and metabolic syndrome is being increasingly recognized as a factor in inflammatory dermatoses e.g., psoriasis and atopic dermatitis

Other conditions – depending upon the primary dermatologic disorder, assess patients for other comorbidities that may be contributing the stress reactivity of the skin disorder