Review

Epidemiological characteristics of psoriatic arthritis

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ABSTRACT

Psoriatic arthritis (PsA) is a specific form of inflammatory arthritis associated with skin psoriasis. PsA makes part of a heterogeneous group of arthritides called the spondyloarthropathies. Several studies regarding the prevalence and incidence of PsA have been published during the last decades, showing a considerable variation of the disease occurrence among different populations. The purpose of this review is to discuss recent observations of epidemiological features for PsA patients. Thus, the literature was reviewed until May 2018 for studies regarding PsA epidemiology, classification criteria and risk factors for PsA development. Systematic reviews based on the international bibliography, are reporting the prevalence of the disease from 1/100.000 inhabitants in Japan to as high as 420/100.000 inhabitants in Italy. The annual incidence also varies, ranging from 1 to 23/100.000 inhabitants, while the average incidence rate is 6.5 cases/100.000 inhabitants. The random effect pooled PsA prevalence and incidence rates are 133/100.000 and 83/100.000 subjects respectively. Thus, a large heterogeneity between studies is observed. This variability could be explained by a number of factors such as the use of multiple and different classification criteria in the studies. Geographical variations are also observed regarding disease occurrence. Differences were found not only between different continents, but also within the same geographic regions. This could be explained by the different genetic background especially the distribution of the human leucocyte antigens. In addition, other factors such as environmental (infections, climate, sun exposure), dietary habits (fish oil consumption, Mediterranean diet) or life style habits (obesity, smoking), could explain the geographic variability in the prevalence estimates. The implementation of unanimous classification criteria and the conformation by the scientific community could lead to a better understanding of the disease epidemiology.

Introduction

Psoriatic arthritis (PsA) is a chronic inflammatory, potentially destructive arthritis which is directly related to psoriasis of the skin (1, 2). The disease often leads to reduction in physical activity, poor quality of life and cardiovascular involvement, resulting in increased costs both for patients and healthcare systems (3-5). PsA makes part of a heterogeneous group of arthritides called the spondyloarthropathies (SpA), including ankylosing spondylitis, reactive arthritis, enteropathic arthritis and undifferentiated arthritis (1, 2).

PsA affects both peripheral joints and the axial skeleton. Moll and Wright described the clinical pattern of SpA which includes: a) distal joint disease, b) arthritis mutilans, c) oligoarthritis affecting 4 or fewer joints, d) polyarthritis similar to rheumatoid arthritis (RA) and spondyloarthritis which occurs alone or in association with peripheral arthritis (6). PsA is characterised by erosions of the bone structures, periosteal reaction and osteolysis with the involvement of ligaments, tendons and enthuses (7, 8). Extra-articular manifestations affecting the skin, eyes, gastrointestinal, and cardiovascular system are not uncommon (9, 10). Skin psoriasis is a chronic inflammatory dermopathy, which appears in approximately 2–3% of the general population (1, 9-11). It is rarely found in African, Asian and Japanese populations (12). Approximately 70–80% of the patients develop psoriasis before the appearance of arthritis. Arthritis usually occurs in these patients 7 to 10 years after the onset of the skin manifestations (13, 14). In 15% of cases, skin is affected simultaneously, while 15%

musculoskeletal manifestations precede the onset of psoriasis by 1–2 years (13, 15). Several studies regarding the prevalence and incidence of PsA have been published during the last decades, showing a considerable variation of the disease occurrence among different populations. The purpose of this review is to discuss recent observations of epidemiological features for PsA patients. Thus, the literature was reviewed until May 2018 for studies regarding PsA epidemiology, classification criteria and risk factors for PsA development.

Table I. Occurrence of psoriatic arthritis in patients with psoriasis.

