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Autoimmunity in 2016

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Abstract The number of peer-reviewed articles published during the 2016 solar year and retrieved using the "autoimmunity" key word remained stable while gaining a minimal edge among the immunology articles. Nonetheless, the quality of the publications has been rising significantly and, importantly, acquisitions have become available through scientific journals dedicated to immunology or autoimmunity. Major discoveries have been made in the fields of systemic lupus erythematosus, rheumatoid arthritis, autoimmunity of the central nervous system, vasculitis, and seronegative spondyloarthrithritides. Selected examples include the role of IL17-related genes and long noncoding RNAs in systemic lupus erythematosus or the effects of anti-pentraxin 3 (PTX3) in the treatment of this paradigmatic autoimmune condition. In the case of rheumatoid arthritis, there have been reports of the role of induced regulatory T cells (iTregs) or fibrocytes and T cell interactions with exciting implications. The large number of studies dealing with neuroimmunology pointed to Th17 cells, CD56(bright) NK cells, and low-level TLR2 ligands as involved in multiple sclerosis, along with a high salt intake or the micriobiome-derived Lipid 654. Lastly, we focused on the rare vasculitides to which numerous studies were devoted and suggested that unsuspected cell populations, including monocytes, mucosal-associated invariant T cells, and innate lymphoid cells, may be crucial to ANCA-associated manifestations. This brief and arbitrary discussion of the findings published in 2016 is representative of a promising background for developments that will enormously impact the work of laboratory scientists and physicians at an exponential rate.

Keywords Tolerance breakdown · Chronic inflammation · Genetics · Immunogenetics · Epigenetics · Microbiome · Th17 · ILC · Rheumatoid arthritis · Psoriatic arthritis · Spondyloarthritis · Systemic lupus erythematosus · Multiple sclerosis · Giant cell arteritis · ANCA-associated vasculitis

2016 and Autoimmunity

As we did for the previous years [1–8], we provide an overview of the publications dedicated to autoimmunity over the past solar year. In this view, 2016 was a stable year (+0.3%) in the absolute number of publications compared to 2015, with 2341 papers retrieved on PubMed (Fig. 1). The ratio of autoimmunity over immunology papers increased slightly in 2016, with a 5.1% prevalence (+0.1%) (Fig. 2).

To retrieve the most important publications regarding autoimmunity in 2016, we performed a literature research on PubMed in May 2017 among the major journals in the areas of immunology (*Nature Immunology*, *Journal of Immunology*, *Nature Medicine*, *Clinical Reviews in Allergy and Immunology*) and autoimmunity (*Autoimmunity Reviews*, *Journal of Autoimmunity*) and divided the articles in the most important clinical topics: systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), nervous system autoimmunity, vasculitis, and seronegative spondyloarthropathies. Indeed, this approach leads to an underrepresentation of other important diseases, such as inflammatory myopathies [9–17], antiphospholipid antibodies and syndrome [18–33], Sjogren syndrome [34–49], and systemic sclerosis [50–62]. Similarly, we overlooked papers regarding bone homeostasis, which were well represented in 2016 [63–68]. Taken altogether, we

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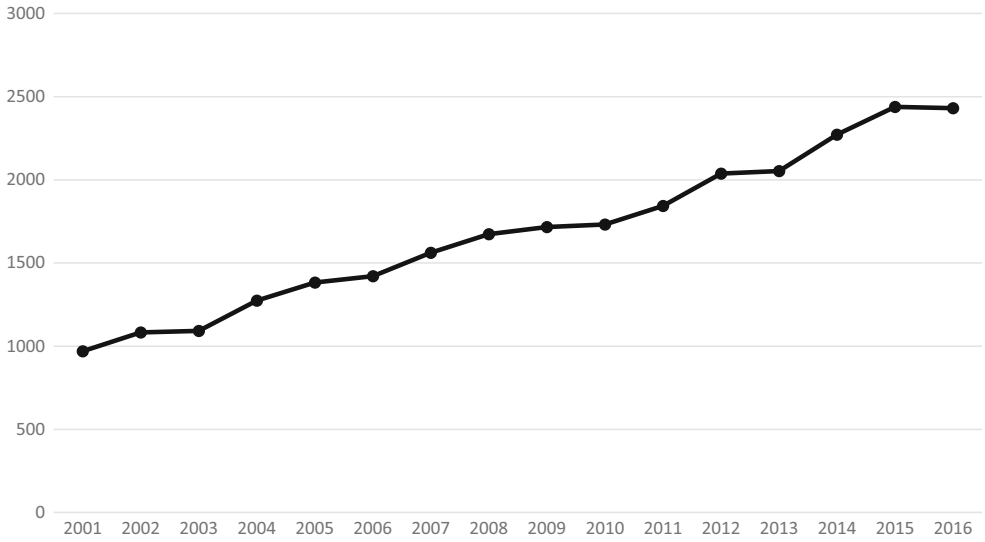

