

# An Epidemiological Study on Trigger Factors and Quality of Life in Psoriatic Patients

Alert Xhaja, Entela Shkodrani, Silvan Frangaj, Loreta Kuneshka, Ermira Vasili

Department of Dermatology, UHC Nene Tereza, Tirane, Albania

Corresponding author: Alert Xhaja, MD. Department of Dermatology, UHC Nene Tereza, Tirane, Albania. Phone: +355 69 206 7146; Email: axhaja@gmail.com:

## ABSTRACT

**Objective:** to evaluate the role of stress, tobacco, drugs, infections, allergies, heredity, alcohol, hormones and skin aggressions as trigger factors and the impact on quality of life in a sample of psoriasis patients. **Methods:** a transversal study performed in 90 patients affected by psoriasis between January and November 2012 at the “Nene Tereza” University Hospital, Tirane, Albania, based on two scored questionnaires. **Results:** more than 70 % of patients reported that stressful events caused a flare-up of their psoriasis ( $p < 0.05$ ). More than 60% of males and 20% of females were smokers ( $p < 0.05$ ). About 20% of our patients were taking one or more of the medications listed in the questionnaire ( $p > 0.05$ ). About 20% of patients reported having had recurrent infections ( $p < 0.05$ ). About 80% of males patients consumed alcohol ( $p < 0.05$ ). More than 40% reported a relative with psoriasis. Statistical comparison of the group that reported skin aggressions with the group that did not revealed a significant difference ( $p < 0.05$ ). Only a few of them reported to have allergies ( $p > 0.05$ ). About 36% of females reported that hormonal changes (puberty and menopause) exacerbated their psoriasis ( $p < 0.05$ ). More than 40% of patients reported that psoriasis seriously affects their quality of life. **Conclusion:** stress, tobacco, infections, heredity, alcohol, hormonal changes and skin aggressions were confirmed as trigger factors for psoriasis in the present sample. Allergies and the investigated drugs seemed not to have any influence in flare-ups. We found that psoriasis had a serious impact in the quality of life in over of 40% of the patients interviewed.

**Key words:** psoriasis, trigger factors, quality of life, Albania.

## 1. INTRODUCTION

Psoriasis is a non-contagious chronic cutaneous inflammatory disease, with a genetic component. It affects 2-3 % of European population. While psoriasis can start at any age, there are two peaks of onset: one at around 20-30 years old subjects, and the other at subjects of over 50 years of age. (1-9) It is a disease without vital prediction, but with a major impact on life quality and self-value.

Epidermal proliferation is its earliest recognized pathogenetic characteristic. DNA synthesis and mitotic activity are dramatically increased in the basal layer. (10-14) The cells divide every 1.5 days and move rapidly to the surface over 3 – 4 days, where they are shed in large amounts as incompletely keratinized scales. A special feature of psoriatic lesions is the migration of neutrophils into the involved epidermis. Intact neutrophils collect in the sub corneal space, forming micro-abscesses. (27-29).

Large numbers of CD4+ and CD8+ T cells are found in the epidermis and upper dermis. They appear to play a key role in the development of the cutaneous manifestations of the disease. (15-20) T cells in psoriasis lesions release Th1 / Th17 mediators, such as IFN- $\gamma$ , IL2, IL17, IL23 and TNF- $\alpha$ . These mediators act on keratinocytes and other cells in the skin, activating them

and inducing the formation of lesions. (21-26). The inheritance of psoriasis is clearly polygenic or multi-factorial. Most likely, multiple psoriasis alleles encoded by several genes are required for the disease to become manifested. (30-34).

Many exogenous or endogenous factors can trigger the eruption of psoriasis. Known exogenous triggers are skin aggression (35), infections (36-38), alcohol and tobacco (39-45), stress (43-46), drugs (lithium, beta-blockers, antimalarials, ACE-inhibitors, NSAIDs). (47-54) Endogenous triggers are hormonal changes (55,56) and allergies (57, 58).

## 2. PATIENTS AND METHODS

The study was conducted in the dermatology department of the “Nene Teresa” University Hospital, Tirane, during the period from January to November 2012. A sample of 90 patients with psoriasis, 45 men and 45 women above 18 years-old were included, either with recurrent disease or their first flare-up. The interviews were carried out using two questionnaires: the first inquiring about potential trigger factors and the second assessing the quality of life. The aim was to identify the importance of trigger factors in the outburst and exacerbation of psoriasis in our sample and the impact of the disease on their quality of life.