Study	Year	Centre	Number of patients	Prevalence %	Reference
Leczinsky *et al*.	1948	Sweden	534	7	30
Vilanova *et al*.	1951	Spain	214	25	31
Little *et al*.	1975	Canada	100	32	32
Leonard *et al*.	1978	Rochester	77	39	33
Green *et al*.	1981	Cape Town	61	42	34
Scarpa *et al*.	1984	Naples	180	34	35
Stern *et al*.	1985	Boston	1285	20	36
Zanolli *et al*.	1992	Winston-Salem	459	17	37
Falk *et al*.	1993	Kautokeino	35	17	38
Barisic-Drusko *et al*.	1994	Osijek region	553	10	39
Salvarani *et al*.	1995	Reggio Emilia	205	36	40
Shbeeb *et al*.	2000	USA	1056	6.25	41
Brockbank *et al*.	2001	Canada	126	31	42
Alenius *et al*.	2002	Sweden	276	48	43
Zachariae *et al*.	2003	Denmark	5795	30	44
Gisondi *et al*.	2005	Italy	936	7.7	45
Ibrahim *et al*.	2009	United Kingdom	168	13.8	46
Mease *et al*.	2013	USA-Europe	949	30	47
Ogdie *et al*.	2013	United Kingdom	4064	8.9	48
Maldonado *et al*.	2014	Argentina	100	17	49
Henesetal.	2014	Germany	404	30.2	50
López Estebaránz *et al*.	2015	Spain	375	23	51
Ranza *et al*.	2015	Brazil	524	33	52
Papadavid *et al*.	2016	Greece	278	30	53

Classification criteria

The lack, until a few years ago, of widely accepted classification criteria has resulted in different and often contradictory data in the literature about the clinical phenotype and the epidemiological characteristics of the disease. The first classification criteria for PsA have been published by Moll and Wright in 1973 (6). They described PsA as a different entity in comparison with RA. Moll and Wright's criteria included the presence of psoriasis or a positive history of psoriasis, a negative rheumatoid factor and some of the clinical manifestations of PsA (6) as described above. For the diagnosis of PsA two basic criteria and one of the five clinical subcategories are required. Although they were used for many years by the scientific community, they have been criticised because of their low sensitivity. Criticism focused primarily on the prerequisite of a negative rheumatoid factor. However, it is known that almost 10% of PsA patients have a positive rheumatoid factor in low titers. In addition, these criteria did not include heredity/genetic predisposition for PsA (6, 16, 17).

In many studies, the Vasey and Espinoza criteria despite their limited acceptance by the scientific community proved to have greater sensitivity when compared to the Moll and Wright criteria. This is probably due to the fact that they include even patients with a positive rheumatoid factor, when the distribution of the affected joints is not symmetrical (17, 18).

The use of the Fournie (19) classification criteria as well as the Bennett criteria was also limited. The latter combined clinical manifestations of the disease with characteristic imaging findings of PsA adding two criteria concerning synovial histology and the microscopical examination of the synovial fluid (20). McGonagle's most recent criteria gave great importance to enthesopathy, (21) while Gladman's criteria essentially altered Moll and Wright's criteria by adding exclusion criteria such as rheumatoid nodules, crystalline arthritis, inflammation of the intestine, etc. (13). The classification criteria of the European Spondyloarthropathy Study Group (ESSG) of seronegative SpA, including PsA, were then formulated (22). The ESSG criteria are characterised by a sensitivity of 86% and a specificity of 87% (22). Amor *et al*. criteria, which have greater specificity and sensitivity (98% and 92% respectively) compared to the ESSG criteria have been used in clinical practice (23). The most recent widely accepted classification criteria of SpA, are the criteria of the Assessment of the Spondyloarthritis International Society (ASAS) which require an inflammatory back pain for more than 3 months in young subjects (less than 45 years of age) and various clinical and laboratory manifestations of the SpA group. ASAS criteria have a specificity of 84.4% and a sensitivity of 82.9% (24). The ASAS criteria were adapted for patients with SpA and peripheral involvement and found to have a sensitivity of 77.8% and a specificity of 82.2% (25).

Approximately ten years ago, GRAPPA published the Classification of Psoriatic Arthritis (CASPAR) criteria. The CASPAR criteria have high sensitivity and specificity, 91.4% and 98% respectively, for patients with established disease (26). In recent studies they have been proved to be reliable criteria also for the diagnosis of early PsA, demonstrating high sensitivity and specificity (27, 28). These criteria include also patients with a positive rheumatoid factor while taking into account the genetic predisposition and heredity of the disease. These criteria are widely accepted by the scientific community and are used to diagnose PsA patients.

Not all patients diagnosed with psoriasis will develop PsA. Different studies report rates ranging from 7 to 48% (Table I). Researchers postulate that the reason could be the different genetic background among different populations, but also to the large diversity of studies using different classification criteria (29-53).

Table II. Psoriatic arthritis prevalence studies.

Country (year of conduct)	Study type	Study method	Diagnosis criteria	Prevalence /10^5 (95%CI)	Reference
USA (2000)	Retrospective	Medical Records	Arthritis and psoriasis	101 (81–121)	41
Japan (2001)	Retrospective	Questionnaires	Amor & ESSG	1	55
Greece (2003)	Retrospective	Medical Records	ESSG	56.6 (49.9–63.2)	56
Australia (2004)	Retrospective	Questionnaires	Psoriasis & pain, arthritis, enthesitis, tendinitis, low back pain	500 (0.0–900)	57
Greece (2005)	Prevalence study	Questionnaires	ESSG	170 (100–240)	58
France (2005)	Prevalence study	Telephone Queries & Examination	ESSG	190 (80–350)	59
Italy (2005)	Prevalence study	Questionnaires & examination	Arthritis / out. Affection and psoriasis	420 (310–610)	60
USA (2005)	Prevalence study	Questionnaires	Patient reports	250 (180–310)	61
Iceland (2007)	Prevalence study	Interview - Medical Records - examination	Psoriasis and arthritis	139 (112–169)	62
China (2008)	Retrospective	Medical Records	Amor & ESSG	From 10 to 100	63
Denamrk (2008)	Prevalence study	Interview - Medical Records - examination	Moll & Wright CASPAR	150 (130-220) 140 (110-190)	64
Greece (2008)	Systematic review	MedLine Search	-	From 1 to 420	65
USA (2009)	Retrospective	Medical Records	CASPAR	158 (132–185)	29
Norway (2009)	Retrospective	ICD diagnosis	Arthritis and psoriasis	127 (106–154)	66
Czech Republic (2010)	Prospective	First-level health centers	Vasey& Espinoza	49.1 (39.5–60.4)	67
Argentina (2011)	Prospective	Medical Records	CASPAR	74 (57–94)	68
Italy (2012)	Systematic review	MedLine Search	-	From 1 to 500	69

ESSG: European Spondyloarthrpathy Study Group; CASPAR: Classification Criteria for Psoriatic Arthritis; ICD: International Classification of Diseases; CI: Confidence Interval.

Prevalence and incidence

To date, various epidemiological studies show PsA prevalence ranging from 0.1% to 1% in the general population (7, 54). The prevalence of PsA in the general population varies significantly due to factors such as genetic background and the recent lack of widely accepted classification criteria. Nevertheless, current data indicates a prevalence of 1/100,000 in Japan to 500/100,000 in Australia (Table II) (29, 41, 55-69).

Special mention should be made of two systematic reviews. The first one, conducted by our group in 2008, recorded the prevalence of PsA ranging from 1/100.000 inhabitants in Japan to 420/100.000 in Italy (65). The 2nd review of Catanoso *et al*., published in 2012, presents the PsA prevalence fluctuating from 1/100000 to 500/100000 (69). It should be noted that these studies included patients who were classified on the basis of different classification criteria, including the ESSG criteria, the CASPAR criteria, diagnostic codes from the International Classification of Diseases (ICD), medical records, clinical findings (psoriasis and other inflammatory arthritides), etc.

The incidence of PsA in the general population also varies greatly in published studies (Table III). In the two systematic reviews mentioned above, the annual incidence of PsA ranges from 0.1 to 23 cases per 100,000 inhabitants, while the median incidence rate is 6.5 cases per 100,000 inhabitants (29, 41, 55, 56, 64, 66-68, 70-73).

A recent systematic review and meta-analysis found an overall pooled prevalence of 123/100.000 subjects (95% CI 107-164/100.000 subjects) and incidence of 83/100.000 patient/years (PY) (95% CI 40-167/100.000 PY) (74). A large heterogeneity between studies was observed. This variability could be explained by a number of factors, such as the use of multiple and different classification criteria in the studies. The clinical manifestations included in the various sets of criteria is another factor that might influenced their performance. For example, to fullfil the CASPAR criteria, a patient must have peripheral joint arthritis, spondylitis or enthesitis. However, it is well known that enthesitis is very difficult to be identified in clinical terms. Thus, overestimation or underestimation of enthesitis may occurs, which changes in the criteria performance. On the other hand, in the ESSG criteria enthesitis is not one of the entry criteria (22). A study by Taylor *et al*. showed that a part from the Bennett and ESSG criteria, the published classification criteria for PsA have similar test-performance characteristics (75).

Geographic differences were observed in various studies and could explain much of the variability among studies. Differences were found not only

Table III. Psoriatic arthritis incidence Studies.

Country (year of conduct)	Study type	Study method	Diagnosis criteria	Incidents /10^5 (95%CI)	Reference
Finland (1996)	Retrospective	Certificates of drug treatment	Per. Arthritis / axial involvement & psoriasis	6.1 (4.6–7.6)	73
USA (2000)	Retrospective	Medical Records	Arthritis & psoriasis	6.6(5.0–8.2)	41
Japan (2001)	Retrospective	Questionnaires	Amor & ESSG	0.05	55
Sweden (2002)	Prospective	Health centres	Arthritis & psoriasis	8 (4–15)	70
Greece (2003)	Retrospective	Medical Records	ESSG	3.02 (1.55–4.49)	56
Finland (2003)	Prospective	Health centres – outpatient clinic	Per. Arthritis / axial involvement & psoriasis	23.1 (13.2–37.5)	71
Denmark (2008)	Epidemiological study	Medical Records – interview & clinical examination	Moll & Wright CASPAR	6 (3–11)	64
USA (2009)	Retrospective	Medical Records	CASPAR	7.2 (6.0–8.4)	29
Norway (2009)	Retrospective	ICD codes	Psoriasis & arthritis	6.9 (3.5–11.7)	66
Czech Republic (2010)	Prospective	Health centres	Vasey & Espinoza	3.6 (1.4–7.6)	67
Argentina (2010)	Prospective	Medical Records & ICD codes	CASPAR	6.26 (4.2–8.3)	68
Norway (2015)	Epidemiological study	Questionnaires & Clinical Examination	Psoriasis & arthritis	41.3–9 years of psoriasis (35.8–47.6)	72

ESSG: European Spondyloarthropathy Study Group; CASPAR: Classification Criteria for Psoriatic Arthritis; ICD: International Classification of Diseases; CI: Confidence Interval.

between different continents, but also within the same geographic area. For example, regarding Southern Europe, the prevalence in Greece varies from 56/100.000 subjects, to 420 cases/100.000 subjects in Italy (65, 69, 74). This could be explained by the different genetic background especially by a different distribution of the human leukocyte antigen (HLA).

In addition, other factors such as environmental (infections, climate, sun exposure), dietary habits (fish oil consumption, Mediterranean diet) or life style habits (obesity, smoking), could explain the geographic variability in the prevalence estimates. Below, the risk factors are discussed in more detail.

Time trends

There is limited data on trends for the incidence and prevalence of PsA over time. A study carried out in NW Greece, for a period of 20 years, showed that both incidence and prevalence are increasing in the last decade (56). Another study from USA showed an increase of the incidence for the last 30 years (29). A recent study from Turkey considering the prevalence between 2003-2012, found also an increase time trend in PsA prevalence (76). This trend may be related either to a true increase in disease expression or more likely to the improvement of the classification criteria and the ability of identifying the disease due to a better recognition of PsA by the physicians such as general practitioners, rheumatologists and dermatologists.

Risk factors

A risk factor is any factor (genetic, environmental or personal) that increases the risk of developing a disease. There is a general consensus that PsA is a multifactorial disease, in which genetics and environmental factors may contribute to disease manifestation, expression and evolution. We describe here the most important genetic, environmental and lifestyle factors that have been reported influencing the occurrence of the disease (9, 10).

Genetics

There is an increased prevalence in first-degree relatives of PsA probands, suggesting a familial aggregation and genetic predisposition. A number of lines of evidence point out the role of genes encoded within the major histocompatibility complex (MHC), especially the HLA. Detailed molecular typing of HLA-B13-B17 and Cw6 showed an association of PsA with CW*0602 allele, which is also found in psoriasis patients as compared to general population. Initial studies of HLA, identified the HLA-CW*0602 as the major locus associated with psoriasis and PsA (77). However, a study published in 2016 found that this locus is associated mainly with psoriasis (78). On the same line, a recent study, confirmed the above results demonstrating also that PsA was not associated with the HLA-CW*0602 (79). However, other specific HLA susceptibility genes were associated with various phenotypes of PsA. For example, the HLA-B*27:05:02 haplotype was reported to be associated with symmetrical sacroiliitis, enthesitis and dactylitis, whereas the HLA-B*08:01:01 – HLA-C*07:01:01 haplotypes were associated with asymmetrical sacroiliitis, joint fusion and deformities (10). Several reports have identified non-HLA loci associated with PsA including interleukin (IL) 23 receptor (IL-23R) and tumour necrosis factor (TNF) A1P3 (80, 81). These genetic associations indicate specific cytokine pathways, like TNF

and IL-23 in the pathogenesis of PsA. Indeed, IL-23R was the strongest associated gene in a large genome wide association study (GWAS) and has been implicated in enthesitis linked to IL-17 production, in disease animal models (80). GWAS have showed also that the majority of genetic susceptibility factors for PsA have been shown to overlap with those of psoriasis (10). Recently, an association with a polymorphism near MARK 14 independent from HLA-B27, together with a significant linkage with a susceptibility locus 13913 has been reported (82-84).

In another study, the role of micro RNA-146$^{\alpha}$ (MIR-146$^{\alpha}$) and its target IL-1 receptor associated kinase (IRAK-1) in PsA patients susceptibility was investigated. It was found that IRAK-1 rs 3027898 polymorphism was associated with disease predisposition (85).

In recent study, micro(mi)RNA assay was performed in peripheral blood cells of PsA patients as compared to healthy controls. It was shown that specific miRNA signatures were associated with PsA and active disease. These miRNAs target pathways relevant in PsA, such as TNF, MARK and WNT signaling pathways (86).

Infectious agents

A potential role of infectious agents in the occurrence of PsA has been suggested over decades. Among them, special attention was done in streptococcal upper respiratory tract infection which is linked with the development of guttate psoriasis. However, the presence of the bacteria or related antigens was not demonstrated in a large number of joints examined from patients with PsA (87, 88). Although the immunoreactivity to streptococcal antigens is accepted, it remains unclear if the infection triggers the disease or if the breakdown of the skin barriers, because of psoriasis, leads to streptococcal exposure and finally to a form of reactive arthritis.

A systematic review, regarding infections and PsA development, showed that the available data is inconsistent and more studies are needed, especially concerning streptococcal inflections and PsA. On the other hand, infection with human immunodeficiency virus (HIV) is associated with the development of psoriasis and PsA. This is particularly true in HIV endemic areas such as Zambia (89). The association of HIV infection and PsA is difficult to explain. It is postulated that the reduction of the number of CD4^{+} T cells from the HIV infection, changes the equilibrium between CD4^{+} helper and CD8^{+} suppressor cells and this plays an important role in the pathogenesis of PsA. However, infections promoted by profound immunodeficiency of HIV seems to be the most plausible explanation of PsA development in HIV patients. Finally, bacteria in the composition of fecal microbiota may responsible for PsA initiation and expression (90).

The interaction between PsA and gut microbiota has been primarily investigated with the SpA group (91). A high incidence of subclinical inflammation has been described in PsA patients (92). It is known that the immune system is able to affect the microbiota composition and on the other hand, microbiota can modulate the immune system (93). A decreased bacterial diversity in PsA patients as compared to psoriasis and healthy individuals has been observed (94). Gut dysbiosis might therefore be a potential modulator of autoimmunity. In a recent study of faecal bacteria in patients with PsA compared to healthy controls, there was found significant reduction in *Akkermansia*, *Ruminococcus* and *Pseudobutyrivibrio* species (95). Faecal supernatants also showed increased soluble IgA and decreased TNF superfamily member 11 (TNFSF11) concentrations, suggesting a link between the alteration of gut microbiota and immune system dysfunction. In addition, high levels of serum antibodies against *Saccharomyces cerevisiae* were found in PsA patients. This is probably related to Th17 cells involved in its pathogenesis, as supported by their role in antifungal immune response (96).

Age and gender

There are two forms of juvenile PsA based on the age of onset, while differences in clinical manifestations of the disease are now distinct. The first peak of the disease occurs around the age of 2 and the second at the end of childhood (9). Studies show that younger children are usually girls, more often have dactylitis and positive anti-nuclear antibodies. Finally, younger children are more likely to develop polyarthritis. In older children, disease appears more often with enthesitis, axial involvement and oligoarthritis (97). These differences have been correlated with the HLA*CW06 gene but the results were not conclusive as far as it concerns the association of genetic factors to different PsA clinical manifestations (98).

PsA in adults occurs in the 4th to 5th decades of age (9). Many investigators report an early and a late onset PsA based on the age of appearance of the clinical manifestations. Although there is no agreement of the age limit, most consider late-onset PsA after the age of 55–60 (99). Data suggests that there are clinical differences between early and late onset PsA, but these differences are not as distinct as in the juvenile form of the disease. Late onset PsA is correlated with more severe articular involvement and erosive disease (100, 101).

Gender has not been linked with the onset of the disease or the outcomes. Nevertheless, slight variations may appear depending on the clinical phenotype of the disease between men and women. Studies show that men often have a severe axial involvement, while female patients present more often peripheral arthritis. Female patients have also significantly impaired quality of life index (health assessment questionnaire) and a greater degree of limitation regarding physical activity (101). It should be noted that it has not been established whether these differences are due to the disease or other unrelated factors, such as a greater physical stress or more severe work at men. Finally, it should be noted that differences between the two genders are also related to the age of the appearance of psoriasis. Male patients with early-onset psoriasis, before the age of 40, exhibit shorter interval time from psoriasis onset to PsA development as compared to females (102).

Smoking

Smoking is an epidemiological factor that appears to have an effect on PSA patients but its role has not been fully

documented as there are conflicting results. Many studies show that smoking is associated with the occurrence of PsA among psoriasis patients (103, 104). To date, many investigators agree that patients with PSA who smoke at the beginning of the disease exhibit more pain, greater fatigue, more painful areas, more affected joints, and poor quality of life compared to those who do not smoke. It also appears that smokers have lower response rates to treatment with biological anti-tumour necrosis factor agents in comparison with non-smokers. However, there are studies that do not confirm the above results (105).

Trauma

Trauma, is a well-known risk factor for patients with psoriasis. For some people with psoriasis a minor scratch or even a mosquito bite can trigger the formation of psoriatic plaques (106). This phenomenon has been also studied in PsA patients. The close anatomical relationship between synovium and enthesis is especially prone to mechanical stress. Clinical imaging studies and experimental animal models of SpA, including TNF transgenic models and IL-23 overexpression systems are associated with primary enthesitis and disease spreading to adjacent joint structures including synovium and bone. This may explain why PsA develops in individuals with high body mass indices most typically in the fourth and fifth decades (107-109). The exact reason of this phenomenon is not well known. It seems that recurrent minor trauma causes local inflammation that leads to upregulation of adhesion molecules and pro-inflammatory cytokines. In this way, the inflammation may expand to the adjacent skin and joints (107, 110). Moreover, one study reported evidence of local trauma before the development of PsA in about 25% of patients (111). In a recent report, preceding bone and joint trauma was associated with PsA with a hazard ratio of 1,46 and 1,50 respectively (112). However, a history of trauma has been described in only a minority of patients with PsA.

Body mass index

In recent years, studies have linked obesity with clinical presentation and outcomes of PsA. Studies have demonstrated the increasing incidence and prevalence of psoriasis and PsA in patients with high Body Mass Index (BMI). This correlation appears to be particularly stronger for patients with psoriasis (113). A study from Italy confirms the specific correlation between BMI and psoriasis, showing the odds ratio of 1.6 to be increased to 1.9 in patients with high BMI (113). This correlation is also applicable in PsA patients. Other studies have demonstrated an increased prevalence and incidence of PsA in overweight patients (114, 115). An epidemiological study from the USA including a large number of patients, confirmed this correlation by demonstrating an increase in the relative risk of PSA occurrence with increasing BMI (116). Recent data also suggests that the increased BMI is associated with more severe disease and a significant impairment in quality of life of these patients (117). A recent meta-analysis has shown that when the BMI is increased, it is associated with a poor response to treatment with biological agents (118) but also an increase in adverse effects (119). Finally, it should be mentioned that the results are not conclusive and more research is needed, as there are other studies that did not find such correlations (120).

Diet

Several epidemiological studies suggest a potential protective effect of lifelong consumption of fish oil and psoriasis development. For example, Eskimos have a low incidence of psoriasis (121). This could be related with a high intake of very long chain n-3 fatty acids (especially eicosapentaenoic acid and decosahexaenoic acid) found mainly in seals and fish oils (103). Psoriasis is also uncommonly seen in Africans, probably due to genetic factors but, the dietary habits in Africans may provide another explanation (122). Maize, the staple diet in most parts of Africa is high in linoic acid but low in other polyunsaturated fatty acids and riboflavin. Linoic acid is a precursor of prostaglandin E2 (PGE2) and its high intake results in an increase production of PGE2. On the other hand, PGE2 is known to suppress cellular immunity resulting in decrease expression of psoriasis (123).

In a study from Italy, psoriasis appeared to be positively associated with BMI. However, a significant inverse correlation with psoriasis was observed for the intake of carrots, tomatoes, and fresh fruits (124). These results proved some evidence for a potential role of diet in psoriasis and could partly explain the geographical variations of the disease occurrence and its severity.

Conclusions

PsA is a chronic inflammatory arthritis characterised by great heterogeneity. The clinical manifestations may range from mild to severe. Precise outcomes regarding the epidemiology of the disease cannot be assessed. Epidemiological studies indicate a large variation regarding disease prevalence and incidence among different populations and geographic areas. This variability could be partly explained by a number of factors, such as the use of multiple and different classification criteria by the investigators. Geographic variability was also observed, could be related to different genetic backgrounds, environmental factors, climate, as well as different dietary and lifestyle habits between countries.

The implementation of unanimous classification criteria and the conformation by the scientific community could lead to a better inderstanding of the diseases epidemiology. Knowledge of PsA risk factors may improve the recognition of PsA among patients with psoriasis.

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