Istanbul Master Class
Prof Sibel Alper- President
November 2014

Torello Lotti
Professor and Chairman of Dermatology and Venereology
University of Rome “Guglielmo Marconi“, Rome, Italy
www.torellolotti.it
DISCLOSURE OF RELEVANT RELATIONSHIPS WITH INDUSTRY

- President World Health Academy Publishing House, Zurich, CH
- Editor, Dermatologic Therapy, Wiley-Blackwell
- Chief Medical Officer, BIOSKIN EVOLUTION®
- Consultant, EVLaser
- Consultant, GLG, USA
- Consultant, Advance Medical, USA
- Scientific Director, Dolce Aqua®, Italy
- Consultant, CLINUVEL, Australia
- Chief Medical Officer, Applied Biology, Inc, Irvine, CA, USA
- Executive, Vitiligo Research Foundation, USA
- Editor in Chief, Journal of Pigmentary Disorders, 2014
Vitiligo: Systemic disease

- Melanocytes Biology
- Systemic investigations
- Stop progression – spreading
- Systemic treatments
- Support patients
- Educate patients and Scientific Community
Melanocytes precursors, known as melanoblasts, are formed in the neural crest: in the 11th week of fetal life, they migrate to various sites, where they proliferate and then differentiate into mature melanocyte.

Melanocytes reside in:

- skin (epidermis and hair follicle)
- inner ear
- eye (choroid and retina)
- brain and leptomeninges
Locations and functions of melanocytes

**EYE**
- **Choroid**: Constitutive eye pigmentation, protection against UV
- **Retinal pigment epithelium**: vision, metabolism of rod outer segments and retinoids

**EAR**
- **Inner ear**: balance
- **Cochlea**: hearing

**HAIR FOLLICLE**
- Melanocyte stem cell reservoir for skin. Hair pigmentation, Removal of toxic byproducts

**BRAIN**
- Neuroendocrine function and detoxification

**HEART**
- unknown

**ADIPOSE TISSUE**
- Anti-inflammation, reduction/binding of ROS

**EPIDERMIS**
- Constitutive skin pigmentation.
- Responses to and protection against the environment (primarily UV)
Vitiligo: Systemic approach - Diagnosis

Vitiligo as a systemic disease.
Lotti T\textsuperscript{1}, D'Erme AM\textsuperscript{2}.

Author information

Abstract
Vitiligo is an acquired depigmentary skin disorder of unknown etiology. Vitiligo is not only a disease of melanocytes of the skin. Human melanocytes are derived from the neural crest and are located on various parts of the body. The involvement of skin melanocytes is the most visible one, but a systemic involvement of melanocytes can be observed. Some types of vitiligo (nonsegmental vitiligo) may also be associated with various diseases, mainly with autoimmune pathogenesis. Vitiligo represents a spectrum of many different disorders with different etiologies and pathogeneses, causing a common phenotype: the loss of melanocytes and/or their products. This phenotype is always consistent with a systemic involvement.

Copyright © 2014 Elsevier Inc. All rights reserved.

PMD: 24767192 [PubMed - in process]
Vitiligo: Systemic investigation

• Thyroid
• Auto-immune gastritis
• Pernicious anaemia
• DM Type -1
• Poly glandular syndrome type 2
• Metabolic disorders
• Others
Vitiligo: Systemic therapeutic approach

- Minocycline : 2011- Parsad
- Statins: 2013- Harris
- Immuno-suppresives
- Low dose cytokines
- Others
Neovir® as monotherapy for progressing vitiligo: a clinical study

THERAPEUTIC HOTLINE

Acridone acetic acid, sodium salt, as an agent to stop vitiligo progression: a pilot study

Igor V. Korobko* & Konstantin M. Lomonosov†
*VR Foundation, New York, New York and †Department of Skin and Venereal Diseases, I.M. Sechenov First Moscow State Medical University, Moscow, Russia

