



Published in final edited form as:

Acta Derm Venereol. 2016 August 23; 96(6): 788–791. doi:10.2340/00015555-2408.

Throat infections are associated with exacerbation in a substantial proportion of patients with chronic plaque psoriasis

Ragna H. Thorleifsdottir, MD^{1,2,3}, Jenna H. Eysteinsdottir, MD^{1,2}, Jon H. Olafsson, MD, PhD¹, Martin I. Sigurdsson, MD, PhD⁴, Andrew Johnston, PhD⁵, Helgi Valdimarsson, MD, FRCP², and Bardur Sigurgeirsson, MD, PhD¹

¹Faculty of Medicine, Section of Dermatology, University of Iceland, Reykjavik, Iceland

²Department of Immunology, Landspítali-The National University Hospital of Iceland, Reykjavik, Iceland ³Department of Dermatology, Akademiska University Hospital, Uppsala, Sweden

⁴Department of Medicine, Landspítali-The National University Hospital of Iceland, Reykjavik, Iceland ⁵Department of Dermatology, University of Michigan Medical School, Ann Arbor, MI, USA

Abstract

Streptococcal throat infections are known to trigger or exacerbate psoriasis, and several studies support the benefit of tonsillectomy. To evaluate the potential of tonsillectomy as a treatment, we used a retrospective study-specific questionnaire to assess the proportion of psoriasis patients with sore throat-associated psoriasis exacerbations. Our survey sampled 275 psoriasis patients. 42% of patients with plaque psoriasis reported sore throat-associated psoriasis exacerbations, and 72% of patients with confirmed streptococcal infections reported aggravation. Notably, women and early onset psoriasis patients were more likely to report psoriasis exacerbation after a sore throat ($p < 0.001$, $p = 0.046$ respectively). Other psoriasis aggravation factors were more common in patients with sore throat-associated exacerbations ($p < 0.01$). 49% of tonsillectomized patients reported subsequent improvement and had more frequent sore throat-associated aggravation of psoriasis than patients who did not improve after tonsillectomy ($p = 0.015$). These findings suggest a closer association between sore throats, streptococcal throat infections and plaque psoriasis than previously reported.

Keywords

Chronic plaque psoriasis; sore throat; streptococcal throat infections; tonsillectomy

INTRODUCTION

Psoriasis is a multifactorial disease caused by a combination of genetic and environmental factors. The disease has a strong genetic basis with over 60 susceptibility loci having now been identified (1, 2), including HLA-Cw6, carriage of which is associated with an

Correspondence: Ragna Hlin Thorleifsdottir MD, Department of Dermatology, Akademiska, University Hospital, 75 185 Uppsala, Sweden, Tel: +46 760649717, ragnahlin@gmail.com.

Conflict of interest: The authors state no conflict of interest.

approximate ten-fold increased risk of developing psoriasis (3). Numerous environmental agents have been reported to trigger and/or exacerbate psoriasis, including psychological stressors, physical trauma, cold weather, cigarette smoking, alcohol intake, and certain drugs (4–7). Similarly, various microorganisms have been implicated, including fungi (*Malassezia*, *Candida albicans*) and viruses (papillomaviruses, retroviruses) (7). However, throat infections with β -haemolytic streptococci have most convincingly been linked with the initiation and exacerbation of psoriasis.

The association between guttate psoriasis and streptococcal infections has been recognized for a 100 years (6, 8–10). However, only a few retrospective (11, 12) and one prospective study (13) have linked exacerbation of plaque psoriasis with streptococcal throat infections. Moreover, such infections are about 10 times more frequent in patients with plaque psoriasis compared to age- and sex-matched household controls (13). Recently it was reported that tonsils from patients with psoriasis are more frequently infected with β -haemolytic streptococci, especially group C streptococci, than recurrently infected tonsils from patients without psoriasis (14). Nevertheless, the immunological basis for the association of psoriasis and streptococcal throat infections is still under investigation.