Fig. 1 Absolute number of articles retrieved in PubMed after searching for the word "autoimmunity"

should be aware that the choice of the articles to be briefly discussed is arbitrary and will lead to some missing references. Nonetheless, we will discuss the most recent findings regarding SLE [61, 69–113], RA [114–144], seronegative spondyloarthritis [121, 145–159], neuroimmunology [160–195], and vasculitis [196–224].

Systemic Lupus Erythematosus

Each year, systemic lupus erythematosus remains one of the most important topics for research, and we selected 42 articles dedicated to the disease in 2016, mostly dealing with the SLE pathophysiology and clinical manifestations. Last year, however, other original papers investigated the co-occurrence of other autoimmune diseases with SLE, in particular thyroid autoimmunity [104, 105], gastrointestinal [72, 98] as well as asthma and rhinitis [99].

It is well known that SLE is a multifactorial disease, where genetics and environmental factors interact. In the last year, the genetic factors predisposing to SLE were reviewed [101], as well as new polymorphisms conferring susceptibility to SLE were reported, in particular within the interleukin (IL)-17 family. In a Polish population of patients with SLE has been reported an increased frequency of the AG genotype as well as the G allele of the IL-17F rs763780 (OR = 3.947;

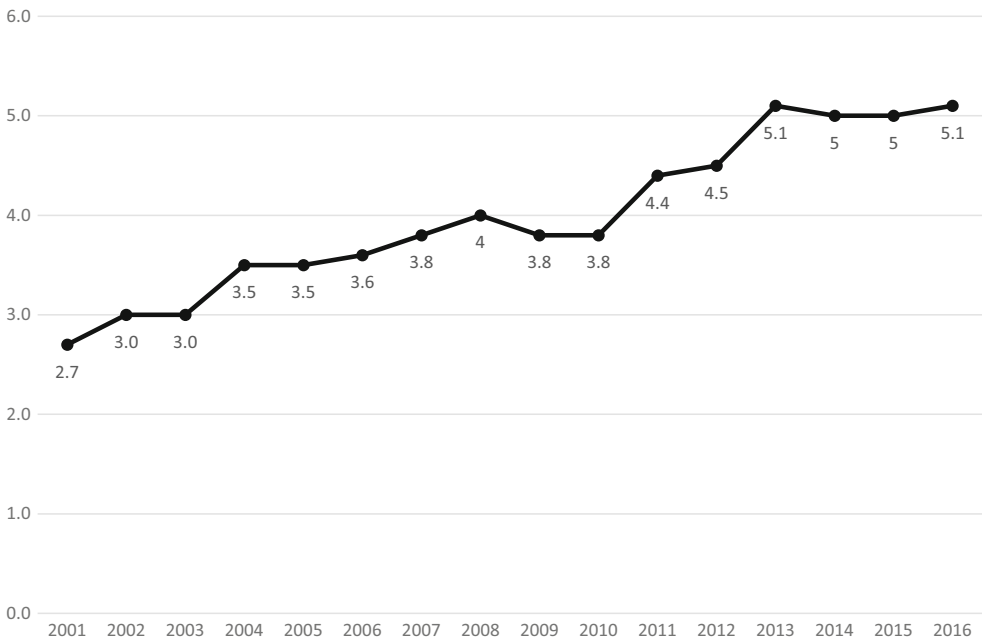

Fig. 2 Ratio of articles retrieved in PubMed after searching for the word "autoimmunity" vs "immunology"