Dermatologic Therapy, 2014
Effective and safe systemic treatments for stopping vitiligo progression and inducing repigmentation

- bFGF
- IL-10
- IL-4
- Low dose oral treatment
Prof. Dr. Davinder Parsad, MD, PhD

• Dr John Harris, PhD
ON GOING RESEARCH

Effects of low dose cytokines on co-coluteres of keratinocytes and melanocytes in Vitiligo (University of Florence, G. Marconi University-Rome, Scientific Departement of Guna)

Presently under evaluation the clinical effects of low dose GUNA-FGF, GUNA ANTI IL-1, GUNA IL-10, GUNA IL-4

... my personal long way in Vitiligo treatment
CXCL10 is required for progression and maintenance of vitiligo

Proposed by: John E. Harris, MD, PhD
Assistant Professor of Medicine
Division of Dermatology
University of Massachusetts Medical School
Gene expression is similar in mouse and human vitiligo
Neutralization of CXCL10 reverses vitiligo in mice

• **Classification of vitiligo:** a challenging endeavor.

• Hercogová J, Schwartz RA, Lotti TM.

Clinical Classification

- **Localized**
  - **Focal**: one or more macules in one area but not clearly in a segmental distribution;
  - **Unilateral/segmental**: one or more macules involving a unilateral segment of the body – lesions stop abruptly at the midline;
  - **Mucosal**: mucous membranes alone.

- **Generalized**
  - **Vulgaris**: scattered patches that are widely distributed;
  - **Acrofacialis**: distal extremities and face;
  - **Mixed**: acrofacialis and vulgaris;

- **Universalis**
  - Complete or nearly complete depigmentation.
Working Classification of Vitiligo

- Clinical
- Genetic
- Pathobiological
- Epidemiological
- Multisystem organ dysfunction based
- Trigger based

**Practical implications**
Working Classification of Vitiligo

• **Clinical**
  • Localized (focal, segmental, mucosal)
  • Generalized (vulgaris, acrofacialis, mixed)
  • Universalis
  • Special forms (trichrome, quadrichrome)
  • Inflammatory

• **Practical implications**
  • Localized – stable, regressive
  • Generalized – progressive, systemic, autoimmune
  • Universalis – comorbidities (eye, inner ear etc.)
Working Classification of Vitiligo

• **Genetic**
  • Inherited in non-Mendelian, multifactorial and polygenic pattern
  • Monozygotic twins with identical DNA have only 23% concordance in developing vitiligo
  • HLA haplotypes associated with ethnicities and autoimmune diseases
  • PTPN22 nucleotide polymorphism – autoimmune diseases