Palmoplantar pustulosis (PPP) is a painful chronic inflammatory condition, restricted to the palms and/or soles, that once was regarded as a variant of pustular psoriasis but is now categorised with acropustular diseases (15). Up to 20 percent of patients with PPP have concomitant plaque psoriasis (16), and although PPP and plaque psoriasis have different sites of predilection and pathomechanisms (16), they may share common triggering mechanisms. Streptococcal throat infections have been linked to PPP and studies have indicated that PPP may improve after tonsillectomy (17–19)

We have recently reported that patients with plaque psoriasis and a history of sore throat-associated psoriasis exacerbation improve after tonsillectomy (20). The main aim of the current study was to estimate the proportion of patients with plaque psoriasis who experienced disease aggravation after sore throats or streptococcal throat infections, and therefore might be more likely to benefit from tonsillectomy.

METHODS

Study design and cohort

This study is a retrospective large case series that took place from January 2011 to April 2011 at the following dermatology outpatient units in Iceland: The dermatology outpatient center at Landspítali-The National University Hospital of Iceland, Reykjavik; Hudlaeknastodin dermatology clinic, Kopavogur; and the Blue Lagoon geothermal clinic, Grindavik (21). A total of 374 psoriasis patients were approached when visiting these clinics and offered to participate, and 275 (127 males and 148 females) agreed to participate in the study (73.5 percent response rate). All participants were over 18 years of age and had been diagnosed with psoriasis by a dermatologist. The study was approved by the National Bioethics Committee of Iceland, the Data Protection Authority of Iceland and performed in compliance with the 1964 Declaration of Helsinki and its later amendments.

We designed a self-report anonymous questionnaire composed of 15 multiple-choice and short-answer questions (supplementary data). Participants were asked to answer as accurately as possible. The questionnaire addressed 5 main topics: (I) General demographics; (II) Psoriasis subtype, age at onset and if the psoriasis onset had been associated with a sore throat or a streptococcal throat infection; (III) Frequency of sore throats, defined by a painful inflammation/infection of the mucus membranes in the pharynx, and the frequency of streptococcal throat infections diagnosed by a throat culture, rapid antigen detection test (strep test) or by a physician. Also, exacerbation of psoriasis during or within 3 weeks of a sore throat or streptococcal throat infection; (IV) Psoriasis aggravating factors other than sore throat, including general malaise, cold climate, stressful life events, alcohol intake, diet and drugs; (V) Whether the participant had been subjected to tonsillectomy after the onset of psoriasis, age at the time of the surgery and whether the tonsillectomy was associated with changes in the activity of their skin disease.

Statistics

Patient demographics were summarised descriptively. Categorical variables were compared with Chi-Square and Fisher's exact test. Level of statistical significance was set at $p < 0.05$. A logistic regression model of sore throat aggravation was pursued. All variables with a $p < 0.1$ were entered into the logistic regression model. Odds ratio and 95 percent confidence intervals were then estimated. All statistics were performed in R, version 2.10 (The R foundation, Austria).

RESULTS

All 275 recruited participants, 127 males and 148 females, completed the study questionnaire and table 1 shows their demographic information. The majority of the responders (75%) had been diagnosed with plaque psoriasis, 14 percent with both guttate and plaque psoriasis and 8 percent with guttate psoriasis. Four patients reported PPP, but 5 of the 275 study participants did not belong to any of the above categories. Early-onset psoriasis, defined as age at onset of 40 years or less (22), was reported by the majority of the study participants (87%).

Psoriasis exacerbation associated with a sore throat was reported by 42 percent of the patients with plaque psoriasis, 67 percent of the patients with guttate psoriasis and 70 percent of patients with a history of both guttate and plaque psoriasis. This was also the case for 2 out of 4 patients with PPP. Moreover, of the 140 participants with a history of confirmed streptococcal throat infections (table II), 75 percent reported streptococcal-associated psoriasis exacerbation. This applied to 72 percent of patients with plaque psoriasis, 94 percent of patients with guttate psoriasis and 79 percent of patients with both guttate and plaque psoriasis. Furthermore, patients who reported sore throat-associated aggravation were more likely to report streptococcal-associated psoriasis exacerbation (93% vs 7%, $p < 0.001$). This also applied to subgroups of psoriasis patients: plaque psoriasis (92% vs 8%, $p < 0.001$), guttate psoriasis (94% vs 6%, $p = 0.01$) and patients with both guttate and chronic plaque psoriasis (100% vs 0%, $p = 0.005$ (table II). A significantly higher ratio of

early-onset psoriatics reported psoriasis exacerbation associated with a sore throat, compared to patients with late-onset psoriasis (51% vs. 30%, $p=0.046$).