$p = 0.001$ and OR = 3.538; $p = 0.002$, respectively) and in the rs1884444 TT genotype (OR = 138.1) and the rs1884444 T allele (OR = 2.176), $p < 0.001$ in both [92]. Moreover, the GGAGAA combined genotype and the GGA haplotype of IL-17A rs2275913, IL-17F rs763780, and rs2397084 can be considered risk factors for the development of SLE in Egyptian children [81]. Interestingly, it has been reported that having any children who carry DRB1*04:01 alleles inherited from the father influences a mother's subsequent risk of SLE (OR 1.9; 95% CI, 1.1–3.2) [71].

Epigenetics may reflect the interaction between genetics and the environment, and last year, this hypothesis was brilliantly reviewed [84, 86, 107]. Long noncoding RNAs (lncRNAs) have recently been identified to be tightly linked to diverse human diseases; lncRNA NEAT1 has been shown last year to contribute to the pathogenesis of SLE and to be abnormally increased in SLE patients and predominantly expressed in human monocytes, while being involved in the TLR4-mediated inflammatory process through affecting the activation of the late MAPK signaling pathway. NEAT1 furthermore correlated with clinical disease activity [108]. 5-Hydroxymethylcytosine (5-hmC), a newly discovered modified form of cytosine, is suspected to be an important epigenetic modification in embryonic development, cell differentiation, and cancer. Last year, it has been shown that increased 5-hmC levels are present in genomic DNA in CD4(+) T cells of patients with SLE compared with healthy controls, accompanied by the upregulated expression of the ten-eleven translocation TET2 and TET3, which can enzymatically convert 5-methylcytosine (5-mC) to 5-hmC [109].

The progression from tolerance breakdown and autoantibodies to SLE is not clear; however, it is supposed that an immune dysregulation involving multiple pathways contributes to SLE pathogenesis. The interferon (IFN) pathways are dysregulated in preclinical SLE, and multiple soluble mediators, including IL-5, IL-6, and IFN-gamma, are significantly elevated in SLE compared to controls more than 3.5 years before classification criteria are met, prior to or concurrent with autoantibody positivity. Moreover, innate cytokines, IFN-associated chemokines, and soluble tumor necrosis factor (TNF) superfamily mediators increase longitudinally in SLE patients approaching SLE classification, but not in controls. In particular, levels of B lymphocyte stimulator (BLyS) and a proliferation-inducing ligand (APRIL) are comparable in cases and controls until less than 10 months before the diagnosis is made [85]. From a clinical point of view, several reviews critically discussed the current understanding of lupus nephritis (LN) [78, 87] and neuropsychiatric SLE (NPSLE) [80, 82, 97, 100, 106]. More importantly, 2016 was the year in which positive pregnancy outcomes and SLE were extensively reported, resulting in long-awaited recommendations for patients with LN [83, 88, 89, 225].

SLE treatment remains a major area of interest, and an elegant study showed how anti-pentraxin 3 (PTX3) antibodies, which were associated with the absence of LN, delay LN and prolong survival of NZB/NZW F1 mice. In vitro observations suggest anti-PTX3 antibodies may dampen complement activation via their Fc fragment, likely hindering renal inflammation [77].

Rheumatoid Arthritis

Rheumatoid arthritis also remained a major research trend in 2016, with most articles dedicated to old and new treatment. In particular, biologic dose reduction risk was reviewed as cessation in established disease usually leads to disease flare; dose-tapering approaches for those achieving low disease activity often appear to be successful in the short term; however, tapering can be associated with a higher risk of losing disease control. Over relatively short periods of follow-up, a number of studies have shown no statistical difference in radiographic progression in patients tapering or discontinuing biologics. However, a Cochrane review found that radiographic and functional outcomes may be worse after TNF inhibitor discontinuation, and over long-term disease follow-up, flares have been associated with radiographic progression and worse patient-reported outcomes [117]. It is increasingly clear that obesity may modify the course of a rheumatologic disease and the clinical response to biotherapies based on a complex relationship of cytokines, hormones, growth factors, and intracellular regulators. A 2016 review article highlighted how obesity impairs the clinical response of RA to anti-TNF-alpha treatment, and this might be an effect limited to TNF-blocking agents, as preliminary studies are not confirming these findings for abatacept or tocilizumab [127].