• **Practical implications**
  • Delineate patients subgroups prone to develop selected comorbidities
  • Personalized treatment
<table>
<thead>
<tr>
<th>Chromosome</th>
<th>Gene</th>
<th>Protein</th>
<th>Function</th>
<th>GV susceptibility variant†</th>
<th>Other autoimmune disease associations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1p36.23</td>
<td>RERE</td>
<td>Atrophin-like protein 1</td>
<td>Regulates apoptosis</td>
<td>282W</td>
<td>Type 1 diabetes, SLE, Graves’ disease, rheumatoid arthritis, Addison’s disease, psoriasis, inflammatory bowel disease</td>
</tr>
<tr>
<td>1p13.2</td>
<td>PTPN22</td>
<td>Lymphoid-specific protein tyrosine phosphatase nonreceptor type 22</td>
<td>Regulates T cell receptor signaling</td>
<td>R620W</td>
<td>Type 1 diabetes, Graves’ disease, Hashimoto’s thyroiditis, inflammatory bowel disease, SLE</td>
</tr>
<tr>
<td>2q33.2</td>
<td>CTLA4*</td>
<td>Cytotoxic T-lymphocyte antigen 4</td>
<td>Inhibits T cells</td>
<td></td>
<td>Celiac disease, rheumatoid arthritis</td>
</tr>
<tr>
<td>3p13</td>
<td>FOXP1</td>
<td>Forkhead box P1</td>
<td>Regulates lymphoid cell development</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3q28</td>
<td>LPP</td>
<td>LIM domain-containing preferred translocation partner in lipoma</td>
<td>Unknown</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5q22.1</td>
<td>TSLP</td>
<td>Thymic stromal lymphoprotein</td>
<td>Regulates T cell and dendritic cell maturation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6p21.3</td>
<td>MHC class I (HLA-A)</td>
<td>Human leukocyte antigen α chain (HLA-A)</td>
<td>Presents peptide antigens</td>
<td>*02</td>
<td>Many</td>
</tr>
<tr>
<td></td>
<td>MHC class II</td>
<td>Unknown</td>
<td>Regulates B cell differentiation, function of dendritic and Th17 cells</td>
<td></td>
<td>Many</td>
</tr>
<tr>
<td></td>
<td>MHC class III</td>
<td>Unknown</td>
<td>Regulates B cell differentiation, function of dendritic and Th17 cells</td>
<td></td>
<td>Inflammatory bowel disease, rheumatoid arthritis, Graves’ disease</td>
</tr>
<tr>
<td>6q27</td>
<td>CCR6</td>
<td>C–C chemokine receptor type 6</td>
<td>Regulates B cell differentiation, function of dendritic and Th17 cells</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10p15.1</td>
<td>IL2RA</td>
<td>Interleukin-2-receptor α chain</td>
<td>Regulates lymphocyte response to bacteria via IL2</td>
<td></td>
<td>Type 1 diabetes, Graves’ disease, multiple sclerosis, rheumatoid arthritis</td>
</tr>
<tr>
<td>11q14.3</td>
<td>TTR</td>
<td>Tyrosinase</td>
<td>Key enzyme of melanin biosynthesis</td>
<td>R402Q</td>
<td></td>
</tr>
<tr>
<td>14q12</td>
<td>GZMB</td>
<td>Granzyme B</td>
<td>Mediates target cell apoptosis by cytotoxic T cells and natural killer cells and activation-induced cell death of effector Th2 cells</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17p13.2</td>
<td>NLRP1</td>
<td>NACHT, LRR, and PYD domains-containing protein 1</td>
<td>Regulates NACHT, LRR, and PYD domains-containing protein 1</td>
<td></td>
<td>Type 1 diabetes, Addison’s disease, celiac disease, systemic sclerosis</td>
</tr>
<tr>
<td>21q22.3</td>
<td>UBA1H3A</td>
<td>Ubiquitin-associated and SH3 domain-containing A</td>
<td>Regulates T cell receptor signaling</td>
<td></td>
<td>Type 1 diabetes</td>
</tr>
<tr>
<td>22q12.1</td>
<td>XBP1</td>
<td>X-box binding protein 1</td>
<td>Regulates expression of MHC class II genes, IL6, B cell and plasma cell differentiation</td>
<td></td>
<td>Crohn’s disease</td>
</tr>
<tr>
<td>22q13.1</td>
<td>CIQNF6</td>
<td>C1q and tumor necrosis factor-related protein 6</td>
<td>Unknown</td>
<td></td>
<td>Type 1 diabetes, rheumatoid arthritis</td>
</tr>
<tr>
<td>Xp11.23</td>
<td>FOXP3</td>
<td>Forkhead box P3</td>
<td>Regulates regulatory T cells</td>
<td></td>
<td>IPEX</td>
</tr>
</tbody>
</table>

Working Classification of Vitiligo

- **Pathobiological**
  - Autoimmune
  - Neurohumoral
  - Autocytotoxic
  - Biochemical
  - Oxydative
  - Melanocytorrhagy
  - Decreased melanocyte lifespan

- **Practical implication**
  - Understand the origin in the single subject
  - Personalized treatment
ETIOPATHOGENESIS