The sore throat-associated aggravation was notably more common among the women than the men (61% vs. 32%, $p<0.001$). Even after adjustment for the influence of age, psoriasis subtypes (see table I), and other psoriasis aggravation factors, females still had a significantly higher risk of sore throat-associated psoriasis aggravation (OR= 2.5, 95% CI 1.37–4.58, $p=0.003$). Psoriasis exacerbation associated with general malaise, cold climate, stress, consumption of alcohol or various diets were significantly more often reported by the patients who also reported sore throat-associated psoriasis aggravation (Table III), and this difference was still significant for cold climate and general malaise, after adjustment for age, gender and psoriasis subtypes (OR= 9.2 and 3.0, 95% CI 3.83–22.3 and 1.58–5.74, $p<0.001$ respectively). There were no differences between males and females in this respect.

Of the 275 participants, had 56 (20%) been tonsillectomized after the onset of psoriasis (Table IV), and 48 percent of these reported that tonsillectomy was associated with improvement in their psoriasis. Interestingly, 18/37 (49%) of plaque psoriatics and 3/4 (75%) of guttate patients noted improvement in psoriasis after tonsillectomy. This also applied to 6/11 (55%) of patients with both guttate and plaque psoriasis (table IV). Patients who noted improvement after tonsillectomy more frequently reported sore throat-associated aggravation ($p=0.015$). All patients who reported improvement after tonsillectomy also reported early onset of psoriasis.

DISCUSSION

Psoriasis is a heterogeneous disease with respect to both genetic (ref 1, 2) and pathologic components (Heterogeneity of Inflammatory and Cytokine Networks in Chronic Plaque Psoriasis, Swindell WR PLoS One. 2012;7(3):e34594.; Molecular dissection of psoriasis: integrating genetics and biology. Elder JT, J Invest Dermatol. 2010 May;130(5):1213–26.) and several external factors have been reported to contribute to the onset and exacerbation of psoriasis (4–6). However, streptococcal throat infection is the only environmental factor that has convincingly been connected to the immunological mechanisms thought to operate in psoriasis (23, 24), especially in patients carrying the HLA-Cw6 allele. With the HLA-Cw6 as the major psoriasis susceptibility allele, the CD8+ T cells are thought to be the major effector cells in psoriasis as they may respond to peptide antigens presented in the context of HLA-Cw6. Chronic stimulation by streptococci in the tonsils gives rise to a set of pathogenic skin-homing (CLA+) T cells (25). The link between streptococcal throat infections and psoriasis is supported by several lines of research, including increased T cell responses to streptococcal-derived peptides (26–28), shared T cell receptor rearrangements in psoriasis tonsil and skin-homing and skin-resident T cells (29), increased streptococcal-reactive IgG titers in the blood of patients with plaque psoriasis (30) and increased throat carriage rate of streptococci amongst patients with psoriasis (13, 14). Once generated in the tonsils, skin-homing T cells can migrate to the dermis and epidermis where they are thought to cross-react with skin-derived epitopes such as keratins (28, 29, 30, Shen Z, J Dermatol Sci. 2005 Apr;38(1):25–39; JID Ferran M et al. J Invest Dermatol.133(4):999–1007 (2013)), maspin, ezrin, PRDX2, hsp27 (30) or melanocyte-derived peptides (Arakawa A J Exp Med.

2015 Dec 14;212(13):2203–12.), driving the cutaneous inflammation characteristic of psoriasis. Several studies have indicated that psoriasis can improve after tonsillectomy (31) (and Sigurdardottir SL, Br J Dermatol. 2013 Feb;168(2):237–42), but indications for such treatment remain to be established. However, most of the patients who have been treated in this way had a history of psoriasis exacerbation in association with sore throats and/or streptococcal throat infections.