In the case of new treatments, both positive and negative results have been achieved in 2016 [226]. The adoptive transfer of induced regulatory T cells (iTregs) may be effective in treating collagen-induced arthritis (CIA). Recently, it has been shown that the adoptive transfer of LAG3(+) Treg-of-B cells alleviates the joint severity as well as local and systemic inflammation, promoting IL-10 production in lymphocytes isolated from the spleen and draining lymph nodes [118]. Connective tissue growth factor (CTGF) contains four distinct modules connected in tandem, namely insulin-like growth factor-binding protein (IGFBP)-like, von Willebrand factor (vWF) type C repeat, thrombospondin type 1 (TSP-1) repeat, and carboxyl-terminal (CT) modules. In RA, it has been shown that the inhibition of each CTGF module affects M-CSF/RANKL-mediated osteoclastogenesis, furthermore, the angiogenesis of RA synoviocytes, and lastly that mAbs against CTGF neutralize the TNF-alpha enhanced the expression of CTGF and matrix metalloproteinase-3 (MMP3) in MH7A cells. Thus, a mAb against CTGF but also mAbs

against each specific module of CTGF might serve as potential therapeutic agents in the treatment of RA [135]. RA-derived adipose-derived mesenchymal stem cell (ASC) secretory and proliferative activities are similar to those of osteoarthritis ASCs, derived from the infrapatellar fat pad of the knee. Both RA and osteoarthritis ASCs inhibit PBMC proliferation and induce IL-10 production but upregulate IL-17A secretion and fail to limit the release of other proinflammatory mediators (TNF, IFN-gamma, and CCL5) by PBMCs. RA and osteoarthritis ASCs do not suppress activation marker expression on T cells and do not trigger Tregs expansion [141]. Thus, RA ASCs may have less immunomodulatory properties compared to previous data.

New single-nucleotide polymorphisms (SNPs) have been associated with the risk of RA, in particular the IL-6-174G > C SNP in the Asian population [122], thus stressing once again the role of IL6 in chronic inflammation and its consequences. Moreover, the CD247 gene SNP (rs858554) was found to be associated with RA Chinese patients, especially with ACCP+ and RF+ phenotype [132]. Conversely to what is reported in the Caucasian population, the PTPN22 1858C/T polymorphism is not associated with RA risk in Asian populations [136]. Genetic susceptibility to RA is often defined by the presence of a shared epitope (QKRAA, QRRAA, or RRRAA) at positions 70–74 in HLA-DRbeta1. However, the DRbeta1*01:01 and 01:02 contain the same QRRAA epitope, but differ considerably in their susceptibility to RA. A recent study shows that DRbeta1*01:01 and *01:02 both exhibited a 6.5-fold preference for citrullinated vimentin (66–78) compared to native vimentin, while DRbeta1*01:01 also exhibited a 1.7-fold preference for citrullinated alpha-enolase (11–25) and bound collagen (258–272), while DRbeta1*01:02 bound neither of these peptides. Conversely, DRbeta1*01:03 preferentially bound native vimentin (66–78) and alpha-enolase (11–25) over the citrullinated forms of these peptides, and also failed to bind collagen (258–272). When site-directed mutagenesis was performed to determine which amino acid residues were responsible for the differences between these alleles, mutating position 86 in DRbeta1*01:01 from glycine to the valine residue found in DRbeta1*01:02 eliminated binding of both citrullinated alpha-enolase (11–25) and collagen (258–272), thereby recapitulating the peptide-binding profile of DRbeta1*01:02 [139].

Recent findings support the role of fibrocytes and T cell interactions as one of the initiating factors in RA. A recent study shows that RA fibrocytes exhibit increased activation, denoted as elevated levels of phosphorylation of STAT3 and NF-kappaB, with a direct correlation with the number of circulating activated Th17 cells and Tregs [125]. A novel transcription factor, YY1, has been identified as a potential key player in RA pathogenesis, since YY1 was over-expressed in RA patients and CIA mice, and the blocking of YY1 action with YY1 shRNA lentivirus ameliorated disease progression in CIA mice [133].