Have you had any stressful events that may be connected to a flare-up of your psoriasis?	Yes, very stressful-4 points Yes, quite stressful - 3 points Yes, somewhat stressful - 2 points No, nothing particular happened - 1 points No, I was in a very relaxed period- 0 points
How many cigarettes do you smoke daily?	> 20 cigarettes - 4 points 10 - 20 cigarettes - 3 points 5 -10 cigarettes - 2 points 1 -5 cigarettes - 1 points None - 0 points
Do you take any of these drugs : lithium, beta-blockers, anti-malarial, ACE-inhibitors, NSAIDS ?	4 medications - 4 points 3 medications - 3 points 2 medications - 2 points 1 medications - 1 points none - 0 points
Do you have recurrent infections, especially throat infections?	Yes - 4 points No - 0 points
Alcohol consumption	>2 glasses - 3 points 2 glasses - 2 points 1 glass - 1 points Not a drinker - 0 points
Do you have any relatives with psoriasis?	Mother and father - 7 points Mother or father - 6 points Brother and sister - 5 points Brother or sister - 4 points Gr.father and gr.moth - 3 points Gr.father or gr.mother - 2 points First cousin - 1 points None - 0 points
Have you had a skin aggression recently? (describe)	Yes - 4 points No - 0 points
Do you have any kind of allergies?	Yes - 4 points No - 0 points
Do your hormonal changes affect your psoriasis (in females patients)?	Yes - 4 points No-- 0 points

Table 1. Questionnaire on potential trigger factors

### 3. STATISTICAL ANALYSIS

Statistical analyses were performed using SPSS for Windows. The significance between variables was tested using the chi-squared statistical test ( $\chi^2$  test).

### 4. RESULTS

More than 70 % of patients reported that stressful events cause their psoriasis to flare- up of. Using the  $\chi^2$  test , a significant difference was found between patients who experienced stressful events and those who did not ( $p < 0.05$ ). This confirmed stress as a trigger factor in both males and females of our sample.

More than 60% of males and 20% of females were smokers. Statistical analysis yielded a significant difference between smoking and non-smoking females ( $p < 0.05$ , regardless of the number of cigarettes). Similar results came out comparing smoking and non-smoking males ( $p=0.026$ ). Smoking is a trigger factor for both males and females in our sample.

About 80% of males patients consumed alcohol. Statistical comparison between the groups of drinkers and non-drinkers resulted statistically significant ( $p < 0.05$ ). Alcohol is a trigger factor in male patients of our sample.

About 20% of our patients were taking one or more of the medications listed in the questionnaire. Comparison of the

\* how much did each of the following affect you during the last week? (not at all-0 p; somewhat-1 p; quite a bit-2 p; a great deal-3 p)  
Presence of scales?  
Shedding of scales?  
Itching?  
Joint pain?  
Treatment cost ?  
Time lost with treatment?  
Getting dirty clothes during treatment?

\* how much did each of the following affect you during the last week? (not at all-0 p; somewhat-1 p; quite a bit-2 p; a great deal-3 p)  
Relationships with family members?  
Relationships with friends?  
Daily activities?  
Professional activities?  
Relaxing activities?  
\* last week because of psoriasis you had: (yes-2p; no-0p)  
Feelings of being not attractive?  
Difficulties in sexual activities?  
Tendency to be isolated, avoiding contacts with other people?  
A state of irritability and/or frustration?

\*Does psoriasis constitute your most important health problem?(yes-3p; no-0p)  
\*Do you try to hide your disease in the presence of other people? (yes-3p; no-0p)

#### Interpretation of data:

10-20 p:-dermatosis slightly affects life quality ;  
21-30 p:-dermatosis averagely affects life quality;  
31-40 p:-dermatosis considerably affects life quality;  
41-50 p:-dermatosis deeply affects life quality;

Table 2. CV-50 questionnaire for quality-of-life assessment

group that received drugs (at least one) with the group that did not receive any resulted statistically insignificant ( $p > 0.05$ ).

About 20% of patients reported having had recurrent infections. Comparison of the group reporting recurrent infections with the group not reporting resulted statistically significant ( $p < 0,05$ ). Recurrent infections are trigger factors in the patients of our sample.

Statistical comparison between the groups reporting skin aggressions and not resulted significant ( $p < 0,05$ ). Skin aggressions are a trigger factor for psoriasis in our sample.