Here we report that 42 percent of patients with plaque psoriasis have experienced worsening of their disease in association with sore throat. Furthermore, 72 percent of the participants with plaque psoriasis and confirmed streptococcal throat infections reported exacerbation of their skin lesions. This is a higher frequency than previously reported by Wardrop *et al*, where 33 percent of patients with plaque psoriasis associated worsening of psoriasis to sore throat compared with 3 percent of matched eczema controls (11). Note, our study was designed as a retrospective questionnaire and could therefore be limited by recall bias. Sore throat-associated psoriasis aggravation was more common amongst women ($p=0.003$). It is not clear why this gender difference exists, but it has been observed that women are more frequently affected with recurrent tonsillitis than males (32). Female sex hormones or altered skin corticosteroid levels might be involved, but we are not aware of any reports on this issue. To that end, it might be interesting to assess prospectively whether postmenopausal women are less sensitive to psoriasis exacerbation after a sore throat and/or a streptococcal throat infection. Patients reporting sore throat-associated aggravation of psoriasis also noted worsening in relation to various other aggravation factors such as general malaise, stress, alcohol or cold weather. Notably, such associations were not reported by those participants who did not associate sore throat with psoriasis exacerbation. Chronic plaque psoriasis has previously been subdivided to stable and dynamic types (33). Patients with the dynamic type have a more fluctuating course, appear to be more influenced by the various exacerbating factors listed above, and are more often carriers of the HLA-Cw6 allele than patients with relatively stable disease. This form of plaque psoriasis has even been considered somewhat similar to guttate psoriasis (34). Beside the association between sore throat-associated psoriasis aggravation and other psoriasis exacerbating factors, our data also show that early-onset psoriasis patients are more prone to sore throat-induced psoriasis aggravation. This suggests that these patients have the dynamic phenotype of plaque psoriasis, and thus, might be appropriate candidates for tonsillectomy. However, our study cohort was limited to Icelandic psoriasis patients and Iceland's geographic isolation might have influenced the development of patients more affected by environmental trigger factors such as streptococcal throat infections.

The tonsils are a major target for streptococcal infections in humans, which are the most common cause of bacterial pharyngitis (35). The high level of streptococcal throat carriage and infections in patients with plaque psoriasis is noteworthy, with the carrier rate for groups A, C and G streptococci as high as 44 percent (14). Long-term treatment with antibiotics has not been effective for psoriasis (36). Streptococci can exist in both the extra- and intracellular spaces, forming intracellular reservoirs inside endothelial cells and macrophages within the tonsils (37). At best, antibiotic therapy only manages to reduce the bacterial load in the tonsils, leaving quiescent intracellular streptococci in the tonsillar

epithelia and macrophages (37). These streptococci can reactivate, recolonize and cause symptoms again, whereas tonsillectomy might remove this pool of streptococci.

This and a number of prior studies support the association between psoriasis and streptococci (11–13, 20). Despite the lack of large controlled clinical trials, tonsillectomy is commonly advocated for patients with recurrent guttate psoriasis. Furthermore, according to an European expert group consensus, tonsillectomy may now be indicated for juvenile psoriasis patients with a positive streptococcal culture and more than three recurrent infections (38). Our findings might help to identify patients with plaque psoriasis that may benefit from tonsillectomy, but they need to be confirmed with prospective and more structured studies.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

We are indebted to the staff at Landspítali-The National University Hospital of Iceland dermatology outpatient centre, Hudlaeknastodin dermatology clinic and the Blue Lagoon geothermal clinic for their assistance during the study. This study was supported in part by the Icelandic Research Fund and Landspítali University Hospital Research Fund. AJ is supported by NIH K01 AR064765, the National Psoriasis Foundation USA and the Babcock Memorial Trust. The funding institutions had no role in the study.