GENETIC PREDISPOSITION
Autoimmune Susceptibility Locus (AIS1)

NEURAL HYPOTHESIS

AUTOIMMUNE HYPOTHESIS

MELANOCYTE DESTRUCTION

AUTOCYTOTOXIC/RADICALIC HYPOTHESIS

ECLECTIC HYPOTHESIS
MELANOCYTorrAGy
SYNERGISTIC THEORY
Working Classification of Vitiligo

• **Epidemiological**
  - Prevalence from 0.3 % to 8.8 %
  - Role of environmental factors
  - Monozygotic twins with identical DNA have only a 23 % concordance in developing vitiligo

• **Practical implication**
  - Discover environmental factors
  - Personalized treatment
The Neuro Immune Cutaneous Endocrine Network relationship between Mind and Skin
The neuro-immuno-cutaneous-endocrine network: relationship between mind and skin

Benedetta Brazzini,* Ilaria Ghersetich,* Jana Hercogova,† & Torello Lotti*

*Department of Dermoscience, University of Florence, Florence, Italy, and
†Department of Dermatology, Charles University, Prague, Czech Republic
Neuropeptides in skin

Torello Lotti, MD, a Giuseppe Hautmann, MD, b and Emiliano Panconesi, MD b
Siena and Florence, Italy

Neuropeptides are a heterogeneous group of more than 50 molecules that play a role in various cutaneous functions and diseases; they act as neuromodulators, neurotransmitters, neurohormones, and hormones. In the skin, neuropeptides are synthesized locally (i.e., in keratinocytes and in endothelial cells) and are transported by nerve fibers or immune cells (i.e., lymphocytes, monocytes, and polymorphonuclear cells). Specific receptors and binding sites for neuropeptides have been described in different cell lines in the skin (keratinocytes, endothelial cells, immune cells, fibroblasts). Many different biologic actions of neuropeptides have been demonstrated. Depletion of cutaneous neuropeptides (i.e., with capsaicin cream) or therapeutic use of neuropeptide agonists and/or antagonists may aid in the treatment of skin diseases. (J AM ACAD DERMATOL 1995;33:482-96.)
Review

Can the brain inhibit inflammation generated in the skin? The lesson of α-melanocyte-stimulating hormone

Torello Lotti, MD, Beatrice Bianchi, PhD, Ilaria Ghersetich, MD, Benedetta Brazzini, MD, and Jana Hercogova, MD
Figure 1 In the brain α-melanocyte stimulating hormone is synthesized predominantly in the pituitary gland. When administered into the cerebral ventriculi (in mice) α-MSH inhibits the cutaneous inflammation induced by application of topical irritants and intradermal injection of cytokines. This action is related to the integrity of the spinal cord descending neurogenic pathways and of β2 receptors in the periphery. α-melanocyte stimulating hormone is also released in the plasma by the pituitary gland and by different cells, including keratinocytes, melanocytes, monocytes, macrophages, endothelial cells, adipocytes, fibroblasts and mast cells. Membrane receptors for α-MSH are present both in the brain and on nearly all the cells that produce and release α-MSH and participate in cutaneous inflammation mainly by reducing and terminating the same flogistic reactions.
Autoimmune markers in vitiligo patients appear correlated with obsession and phobia

S. Moretti,† M. Arunachalam,†, R. Colucci,† S. Pallanti,‡ J.A. Kline,§ S. Berti, † F. Lotti, † T. Lotti†

†Department of Critical Care Medicine and Surgery, Division of Dermatology, University of Florence, Florence, Italy
‡Department of Psychiatry, University of Florence, Florence, Italy
§Department of Emergency Medicine Research, Carolinas Medical Center, Charlotte, NC, USA

**Figure 1** Psychological discomfort factors and social aspects shown in the graph are significantly higher in autoimmune markers vs. negative autoimmune markers vitiligo patients.
Working Classification of Vitiligo

- **Multisystem organ dysfunction**
- Many disorders or syndromes are variably associated
- Most of such cases are discovered at birth or during infancy
- **Practical implications**
- Great help for pediatricians and geneticists
- Discover yet unidentified causes of vitiligo
- Personalized treatment
Vitiligo: Disease Association

More common associations
- Addison disease
- Alopecia areata
- Atopic dermatitis
- Autoimmune thyroid disease
- Chronic urticaria
- Diabetes mellitus
- Halo nevi
- Hypoacusis
- Hypoparathyroidism
- Ichthyosis
- Ocular abnormalities
- Pernicious anemia
- Psoriasis
- Rheumatoid arthritis

Less common associations
- Acrokeratosis paraneoplastica Bazex
- Alezzandrini syndrome
- APECED syndrome
- Asthma
- Ataxia-telangiectasia
- Deafness
- DOPA-responsive dystonia
- Dysgammaglobulinemia
- HIV
- Inflammatory bowel disease
- Kabuki syndrome
- Kaposi sarcoma
- Melanoma
- MELAS syndrome
- Morphea
- Multiple sclerosis
- Myasthenia gravis
- Nonmelanoma skin cancer
- Pemphigus vulgaris
- Sarcoidosis
- Schmidt syndrome
- Systemic lupus erythematosus
- Turner syndrome
- Twenty-nail dystrophy
- Vogt-Koyanagi-Harada syndrome

Working Classification of Vitiligo

- **Trigger based**
- Vitiligo, leukodermas, depigmented patches
- Trichrome, quadrichrome vitiligos
- Inflammatory vitiligo

**Practical implications**
- Leukodermas and special triggers will facilitate understanding and treating the individual patient since childhood
**CHEMICALLY INDUCED LEUKODERMA**

Most potent phenol/catechol derivatives
- Monobenzyl ether of hydroquinone (MBH)
- Hydroquinone
- p-tert-Butylcatechol (PTBC)
- p-tert-Butylphenol (PTBP)
- p-tert-Amylphenol (PTAP)

Additional phenol/catechol derivatives
- Monomethyl ether of hydroquinone (MMH)
- Monoethyl ether of hydroquinone (MEH)
- p-Phenylphenol
- p-Octylphenol
- p-Cresol

Sulfhydryls
- Cysteamine
- Sulfanolic acid
- Cystamine dihydrochloride

Miscellaneous
- Mercurials
- Arsenic
- Cinnamic aldehyde
- PPD
- Corticosteroids
- Chloroquin
- Soymilk and derived protein
  - Thiotepa (inhibits PAR-2)
  - (Miyamoto and Taylor[^9] and Ortonne[^10])

Tretinoin
- Benzoyl peroxide
- Ammoniated mercury
- Azelaic acid
- Fluorouracil
- Brilliant lake red R

---

Working Classification of Vitiligo

• **Molecular**

  Genome-wide linkage and genome-wide association studies of generalized vitiligo have identified multiple loci which contribute both to vitiligo susceptibility and to other autoimmune diseases (PTPN22 nucleotide polymorphism)

  • Most genes encode molecules relevant for vitiligo

• **Practical implications**

  • “Vitilogenic molecules” specific for individuals/subgroups
  • Limited number of molecules, same phenotype
  • Personalized treatment/ common way treatment
Working Classification of Vitiligo

• **Vitiligo:**
  • Spectrum of diseases with different clinical presentations, unknown etiology, fragmented genetic data and pathogenetical hypothesis
  • Our working classification is proposed as a fertile ground to the scientific community
  • The phenotypic manifestation is not a disease!

• **TAKE HOME MESSAGE FOR VITILIGO**
• **Treatments of vitiligo: what's new at the horizon.**

• Lotti TM, Hercogová J, Schwartz RA, Tsampau D, Korobko I, Pietrzak A, Mitrevska NT, Valle Y, Buggiani G.