One of the major issues in RA management is the recognition and treatment of comorbidities, of which cardiovascular disease remains one of the most important. A recent study on 11,782 patients with RA and 57,973 age- and sex-matched controls confirms that the prevalence of ischemic heart disease in RA patients is increased compared to that in controls (16.6 and 12.8% respectively, $p < 0.001$) [126]. Moreover, heart impairment as a result of chronic inflammation and secondarily myocardial fibrosis markedly participates in heart failure development. Early detection of heart dysfunction is based on echocardiographic detection of diastolic dysfunction resulting from myocardial inflammation and fibrosis. The impact of biological treatment on the progression of atherosclerosis and heart failure is still controversial [131]. Overall, cardiovascular risk assessment should be performed routinely in RA patients, since cardiovascular disease is the most common cause of mortality. However, current cardiovascular screening and management strategies underestimate the real risk associated with RA [134].

Seronegative Spondyloarthritis

The increasing knowledge of psoriatic arthritis (PsA) pathogenesis [146, 148, 156] and the emerging treatments [151] currently approved for the disease have caused a significant increase of papers devoted to PsA, as well as psoriasis [145] and other spondyloarthrithritides, i.e., ankylosing spondylitis. Albeit psoriasis and PsA are considered more an autoinflammatory than autoimmune disease, PsA and psoriasis are mainly T cell-driven diseases, and the IL-23/IL-17 axis plays a critical pathogenic role for both PsA and psoriasis, while biologics neutralizing IL-17A or IL-23/IL-12 are effective therapies for both diseases [153, 155]. Growing evidence shows that albeit PsA is considered and classified as a seronegative disease, autoantibodies may be present in patient sera; in particular, anti-CarP antibodies have been reported in sera from patients with active PsA [121]. Moreover, autoantibodies have been detected also in ankylosing spondylitis patients, particularly anti-CD74 antibodies [150].

Biomarkers are of pivotal importance in spondyloarthritis to determine disease severity and prognosis. CRP currently appears to be the best circulating measure for assessing disease activity, predicting structural progression and therapeutic response, while key molecules in the pathogenesis of the disease and essential therapeutic targets show only limited association with disease characteristics or disease progression [149]. Spondyloarthritis is classically associated with the HLA-B27 allele, which may play a role also in disease pathogenesis, and more recently, nonconventional heavy chain

forms of B27 expression in joints and lymphoid tissues from B27 TG(1) rats prior to the onset of arthritis are consistent with the hypothesis that they play a pathogenic role in spondyloarthritis [152].

Similarly to RA, also spondyloarthritis is associated with a significantly increased cardiovascular risk. The impact of treatment of psoriasis and PsA with anti-TNF on cardiovascular risk has been recently investigated in a meta-analysis, which included almost 50,000 patients. The results show that anti-TNF are associated with a lower risk of cardiovascular events, both compared to topical/photo treatment and methotrexate, and also to a decreased rate of mortality [157].

Neuroimmunology

As in previous years, the field of neuroimmunology has enormously increased in the number and quality of publications [191], with multiple sclerosis (MS) representing the most investigated disease [167, 174, 183, 189]. The importance of B cells in the pathogenesis [172, 173, 178] of the disease is supported by large lines of evidence also regarding B cell-depleting therapy [172, 185] and modulation of B cells by other treatments, as fingolimod [164]. However, increasing evidence shows that also T cells and especially Th17 [193] have a pivotal role in MS pathogenesis [160, 161, 187] and may represent possible future treatment strategies. It has been recently shown that mast cell-T cell co-localization in the meninges and CNS resident meningeal mast cells are an early source of caspase-1-dependent IL-1beta that licenses Th cells to produce GM-CSF and become encephalitogenic [168, 190]. Tregs have been reported to be defective in MS and correlate with disease phase, i.e., remission and relapse. Injections of expanded ex vivo autologous Tregs (eTregs) could be helpful in bringing up the level of Tregs in patients' blood, and could be applied as immunotherapy for MS [182].

CD56(bright) NK cells may have immunoregulatory functions, and the expansion of CD56(bright) NK cells has been associated with successful response to different treatments and to remission of MS during pregnancy. In MS, CD56(bright) NK cells, albeit being in equal number as healthy subjects, have significantly lower ability to inhibit autologous T cell proliferation, due to increased HLA-E expression on T cells from MS/CIS subjects, which could enhance the inhibitory effect mediated by NKG2A that is homogeneously expressed on CD56(bright) NK cells [180].

Recent studies investigated the role of toll-like receptors (TLRs) in MS and animal models. In the EAE model, increased IFN-gamma production and presence of NK cells and reduced expression of TLR-3 and TLR-9 were observed [170]. A microbiome-derived TLR2 ligand, Lipid 654 (L654), which is present in healthy human serum, is significantly decreased in the serum of MS patients. The administration of low-level TLR2 ligands in adoptive transfer EAE induces TLR2 tolerance and attenuates disease [162]. Novel mechanisms increasing MS susceptibility have been identified, in particular salt (NaCl) intake, which promotes pathogenic T cell responses contributing to CNS autoimmunity [177].

Vasculitis

A large amount of publications was devoted during 2016 to systemic vasculitides [196–221], despite the cumulative rare prevalence of these conditions. Most of the published articles were dedicated to vasculitis associated with anti-neutrophil cytoplasm antibody (ANCA) [196, 197, 199, 201, 207–210, 212, 220] and giant cell arteritis [204, 205, 213, 214, 216, 218].

In the field of ANCA-associated vasculitis, the most recent findings highlight the importance of new cell types, i.e., monocytes, which contain the major autoantibody antigens (i.e., PR3 and MPO) in lysosomes, can express these at the cell surface, and can respond to ANCA by producing pro-inflammatory and chemotactic cytokines and reactive oxygen species and by upregulating CD14 [197]. Other previously unsuspected cell types such as the mucosal-associated invariant T cells (MAIT) and the innate lymphoid cells (ILCs) have been studied in vasculitis based on their involvement in various inflammatory and autoimmune diseases, via their immunoregulatory functions at mucosal sites. Importantly, MAIT cells are significantly decreased in ANCA-associated vasculitis during both acute and remission phases [196]. The pathogenesis of ANCA-associated vasculitis was investigated using phenotyping, transcriptome and functional analyses of T cell populations to evaluate triggers of memory T cell expansion. The data showed an increased percentage of circulating CD4+ CD28−, CD8+CD28−, and CD4+CD161+ single-positive and CD4+CD8+ double-positive T cells in ANCA-associated vasculitis, while transcriptomic profiling of sorted T cell populations showed major differences between ANCA-associated vasculitis and healthy controls reflecting antigen- (bacteria, viruses, fungi) and cytokine-driven impact on T cell populations [209]. From a semantics standpoint, the name of eosinophilic granulomatosis with polyangiitis, previously known as Churg Strauss syndrome, was proposed to be refined, suggesting hypereosinophilic asthma with systemic (nonvasculitic) manifestations, as ANCA alone were reported to be insufficient to categorize patients with vasculitis features [199, 210].

One of the most important achievement of 2016 was the identification of IL-6 and IL-6 receptor inhibition as an effective therapy in giant cell arteritis [205], but also nonbiologic DMARDs have important beneficial effects, despite the limited evidence [213]. However, it was recently reported that

long-term remission after glucocorticoids withdrawal in an Italian cohort of patients with biopsy-proven GCA is achieved in 56% of patients [216].

A Wishlist for 2017

During 2016, new scenarios were proposed in the autoimmunity field and spanned from new pathogenetic mechanisms to biomarkers to treatment optimization. What results is ultimately the possibility that basic science and clinics do indeed concur to make a difference in the understanding and management of patients with autoimmune diseases, often by connecting lines of evidence which are not intuitively close. In the oncology field, this is ideally represented by the checkpoint inhibitors and the birth of a new avenue of treatments. It is quite obvious that the number of published articles may not well represent the activity and success in any research area, but we are convinced that new developments are expected to stem from these reports, as in previous years for the microbiota, microRNA, or monoclonal antibodies.

Compliance with Ethical Standards

Conflict of Interest The author declares that he has no conflict of interest.

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