REFERENCES

1. Tsoi LC, Spain SL, Knight J, Ellinghaus E, Stuart PE, Capon F, et al. Identification of 15 new psoriasis susceptibility loci highlights the role of innate immunity. *Nat Genet.* 2012; 44(12):1341–1348. [PubMed: 23143594]
2. Tsoi LC, Spain SL, Ellinghaus E, Stuart PE, Capon F, Knight J, et al. Enhanced meta-analysis and replication studies identify five new psoriasis susceptibility loci. *Nat Commun.* 2015; 6:7001. [PubMed: 25939698]
3. Nair RP, Stuart PE, Nistor I, Hiremagalore R, Chia NV, Jenisch S, et al. Sequence and haplotype analysis supports HLA-C as the psoriasis susceptibility 1 gene. *Am J Hum Genet.* 2006; 78(5):827–851. [PubMed: 16642438]
4. Eyre RW, Krueger GG. Response to injury of skin involved and uninvolved with psoriasis, and its relation to disease activity: Koebner and 'reverse' Koebner reactions. *The British journal of dermatology.* 1982; 106(2):153–159. [PubMed: 7037041]
5. Naldi L, Parazzini F, Brevi A, Peserico A, Veller Fornasa C, Grosso G, et al. Family history, smoking habits, alcohol consumption and risk of psoriasis. *Br J Dermatol.* 1992; 127(3):212–217. [PubMed: 1390163]
6. 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; 124(3):499–504. [PubMed: 15737189]
7. Fry L, Baker B. Triggering psoriasis: the role of infections and medications. *Clin Dermatol.* 2007; 25(6):606–615. [PubMed: 18021899]
8. Winfield JM. Psoriasis as a sequel to acute inflammations of the tonsils: a clinical note. *J Cutan Dis.* 1916; 34:441–443.
9. Whyte H, Baughman R. Acute guttate psoriasis and streptococcal infection. *Arch Dermatol.* 1964; 89:350–356. [PubMed: 14096349]
10. Telfer N, Chalmers R, Whale K, Colman G. The role of streptococcal infection in the initiation of guttate psoriasis. *Arch Dermatol.* 1992; 128(1):39–42. [PubMed: 1739285]