The Involvement of Smac/DIABLO, p53, NF-kB, and MAPK Pathways in Apoptosis of Keratinocytes from Perilesional Vitiligo Skin: Protective Effects of Curcumin and Capsaicin

Matteo Becatti, Francesco Prignano, Claudia Fiorillo, Leonardo Pescitelli, Paolo Nassi, Torello Lotti, and Niccolò Taddei
Positive effects of the supplementation of antioxidants in cultured cells

- Total Antioxidant Capacity (marker of cellular scavenging activity)
- Mitochondrial membrane depolarization (marker of mitochondrial and cellular integrity)
Vitiligo Road Map.
Convergence Theory

Vitiligo is a systemic disease
Lotti T. D’Erme E. Hercogova J.
Dermatol Clin 2014
Metabolic syndrome in vitiligo.
Pietrzak A, Bartosińska J, Hercogová J, Lotti TM, Chodorowska G.
VITILIGO: NOT ONLY A MELANOCYTIC DISEASE

Ultrastructural and functional alterations of mitochondria in perilesional vitiligo skin
Francesca Prignano a, Leonardo Pescitelli a, Matteo Becatti b, Paola Di Gennaro a, Claudia Fiorillo b, Niccolò Taddei b, Torello Lotti a

aDepartment of Dermatological Sciences, University of Florence, Florence, Italy
bDepartment of Biochemical Sciences, University of Florence, Florence, Italy

Arch Dermatol Res
DOI 10.1007/s00403-010-1109-5

ORIGINAL PAPER

Dendritic cells: ultrastructural and immunophenotypical changes upon nb-UVB in vitiligo skin
Francesca Prignano · F. Ricceri · B. Bianchi · D. Guasti · V. Bonciolini · T. Lotti · N. Pimpinelli
Vitiligo: recent insights and new therapeutic approaches.

Treatment of vitiligo at the Dead Sea: An integrated concept research and practice
SIRT1 regulates MAPK pathways in vitiligo skin: insight into the molecular pathways of cell survival.

Becatti M, Fiorillo C, Barygina V, Cecchi C & Lotti T
Abstract
Vitiligo is an acquired and progressive hypomelanotic disease that manifests as circumscribed depigmented patches on the skin. The aetiology of vitiligo remains unclear, but recent experimental data underline the interactions between melanocytes and other typical skin cells, particularly keratinocytes. Our previous results indicate that keratinocytes from perilesional skin show the features of damaged cells. Sirtuins (silent mating type information regulation 2 homolog) 1, well-known modulators of lifespan in many species, have a role in gene repression, metabolic control, apoptosis and cell survival, DNA repair, development, inflammation, neuroprotection and healthy ageing. In the literature there is no evidence for SIRT1 signalling in vitiligo and its possible involvement in disease progression. Here, biopsies were taken from the perilesional skin of 16 patients suffering from non-segmental vitiligo and SIRT1 signalling was investigated in these cells. For the first time, a new SIRT1/Akt, also known as Protein Kinase B (PKB)/mitogen-activated protein kinase (MAPK) signalling has been revealed in vitiligo. SIRT1 regulates MAPK pathway via Akt-apoptosis signal-regulating kinase-1 and down-regulates pro-apoptotic molecules, leading to decreased oxidative stress and apoptotic cell death in perilesional vitiligo keratinocytes. We therefore propose SIRT1 activation as a novel way of protecting perilesional vitiligo keratinocytes from damage.

© 2013 The Authors. Journal of Cellular and Molecular Medicine published by John Wiley & Sons Ltd and Foundation for Cellular and Molecular Medicine.

KEYWORDS:
MAPK, Akt, SIRT1, oxidative stress, vitiligo
PMID 24410795
[PubMed - as supplied by publisher]
The Sirtuin System: The Holy Grail of Resveratrol?

Mohar DS, Malik S.

Abstract

The oxidative stress theory has been associated with atherosclerosis and has prompted a multitude of studies to evaluate the effects of antioxidants on cardiovascular disease prevention. Resveratrol, a relatively new antioxidant has gained considerable curiosity. This polyphenol stilbene identified in grape skin, is believed to be the main component contributing to the anti-atherosclerotic benefits linked to red wine consumption. It has demonstrated the ability to protect endothelial cells from lipid damage, promote vasodilation via modulation of nitric oxide synthesis, and inhibit platelet aggregation and smooth muscle proliferation. Although the complete mechanism of Resveratrol has yet to be fully elucidated, the Sirtuin system, consisting of 7 highly conserved families of regulator genes, are thought to be instrumental in establishing the various health benefits. In this article we assess the current applications, mechanism, pharmacokinetics, bioavailability, and safety profile of the novel antioxidant Resveratrol and provide an in-depth review of the influence of the Sirtuin system on the Resveratrol mechanism of action. We resolve that while early data on Resveratrol are promising, the anti-oxidative and ultimately, anti-atherosclerotic potential depends on further clarification of the intricate and complex relationship between Resveratrol and the Sirtuin system.
High prevalence of circulating autoantibodies against thyroid hormones in vitiligo and correlation with clinical and historical parameters of patients.

Colucci R¹, Lotti F, Dragoni F, Arunachalam M, Lotti T, Benvenga S, Moretti S.

Abstract

BACKGROUND: Autoantibodies against thyroid hormones (THAbs) directed towards triiodothyronine (T3-Ab) and/or thyroxine (T4-Ab) are very rare in the general population. They are increased in some nonthyroidal autoimmune diseases, where they seem to predict autoimmune thyroid disorders (ATDs). So far, their presence in patients with vitiligo has not been evaluated, but it might have a possible predictive role.

OBJECTIVES: To assess the prevalence of THAbs in a group of vitiligo patients and to correlate their presence with clinical and historical parameters.

METHODS: In total 79 patients with nonsegmental vitiligo and 100 controls were examined. Clinical characteristics of vitiligo and family and personal medical history were evaluated. Antinuclear autoantibodies, thyroid hormones and thyroid autoantibodies were measured. IgM T3-Ab, IgG T3-Ab, IgM T4-Ab and IgG T4-Ab were assayed by a radioimmunoprecipitation technique. Fisher's test, Student's t-test and χ²-test were used for statistical analysis.

RESULTS: Overall 77 of 79 patients (97%) had at least one type of THAb (11 T3-Ab, 10 T4-Ab, 56 both). In the control group, only one person (1%) had THAbs. In patients with vitiligo, T3-Ab were significantly associated with leucotrichia (lgM+lgG, P = 0.033; lgG, P = 0.039; lgM, P = 0.005) and thyroglobulin autoantibodies (lgM+lgG, P = 0.031; lgG, P = 0.058), while the absence of T3-Ab was related to personal history of cancer (lgM+lgG, P = 0.021; lgG, P = 0.039). T4-Ab were significantly associated with vitiligo activity (lgM+lgG, P < 0.001; lgM, P = 0.037) and duration (lgG, P = 0.013).

CONCLUSIONS: The surprisingly high prevalence of THAb in patients with vitiligo and their associations suggest a possible pathogenetic role in the disease and stress the tight link between vitiligo and ATDs. Further evaluation in a larger group of patients and an adequate follow-up are needed to define their potential predictive role.
Future Take Home Message::
Low dose cytokines and systemic neuropeptidergic regulation
Pigmentary Disorders and Vitiligo VRF MasterClass, Amristar, India
November 28 - 30, 2014
President: Prof. Davinder Parsad
2014 News

Istituto di Scienze Dermatologiche
Cattedra di Dermatologia e Venereologia
Dir. Prof. Torello Lotti

Thank you for your attention
Pigmentary Disorders and Vitiligo VRF MasterClass, Istanbul, Turkey, November 21 - 23, 2014
President: Prof. Sibel Alper

Thank You
Prof
Sibel Alper