Only a few patients reported having allergies. Statistically it resulted not significant ( $p > 0,05$ ).

More than 40% reported to have a relative with psoriasis.

About 36% of females reported the effect of hormonal changes in the exacerbation of psoriasis. Hormonal changes resulted statistically significant in females ( $p < 0,05$ ). They are a trigger factor for psoriasis in female patients of our sample.

In this study more than 40% of patients reported that psoriasis seriously affects their life. The most affected areas are everyday activities, professional activities, relationships with family members and friends. Most of the patients avoid public interaction, dress to hide their condition and feel like outcasts (64%). Psoriasis affects daily interpersonal interactions in four out of ten (38%) interviewed subjects. For those with severe psoriasis, this increases to 57%. Three fourths (74%) do not like being in public during a flare- up. One third (37%) admit that the disease affects work or school.

### 5. DISCUSSION

In the recent years, there has been an increase in the number of instruments for assessing the quality of life of psoriasis

patients and the role of risk factors in the onset of disease. They can be subdivided into two groups: generic and specific. Generic ones assess the quality of life outside a clinical context. These questionnaires find application in the general population or in various clinical pathologies. Specific questionnaires apply to a single disease only. The CV-50 questionnaire is a specific instrument dedicated to cutaneous diseases and comprises 18 questions. It can be used to assess and compare the quality of life among different skin conditions. The score ranges from 10 to 50, the higher the figures, the deeper the impact on quality of life. The CV-50 questionnaire assesses the impact of psoriasis in four directions: daily activities, job or school, entertainment and personal relations. The patient must recollect his last week and gauge the influence of psoriasis in each of these fields.

Based on the answers yielded by the questionnaire, the fields on which psoriasis had the greatest impact were daily activities and relaxing activities. The results pointed out that the biggest emotional upsets were noted when the disease was located in the face, neck and genital region.

For most cutaneous diseases, risk factors are multiple and often connected with each other. A case-control study represents one of the epidemiological-analytical methods used to test the correlation between risk factors and specific disease frequency. The genetic bases of psoriasis are well-known, its occurrence being under the influence of various factors that can cause the onset, aggravation or remission as well as contribute to chronicization and therapeutic failure. The present study has been designed to investigate the correlation with selected risk factors: alcohol consumption, smoking, stress, medications, recurrent infections, family history, cutaneous aggressions, allergies and hormonal changes.

The study showed that 50% of psoriasis patients blamed stress as the cause of their disease, with a significant difference. Various studies related to the assessment of stressful life events have pointed out that stress can induce the occurrence of the disease, with early onset psoriasis (<40 y.o.) more frequently related to psychological factors such as stress. (43, 44).

Smoking seriously affects internal organs, especially the heart and lungs, but also influences the external appearance including the skin, weight and corporeal forms. The present study, like many similar ones (41-45), revealed a significant difference between smokers and non-smokers, especially among women. Many studies showed that alcohol consumption is closely correlated to the onset of disease (39-43, 45). 80 % of the male psoriasis patients included in the present study consumed alcohol in various amounts.

As for hormonal changes, as in other studies (56), it was noted that they influenced in the aggravation of disease in 36% of female patients.

A vast number of clinical reports show that bacterial infections can induce the occurrence of psoriasis and it is well known that disease aggravations are often preceded by streptococcal infections. (36- 38) In the present study 20% of the patients confirmed that they had recurrent infections.

## 6. CONCLUSION

Stress, tobacco, infections, alcohol, hormones, skin aggressions were confirmed as trigger factors in this study. It was found that the disease affects the quality of life in more than 40% of patients. The scale of impairment from psoriasis is comparable

to other chronic diseases like diabetes or asthma. It is important to point out that the same degree of gravity may have a different impact on different patients, depending on individual characteristics and lifestyles. It is crucial to evaluate the perception of the psoriatic patient about other medical conditions, disabilities and quality of life in order to tailor an adequate individual treatment schedule.

CONFLICT OF INTEREST: NONE DECLARED.

## REFERENCES

- Lomholt G. Prevalence of skin diseases in a population: a census study from the Faroe islands. *Dan Med Bull.* 1964 Feb; 11: 1-7.
- Brandrup F, Green A. The prevalence of psoriasis in Denmark. *Acta Derm Venereol.* 1981; 61(4): 344-346.
- Barisić-Drusko V, Paljan D, Kansky A, Vujasinović S. Prevalence of psoriasis in Croatia. *Acta Derm Venereol Suppl (Stockh).* 1989; 146: 178-179.
- Naldi L, Tognoni G, Cainelli T. Analytic epidemiology in psoriasis. *J Invest Dermatol.* 1994 Jun; 102(6): 19S-23S.
- Stern RS. Epidemiology of psoriasis. *Dermatol Clin.* 1995 Oct; 13(4): 717-722.
- Christophers E. Psoriasis - epidemiology and clinical spectrum. *Clin Exp Dermatol.* 2001 Jun; 26(4): 314-320.
- Naldi L. Epidemiology of psoriasis. *Curr Drug Targets Inflamm Allergy.* 2004 Jun; 3(2): 121-128.
- Schäfer T. Epidemiology of psoriasis. Review and the German perspective. *Dermatology.* 2006; 212(4): 327-337.
- Gudjonsson JE, Elder JT. Psoriasis: epidemiology. *Clin Dermatol.* 2007 Nov-Dec; 25(6): 535-546.
- Braun-Falco O, Christophers E. Structural aspects of initial psoriatic lesions. *Arch Dermatol Forsch.* 1974; 251(2): 95-110.
- Pullmann H, Lennartz KJ, Steigleder GK. In vitro examination of cell proliferation in normal and psoriatic epidermis, with special regard to diurnal variations. *Arch Dermatol Forsch.* 1974; 250(3): 177-184.
- Wiley HE 3rd, Weinstein GD. Abnormal proliferation of uninvolved psoriatic epidermis: differential induction by saline, propranolol, and tapestripping in vivo. *J Invest Dermatol.* 1979 Dec; 73(6): 545-547.
- Van de Kerkhof PC, Van Erp PE. The role of epidermal proliferation in the pathogenesis of psoriasis. *Skin Pharmacol.* 1996; 9(6): 343-354.
- Hatta N, Takata M, Kawara S, Hirone T, Takehara K. Tape stripping induces marked epidermal proliferation and altered TGF- $\alpha$  expression in non-lesional psoriatic skin. *J Dermatol Sci.* 1997 Feb; 14(2): 154-161.
- Prinz JC, Gross B, Vollmer S, Trommler P, Strobel I, Meurer M, Plewig G. T cell clones from psoriasis skin lesions can promote keratinocyte proliferation in vitro via secreted products. *Eur J Immunol.* 1994 Mar; 24(3): 593-598.
- Schlaak JF, Buslau M, Jochum W, Hermann E, Girndt M, Gallati H, Meyer zum Büschenfelde KH, Fleischer B. T cells involved in psoriasis vulgaris belong to the Th1 subset. *J Invest Dermatol.* 1994 Feb; 102(2): 145-149.
- Nickoloff BJ, Wrona-Smith T. Injection of pre-psoriatic skin with CD4+ T cells induces psoriasis. *Am J Pathol.* 1999 Jul; 155(1): 145-158.
- Lowes MA, Kikuchi T, Fuentes-Duculan J, Cardinale I, Zaba LC, Haider AS, Bowman EP, Krueger JG. Psoriasis vulgaris lesions contain discrete populations of Th1 and Th17 T cells. *J Invest Dermatol.* 2008 May; 128(5): 1207-1211. doi: 10.1038/sj.jid.5701213.
- Boyman O, Hefi HP, Conrad C, Nickoloff BJ, Suter M, Nestle FO. Spontaneous development of psoriasis in a new animal model shows an essential role for resident T cells and tumor necrosis factor- $\alpha$ . *J Exp Med.* 2004 Mar 1; 199(5): 731-736.

20. Prinz JC. The role of T cells in psoriasis. *J Eur Acad Dermatol Venereol.* 2003 May; 17(3): 257-270.
21. Krueger JG, Krane JF, Carter DM, Gottlieb AB. Role of growth factors, cytokines, and their receptors in the pathogenesis of psoriasis. *J Invest Dermatol.* 1990 Jun; 94(6 Suppl): 135S-140S.
22. Uyemura K, Yamamura M, Fivenson DF, Modlin RL, Nickoloff BJ. The cytokine network in lesional and lesion-free psoriatic skin is characterized by a T-helper type 1 cell-mediated response. *J Invest Dermatol.* 1993 Nov; 101(5): 701-705.
23. Austin LM, Ozawa M, Kikuchi T, Walters IB, Krueger JG. The majority of epidermal T cells in Psoriasis vulgaris lesions can produce type 1 cytokines, interferon-gamma, interleukin-2, and tumor necrosis factor-alpha, defining TC1 (cytotoxic T lymphocyte) and TH1 effector populations: a type 1 differentiation bias is also measured in circulating blood T cells in psoriatic patients. *J Invest Dermatol.* 1999 Nov; 113(5): 752-759.
24. Chan JR, Blumenschein W, Murphy E, Diveu C, Wiekowski M, Abbondanzo S, Lucian L, Geissler R, Brodie S, Kimball AB, Gorman DM, Smith K, de Waal Malefyt R, Kastelein RA, McClanahan TK, Bowman EP. IL-23 stimulates epidermal hyperplasia via TNF and IL-20R2 dependent mechanisms with implications for psoriasis pathogenesis. *J Exp Med.* 2006 Nov 27; 203(12): 2577-2587.
25. Lima HC, Kimball AB. Targeting IL-23: insights into the pathogenesis and the treatment of psoriasis. *Indian J Dermatol.* 2010 Apr-Jun; 55(2): 171-175.
26. Krueger JG, Fretzin S, Suárez-Fariñas M, Haslett PA, Phipps KM, Cameron GS, McCollm J, Katcherian A, Cueto I, White T, Banerjee S, Hoffman RW. IL-17A is essential for cell activation and inflammatory gene circuits in subjects with psoriasis. *J Allergy Clin Immunol.* 2012 Jul; 130(1): 145-54.e9. doi: 10.1016/j.jaci.2012.04.024.
27. Burks J W, Montgomery H. Histopathologic study of psoriasis. *Arch Derm Syphilol.* 1943; 48(5): 479-493.
28. Gordon M, Johnson WC. Histopathology and histochemistry of psoriasis. I. The active lesion and clinically normal skin. *Arch Dermatol.* 1967 Apr; 95(4): 402-407.
29. Murphy M1, Kerr P, Grant-Kels JM. The histopathologic spectrum of psoriasis. *Clin Dermatol.* 2007 Nov-Dec; 25(6): 524-528.
30. Watson W, Cann HM, Farber EM, Nall ML. The genetics of psoriasis. *Arch Dermatol.* 1972 Feb; 105(2): 197-207.
31. Elder JT, Nair RP, Guo SW, Henseler T, Christophers E, Voorhees JJ. The genetics of psoriasis. *Arch Dermatol.* 1994 Feb; 130(2): 216-224.
32. Henseler T. Genetics of psoriasis. *Arch Dermatol Res.* 1998 Sep; 290(9): 463-476.
33. Bhalerao J, Bowcock AM. The genetics of psoriasis: a complex disorder of the skin and immune system. *Hum Mol Genet.* 1998; 7(10): 1537-1545.
34. Bowcock AM1, Barker JN. Genetics of psoriasis: the potential impact on new therapies. *J Am Acad Dermatol.* 2003 Aug; 49(2 Suppl): S51-S6.
35. Camargo CM, Brotas AM, Ramos-e-Silva M, Carneiro S. Isomorphic phenomenon of Koebner: facts and controversies. *Clin Dermatol.* 2013 Nov-Dec; 31(6): 741-749.
36. Noah PW. The role of microorganisms in psoriasis. *Semin Dermatol.* 1990 Dec; 9(4): 269-276.
37. Weisenseel P, Laumbacher B, Besgen P, Ludolph-Hauser D, Herzinger T, Roecken M, Wank R, Prinz JC. Streptococcal infection distinguishes different types of psoriasis. *J Med Genet.* 2002 Oct; 39(10): 767-768.
38. Fry L, Baker BS. Triggering psoriasis: the role of infections and medications. *Clin Dermatol.* 2007 Nov-Dec; 25(6): 606-615.
39. Poikolainen K, Reunala T, Karvonen J, Lauharanta, Kärkkäinen P. Alcohol intake: a risk factor for psoriasis in young and middle aged men? *BMJ.* Mar 24, 1990; 300(6727): 780-783.
40. Behnam SM, Behnam SE, Koo JY. Alcohol as a risk factor for plaque-type psoriasis. *Cutis.* 2005 Sep; 76(3): 181-185.
41. Naldi L, Parazzini F, Brevi A, Peserico A, Veller Fornasa C, Grosso G, Rossi E, Marinaro P, Polenghi MM, Finzi A, et al. Family history, smoking habits, alcohol consumption and risk of psoriasis. *Br J Dermatol.* 1992 Sep; 127(3): 212-217.
42. Naldi L, Peli L, Parazzini F. Association of early stage psoriasis with smoking and male alcohol consumption: evidence from an Italian case-control study. *Arch Dermatol.* 1999 Dec; 135(12): 1479-1484.
43. Jankovic S, Raznatovic M, Marinkovic J, Jankovic J, Maksimovic N. Risk factors for psoriasis: A case-control study. *J Dermatol.* 2009 Jun; 36(6): 328-334.
44. Naldi L, Chatenoud L, Linder D, Belloni Fortina A, Peserico A, Virgili AR, Bruni PL, Ingordo V, Lo Scocco G, Solaroli C, Schena D, Barba A, Di Landro A, Pezzarossa E, Arcangeli F, Gianni C, Betti R, Carli P, Farris A, Barabino GF, La Vecchia C. Cigarette smoking, body mass index, and stressful life events as risk factors for psoriasis: results from an Italian case-control study. *J Invest Dermatol.* 2005 Jul; 125(1): 61-67.
45. Poikolainen K, Reunala T, Karvonen J. Smoking, alcohol and life events related to psoriasis among women. *Br J Dermatol.* 1994 Apr; 130(4): 473-477.
46. Mallbris L, Larsson P, Bergqvist S, Vingård E, Granath F, Ståhle M. Psoriasis phenotype at disease onset: clinical characterization of 400 adult cases. *J Invest Dermatol.* 2005 Mar; 124(3): 499-504.
47. Cohen AD, Bonneh DY, Reuveni H, Vardy DA, Naggan L, Halevy S. Drug Exposure and Psoriasis Vulgaris: Case-Control and Case-Crossover Studies. *Acta Derm Venereol.* 2005; 85: 299-303.
48. Abel EA, DiCicco LM, Orenberg EK, Fraki JE, Farber EM. Drugs in exacerbation of psoriasis. *J Am Acad Dermatol.* 1986; 15: 1007-1022.
49. Cohen AD, Kagen M, Friger M, Halevy S. Calcium channel blockers intake and psoriasis: a case-control study. *Acta Derm Venereol.* 2001; 81: 347-349.
50. Ikai K. Exacerbation and induction of psoriasis by angiotensin-converting enzyme inhibitors. *J Am Acad Dermatol.* 1995; 32: 819.
51. Steinkraus V, Steinfath M, Mensing H. Beta-adrenergic blocking drugs and psoriasis. *J Am Acad Dermatol.* 1992; 27: 266-267.
52. Ferrier MC, Souteyrand P. Psoriasis and non steroidal anti-inflammatory agents. *Ann Dermatol Venereol.* 1992; 119: 591-595.
53. Rongioletti F, Fiorucci C, Parodi A. Psoriasis induced or aggravated by drugs. *J Rheumatol Suppl.* 2009 Aug; 83: 59-61.
54. Skott A, Mobacken H, Starmark JE. Exacerbation of psoriasis during lithium treatment. *Br J Dermatol.* 1977 Apr; 96(4): 445-448.
55. Kanda N, Watanabe S. Regulatory roles of sex hormones in cutaneous biology and immunology. *J Dermatol Sci.* 2005 Apr; 38(1): 1-7.
56. Mowad CM, Margolis DJ, Halpern AC, Suri B, Synnestvedt M, Guzzo CA. Hormonal influences on women with psoriasis. *Cutis.* 1998 May; 61(5): 257-260.
57. Fransson J, Storgårds K, Hammar H. Palmoplantar lesions in psoriatic patients and their relation to inverse psoriasis, tinea infection and contact allergy. *Acta Derm Venereol.* 1985; 65(3): 218-223.
58. Lipozencić J, Milavec-Puretić V, Pasić A. Contact allergy and psoriasis. *Arh Hig Rada Toksikol.* 1992 Sep; 43(3): 249-254.
59. de Arruda LH, De Moraes AP. The impact of psoriasis on quality of life. *Br J Dermatol.* 2001 Apr; 144 Suppl 58: 33-36.
60. Finlay AY, Coles EC. The effect of severe psoriasis on the quality of life of 369 patients. *Br J Dermatol.* 1995; 132: 236-244.