11. Wardrop P, Weller R, Marais J, Kavanagh G. Tonsillitis and chronic psoriasis. *Clin Otolaryngol Allied Sci.* 1998; 23(1):67–68. [PubMed: 9563669]
12. Bartenjev I, Rogl Butina M, Potocnik M. Subclinical microbial infection in patients with chronic plaque psoriasis. *Acta Derm Venereol Suppl (Stockh).* 2000; (211):17–18. [PubMed: 11234557]
13. Gudjonsson J, Thorarinsson A, Sigurgeirsson B, Kristinsson K, Valdimarsson H. Streptococcal throat infections and exacerbation of chronic plaque psoriasis: a prospective study. *The British journal of dermatology.* 2003; 149(3):530–534. [PubMed: 14510985]
14. Sigurdardottir SL, Thorleifsdottir RH, Valdimarsson H, Johnston A. The association of sore throat and psoriasis might be explained by histologically distinctive tonsils and increased expression of skin-homing molecules by tonsil T cells. *Clinical and experimental immunology.* 2013; 174(1): 139–151. [PubMed: 23750651]
15. de Waal AC, van de Kerkhof PC. Pustulosis palmoplantaris is a disease distinct from psoriasis. *J Dermatolog Treat.* 2011; 22(2):102–105. [PubMed: 20687846]
16. Mrowietz U, van de Kerkhof PC. Management of palmoplantar pustulosis: do we need to change? *Br J Dermatol.* 2011; 164(5):942–946. [PubMed: 21275942]
17. Andrews GC, Machacek GF. Pustular bacterids of the hands and feet. *Arch Derm Syphilol.* 1935; 32:837–835.
18. Yokoyama M, Hashigucci K, Yamasaki Y. Effect of tonsillectomy in patients with pustulosis palmaris et plantaris. *Acta Otolaryngol.* 2004; 124(9):1109–1110. [PubMed: 15513559]
19. Takahara M. Clinical outcome of tonsillectomy for palmoplantar pustulosis and etiological relationship between palmoplantar pustulosis and tonsils. *Adv Otorhinolaryngol.* 2011; 72:86–88. [PubMed: 21865698]
20. Thorleifsdottir RH, Sigurdardottir SL, Sigurgeirsson B, Olafsson JH, Sigurdsson MI, Petersen H, et al. Improvement of psoriasis after tonsillectomy is associated with a decrease in the frequency of circulating T cells that recognize streptococcal determinants and homologous skin determinants. *J Immunol.* 2012; 188(10):5160–5165. [PubMed: 22491250]
21. Eysteinsdóttir JH, Ólafsson JH, Agnarsson BA, Lúðvíksson BR, Sigurgeirsson B. Psoriasis treatment: faster and long-standing results after bathing in geothermal seawater. A randomized trial of three UVB phototherapy regimens. *Photodermatol Photoimmunol Photomed.* 2014; 30(1):25–34. [PubMed: 24255991]
22. Henseler T, Christophers E. Psoriasis of early and late onset: characterization of two types of psoriasis vulgaris. *J Am Acad Dermatol.* 1985; 13(3):450–456. [PubMed: 4056119]
23. Valdimarsson H, Thorleifsdottir R, Sigurdardottir S, Gudjonsson J, Johnston A. Psoriasis--as an autoimmune disease caused by molecular mimicry. *Trends in immunology.* 2009; 30(10):494–501. [PubMed: 19781993]
24. Sigurdardottir SL, Thorleifsdottir RH, Valdimarsson H, Johnston A. The role of the palatine tonsils in the pathogenesis and treatment of psoriasis. *Br J Dermatol.* 2013; 168(2):237–242. [PubMed: 22901242]
25. Sigmundsdottir H, Gudjonsson JE, Valdimarsson H. Interleukin-12 alone can not enhance the expression of the cutaneous lymphocyte associated antigen (CLA) by superantigen-stimulated T lymphocytes. *Clin Exp Immunol.* 2003; 132(3):430–435. [PubMed: 12780689]
26. Sigmundsdottir H, Sigurgeirsson B, Troye-Blomberg M, Good M, Valdimarsson H, Jonsdottir I. Circulating T cells of patients with active psoriasis respond to streptococcal M-peptides sharing sequences with human epidermal keratins. *Scand J Immunol.* 1997; 45(6):688–697. [PubMed: 9201310]
27. Johnston A, Gudjonsson J, Sigmundsdottir H, Love T, Valdimarsson H. Peripheral blood T cell responses to keratin peptides that share sequences with streptococcal M proteins are largely restricted to skin-homing CD8(+) T cells. *Clin Exp Immunol.* 2004; 138(1):83–93. [PubMed: 15373909]
28. Besgen P, Trommler P, Vollmer S, Prinz JC, Ezrin, maspin, peroxiredoxin 2, and heat shock protein 27: potential targets of a streptococcal-induced autoimmune response in psoriasis. *J Immunol.* 2010; 184(9):5392–5402. [PubMed: 20363977]

29. Diluvio L, Vollmer S, Besgen P, Ellwart J, Chimenti S, Prinz J. Identical TCR beta-chain rearrangements in streptococcal angina and skin lesions of patients with psoriasis vulgaris. *Journal of immunology*. 2006; 176(11):7104–7111.
30. El-Rachkidy RG, Hales JM, Freestone PP, Young HS, Griffiths CE, Camp RD. Increased blood levels of IgG reactive with secreted *Streptococcus pyogenes* proteins in chronic plaque psoriasis. *J Invest Dermatol*. 2007; 127(6):1337–1342. [PubMed: 17344934]
31. Rachakonda TD, Dhillon JS, Florek AG, Armstrong AW. Effect of tonsillectomy on psoriasis: a systematic review. *J Am Acad Dermatol*. 2015; 72(2):261–275. [PubMed: 25455609]
32. Kvestad E, Kvaerner KJ, Røysamb E, Tambs K, Harris JR, Magnus P. Heritability of recurrent tonsillitis. *Arch Otolaryngol Head Neck Surg*. 2005; 131(5):383–387. [PubMed: 15897415]
33. Gudjonsson J, Johnston A, Sigmundsdottir H, Valdimarsson H. Immunopathogenic mechanisms in psoriasis. *Clin Exp Immunol*. 2004; 135(1):1–8. [PubMed: 14678257]
34. Gudjonsson J, Karason A, Runarsdottir E, Antonsdottir A, Hauksson V, Jónsson H, et al. Distinct clinical differences between HLA-Cw*0602 positive and negative psoriasis patients--an analysis of 1019 HLA-C- and HLA-B-typed patients. *J Invest Dermatol*. 2006; 126(4):740–715. [PubMed: 16439971]
35. Cunningham MW. Pathogenesis of group A streptococcal infections. *Clin Microbiol Rev*. 2000; 13(3):470–511. [PubMed: 10885988]
36. Owen C, Chalmers R, O'Sullivan T, Griffiths C. A systematic review of antistreptococcal interventions for guttate and chronic plaque psoriasis. *The British journal of dermatology*. 2001; 145(6):886–890. [PubMed: 11899140]
37. Osterlund A, Popa R, Nikkila T, Scheynius A, Engstrand L. Intracellular reservoir of *Streptococcus pyogenes* in vivo: a possible explanation for recurrent pharyngotonsillitis. *Laryngoscope*. 1997; 107:640–647. [PubMed: 9149167]
38. Stahle M, Atakan N, Boehncke WH, Chimenti S, Dauden E, Giannetti A, et al. Juvenile psoriasis and its clinical management: a European expert group consensus. *J Dtsch Dermatol Ges*. 2010; 8(10):812–818. [PubMed: 20738459]

Table I

Baseline demographic data of the 275 participating psoriasis patients

	Percent (n)
Males (n)	47% (127)
Females (n)	53% (148)
Age, mean yr \pm SD	42.3 \pm 14.2
Age at psoriasis onset, mean yr \pm SD	22.3 \pm 13.4
Early onset psoriasis (onset before or at 40 years)	87% (240)
Late onset psoriasis (onset after the age of 40 years)	10% (27)
Psoriasis subtype:	
Plaque psoriasis	75% (207)
Guttate psoriasis	8% (21)
Guttate and plaque psoriasis	14% (38)
Palmoplantar pustulosis (PPP)	1% (4)
Psoriasis nail changes	42% (116)
Psoriatic arthritis	20% (56)

n: number, SD: standard deviation

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table IIStreptococcal-associated psoriasis exacerbations amongst participants with confirmed^a streptococcal infections

	Percent (n)
Participants with confirmed streptococcal throat infections ^a	51% (140)
Streptococcal-associated psoriasis exacerbation	75% (105)
Plaque psoriasis	72% (69/96)
Guttate psoriasis	94% (15/16)
Guttate and plaque psoriasis	79% (19/24)
Palmoplantar pustulosis (PPP)	50% (1/2)

^aConfirmed by a throat culture, rapid antigen detection test or a physician

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table III

Associations between sore throat-associated exacerbations and other factors reported to aggravate psoriasis

Psoriasis exacerbating factors	Number	Patients with sore throat-induced exacerbation	Patients without sore throat-induced exacerbation	<i>p</i> -value
General malaise	65	88% (57)	12% (8)	< 0.001
Cold climate	162	64% (103)	36% (59)	< 0.001
Stress	173	64% (110)	36% (63)	< 0.001
Alcohol	68	71% (48)	29% (20)	0.005
Diet ^a	36	75% (27)	25% (9)	0.01
Drugs ^b	9	78% (7)	22% (1)	n.s.

^aNot specified^bIncluding Lithium, Beta-blockers, Penicillin and Methotrexate

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table IV

The effects of tonsillectomy reported by 56 psoriasis patients who were tonsillectomized after the onset of their psoriasis

	Percent (n)
Improvement after tonsillectomy	48% (27/56)
Plaque psoriasis	49% (18/37)
Guttate psoriasis	75% (3/4)
Guttate and plaque psoriasis	55% (6/11)
Not sure or no improvement	52% (29/56)

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript