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Autoimmunity in 2018

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Abstract
In the vast database of peer-reviewed articles, the number of 2018 papers published retrieved using the "autoimmunity" keyword remained unchanged compared with the brilliant results of 2017 while returning above a 5% share within the immunology field, after the brisk decrease of this ratio in 2017. As in the past 12 years, we have now searched PubMed for publications related to autoimmunity in the major immunology and autoimmunity peer-reviewed journals and provide here an arbitrary discussion of the major themes encountered. Once again, we are happy to notice that similarities between autoimmune diseases and the common mechanisms significantly outnumber differences. Some examples include data on Th17 cells, cytokines, or other mediators variably involved in the autoimmunity mechanisms such as BLIMP-1, IL-10, IFN, or NF-kB. The study of the microbiome remains central to autoimmunity development and data are being gathered in a growing number of conditions, similar to epigenetics and long non-coding RNA. In the cases of specific diseases, such as systemic lupus erythematosus, rheumatoid arthritis, or psoriatic arthritis, multiple encouraging findings underline the importance of a strict relationship between basic and clinical science to define new pathogenetic and therapeutic developments. Cumulatively, the present scenario of autoimmunity appears bright and should be regarded as one of the fastest growing in the scientific field of immunology, despite the enormous attention paid to cancer immune mechanisms. The parallel observation that the rheumatology therapeutic pipeline is second only to oncology increases the hopes that more and more patients will be satisfactorily treated in the near future.

Keywords Tolerance · Immune checkpoint inhibitors · Autoantibody · Th17

2018 and Autoimmunity

Every year since 2007, we performed a search for publications using the keywords "autoimmunity" and "immunology" to provide a brief overview of the findings and observe how the number of publications on these topics has increased over time, with a specific focus on systemic autoimmune rheumatic diseases and their disease mechanisms and therapies. Indeed, 2018 was a very productive year with a stable absolute number of publications compared with 2017, with 2934 papers retrieved on PubMed using "autoimmunity" as the search word (Fig. 1, panel a). This enormous increase was now associated with a less significant global raise in immunology as the ratio of "autoimmunity" over "immunology" hits returned to values observed until 2016, with a total 5.1% ratio (+ 0.8% compared with 2017) (Fig. 1, panel b).

In detail, our literature research was performed on PubMed in April 2019 and it was focused on 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).* We selected all articles in English published between January 1 and December 31, 2018, subdivided into relevant topics: systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), psoriatic arthritis (PsA), autoantibodies (autoAbs), common disease mechanisms, and therapies. As for previous years, also in 2018, we identified several articles focused on mechanisms involved in the autoimmune response related in particular to the identification of cytokines such as IL-17 and IL-12/23 which are today accepted as of major importance for specific therapies in PsA and skin psoriasis [1–14], while a better understanding is being reached for Th17 cells involved in disease mechanisms [15–18] or other immune cells and cytokines variably

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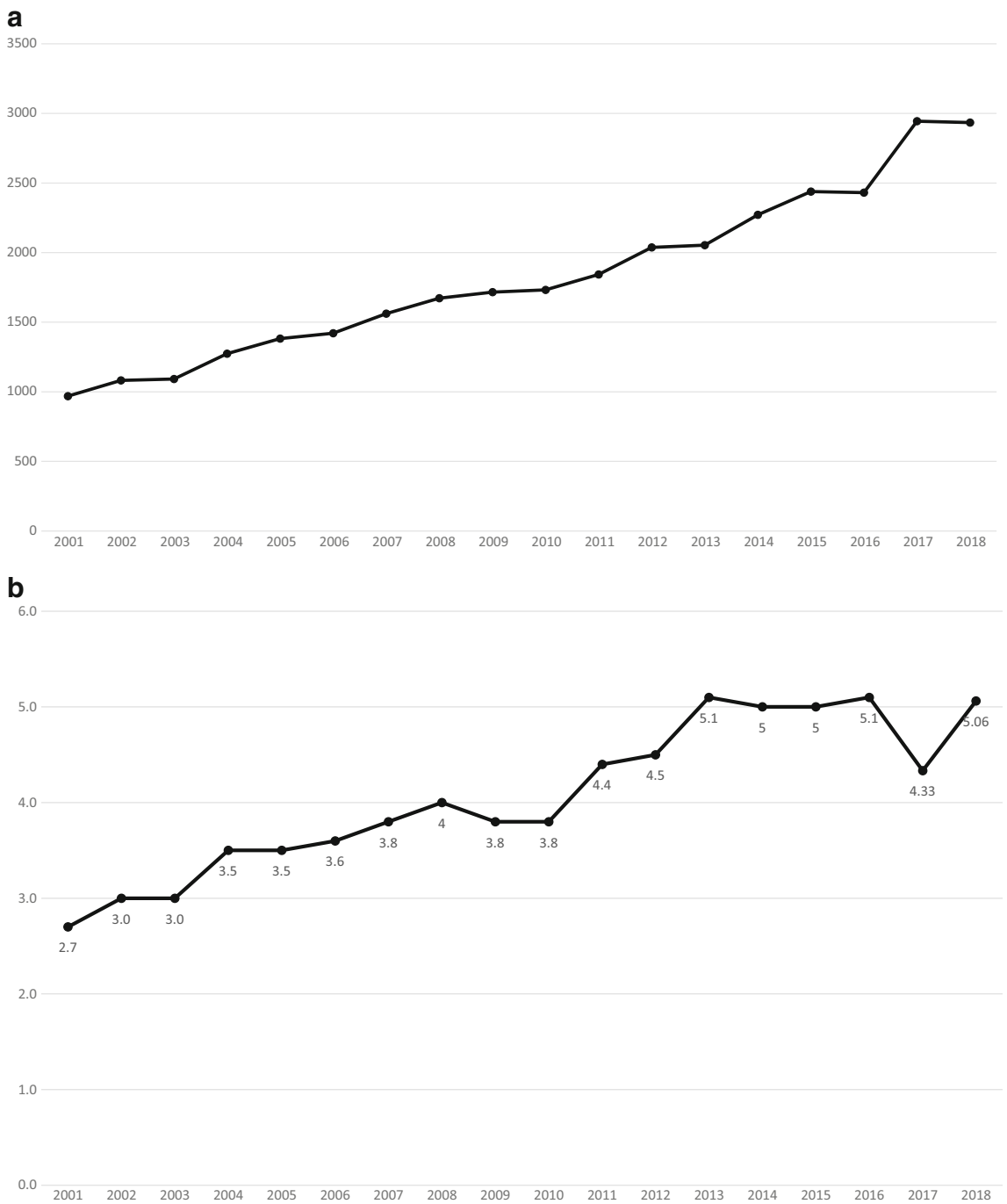

Fig. 1 Panel **a** Absolute number of articles retrieved in PubMed searching the word "autoimmunity" between 2001 and 2018. Panel **b** Ratio of articles retrieved in PubMed searching the word "autoimmunity" vs "immunology" in 2001 through 2018

involved in the autoimmunity mechanisms [19], as in the case of BLIMP-1 [20], IL-10 [21], IFN [22, 23], NF-kB [24], and others [25, 26]. Similarly, there remains major attention on the involvement of the microbiome in autoimmunity development [27–31], possibly via epigenetics [32, 33], long non-coding RNA [34], or other mechanisms [35–38].

Different from previous years and to reflect the wider importance of the chosen issues, we did not include additional important systemic rheumatic diseases such as systemic sclerosis [39–55], Sjogren's syndrome [28, 56–68], vasculitis [5, 27, 69–96], and organ-disease autoimmune conditions (i.e., type I diabetes, autoimmune liver diseases, gender medicine, and immunodeficiency) for which numerous articles were not selected for further discussion.

We are well aware that we will only address an arbitrary selection of the most relevant findings identified with the described search guidelines, even though this will certainly lead to missing points and missing references and we apologize in

advance to the authors who will not see their work recognized herein.

Systemic Lupus Erythematosus

As in past years, SLE has been widely investigated in 2018 and we identified several articles on the disease pathogenesis, its association with anti-phospholipid syndrome (APS), and the new advances in available therapies.

SLE is characterized by a strong immunological response [97], with the production of several serum autoAbs that are the hallmark of the condition [98]. The methods used to identify correctly autoAbs were studied [41] and anti-nuclear antibodies (ANA) were tested in several conditions including SLE; the data demonstrate that new techniques need to be associated with classical approaches to improve disease diagnosis through serum markers, as observed in dermatomyositis. Further, new autoAbs have been identified and are currently studied in SLE, as for the anti-TLR (Toll-like Receptors) and SMAD proteins that are involved in specific pathways responsible for a specific disease organ in SLE [99]. We are well aware that SLE pathogenesis is strictly dependent on immunocomplex formation with a potential application in the use of immunocomplex levels in the monitoring of activity of SLE and in the study of underlying FcgammaR biology [100]. Another significant set of articles was focused on the role of NET (neutrophil extracellular traps) in SLE, both in basal conditions and in association with the use of specific B cell targeting therapies such as rituximab and belimumab [101–103], demonstrating that the reduction of NET production by these therapies leads to a clinical benefit. Another target studied in 2018 for its therapeutic potential is represented by the JAK-STAT pathway, now blocked by new oral therapies recently approved for RA. In particular, the phosphorylated STAT5 component seems to be relevant in patients with SLE treated with methotrexate, and the study of these molecules in SLE may pave the way to the use of anti-JAK therapies also for this disease [104].

The second issue that was widely investigated is SLE comorbidities; indeed, SLE is a complex systemic autoimmune disease characterized by several manifestations that develop overtime and are often responsible of morbidity and mortality associated with SLE [105–107]. Among these complications, in 2018, a wide population of over 400 SLE patients with childhood onset disease was studied to identify cases affected by symptomatic polyautoimmunity [108] such as autoimmune thyroiditis and hepatitis and diabetes mellitus. This study demonstrates that approximately 10% of childhood SLE cases had symptomatic polyautoimmunity at diagnosis, but surprisingly they had a mild disease onset, thus suggesting a possible distinct genetic background with less severe disease and better long-term outcome. Another comorbidity frequently reported in SLE is atherosclerosis, which is more rapid and severe in SLE compared with matched controls despite an undefined underlying mechanism. In the past year, Ryu and Colleagues [109] showed that atherogenic dyslipidemia augments autoimmune follicular T helper cells and IgG2c production through the activation of TLR4- and IL-27-dependent pathways, thus increasing our knowledge of these disease mechanisms. As infections remain a large cause of mortality in SLE, several reports published in 2018 also refer to the need to improve the vaccination coverage in SLE, particularly for the anti-pneumococcal [110] and other vaccinations [111], and also the vaccination campaign for HPV infection may find strong application in young female SLE patients [112].

As for the novel therapeutic targets that are currently evaluated for SLE patients, in 2018 a comprehensive review on this topic was published by Felten and Colleagues [113], describing the ongoing trials with specific targeted therapies for a rationale selection of the personalized and optimal pharmacological agent. A new therapeutic target suggested in another report [56] is the TANK-binding kinase (TBK1) involved in the regulation of the interferon pathway in SLE, and its inhibition may reduce the expression of interferon stimulated genes involved in disease mechanisms.

Rheumatoid Arthritis

In 2018, we identified several articles related to novel findings in multiple aspects of RA. In particular, the pathogenic role of autoantibodies has been studied in association with the link between innate and adaptive immunity mediated by the anti-citrullinated peptide antibody [114]. On the one hand, active innate immune response induces the production of citrullinated auto-antigens that stimulate the autoimmunity and inflammatory environment to break self-tolerance and stimulate the proliferation of self-reactive T/B cells and the production of ACPA. On the other hand, ACPA produced by plasma B cells can interact with innate immunity to induce RA disease chronic manifestations [114]. Additional targets studied in RA included vimentin, a key role player in cellular interactions and functioning of the immune system involved in the pathogenesis of inflammation and many autoimmune diseases such as RA [115], and novel anti-inflammatory peptides based on chemokine-glycosaminoglycan interactions that are able to reduce leukocyte migration and disease severity in an in vitro model of RA [116]. Epigenetics may be involved in mechanisms through which IgG immunocomplexes sensitize human monocytes for inflammatory hyperactivity, not only via epigenetics but also via transcriptomic reprogramming [117] and we may expect that a growing number of studies will be reported in 2019.

As for novel therapeutic targets in RA, the role of neutrophils is perhaps the most fascinating, as neutrophils can

activate other immune cells, maintain inflammation, and lead to the destruction of cartilage and bone of the affected joint [118]. Because of this strong role in the pathophysiology of RA, several studies have tried to clarify the effects of therapeutic agents on this subtype of leukocyte. The now widely used "immune checkpoint inhibitors" for cancer are currently under strict evaluation in rheumatology because they are able to induce rheumatic manifestations as part of their side effects, but this is important to understand what is the correct management of these patients and to have a better insight of the mechanisms shared by cancer and autoimmune diseases [119].

Psoriatic Arthritis

As for other rheumatic diseases, the PsA pathogenesis remains unclear, despite the significant progress in recent years, in particular on the skin and gut microbiome and its influence on psoriatic disease pathogenesis [4]. In the case of skin psoriasis, altered keratinocyte signaling and predisposition to type 17 response activate a pathogenic IL-17 loop which induces disease manifestations [4]. Beside the skin microbiome, the human intestinal microbiome can modulate the mucosal and the systemic immune system, and it is possible that pro-inflammatory responses in the gut mucosa can induce and maintain specific autoimmune conditions [120]. A relationship is present between bacterial changes, immune system, and host genetic background, and a specific cell type represented by innate lymphoid cells (ILCs) may be responsible for homeostasis control and disease activity in PsA patients [12]. Similar to what observed for RA, epigenetics is also crucial to PsA, and the study of microRNA signature in PsA patients has led to the identification of miR23a as a key player of inflammation in PsA, as dysregulated miR-23a expression can induce synovial inflammation through enhanced synovial fibroblast activation, via PDE4B signaling, with a novel anti-inflammatory mechanism of PDE4 blockade with the approved use of apremilast [121]. The advances in the knowledge of pathogenic mechanisms in PsA have finally led to the development of specific therapies really effective for PsA [3], in particular those targeting the pathogenic TNF/IL-23/IL-17-pathway which are known to be safe and effective in the management of most PsA patients, and are currently included in the different sets of PsA management recommendations.

Autoantibodies

As a cornerstone in the pathogenesis and diagnosis of autoimmune diseases, the number of available serum autoAbs identified in research and with a putative role in clinical practice is constantly growing and also in 2018, several reports were published on new specificities identified in rheumatic diseases. For example, anti-ubiquitin carboxyl-terminal hydrolase L1 (UCHL1) IgG antibodies have been described in idiopathic steroid sensitive nephrotic syndrome (INS), which is the most frequent childhood kidney disease [122]. The published results showed that UCHL1 is a podocyte protein targeted by autoantibodies in a group of patients relapsing for this condition, and this supports a causative role of anti-UCHL1 autoantibodies in the development of INS. The vasoactive intestinal peptide (VIP) has been described in the pathogenesis of inflammatory cardiovascular diseases, as it can block proliferation and migration of smooth muscle cells and neo-intima formation in a mouse model of complete carotid ligation [123]. This peptide could be used as a new pharmaceutical agent to treat heart inflammation and atherosclerosis, also in the setting of systemic rheumatic diseases [123].

Therapy

During 2018, several reports were published on the first results of the use of biosimilars in rheumatic diseases, as the use of these therapies has been introduced and rapidly increasing over the past 5 years. Biosimilars have greatly improved the management of rheumatic patients with advantages for pharmaceutical industries, hospitals, and patients access to therapies worldwide. However, the development and the use of biosimilars are very variable and heterogenous, and sometimes, it is based on local rules and regulations; thus, it is necessary to describe in detail the current knowledge on biosimilars for rheumatic diseases [124].

New therapeutic targets are under evaluation and additional molecules have been described as potential target to stop inflammation and autoimmune response in rheumatic diseases. This is the case of NLRP3, a member of the NOD (nucleotide-binding domain)-like receptor family, that has been identified in various immune and non-immune cells. When activated, NLRP3 interacts with other proteins to form the NLRP3 inflammasome which regulates the activation of innate immunity in diseases such as SLE and RA. Thus, the knowledge of the biological features of NLRP3 and its link with the pathogenesis of autoimmune diseases is fundamental for its clinical implications and therapeutic potential [125]. Among promising cytokines, IL-35 has been studied extensively in systemic autoimmune rheumatic diseases because it activates signaling pathways that are able to regulate the differentiation, function of T, B cells, macrophages, and dendritic cells, and its expression has been found to be altered in SLE and RA [14]. The signaling lymphocytic activation molecule (SLAM) family is

composed of nine distinct receptors expressed on hematopoietic cells that are potential targets for inflammatory and autoimmune diseases, and they may also represent new potential therapeutic targets [126].

Cancer and Immunology

One of the most interesting aspects that are emerging in recent years is the link between cancer and immunology which is demonstrated by the immunological adverse events that we are facing nowadays with the use of immune checkpoint inhibitors (ICIs) [119]. ICIs therapy may induce autoimmune conditions (i.e., arthritis, myositis, sicca syndrome) [94, 127–129] in patients with preexisting autoimmunity, and these life-saving therapies may cause disease exacerbation that needs to be foreseen for prompt therapeutic options [94]. As in the case of patients affected by SLE, PD-1 (programmed cell death-1) and its receptors have been identified as key players etiology and pathogenesis as they crosstalk with molecules important for the maintenance of immune tolerance and development of distinct immune subsets [128]. PD-1 is one of the targets of the abovementioned ICIs and its link with SLE may explain the strict relationship existing between cancer, cancer therapies, and immunological mechanisms leading to autoimmune adverse events of these therapies, as suggested also by other lines of evidence [33, 79, 94, 119, 130–134].

Neuroimmunology

Among new reports on neuroimmunology, multiple sclerosis (MS) represents a chronic inflammatory autoimmune condition affecting the central nervous system (CNS) characterized by demyelination and neurodegeneration [135] and the predominant subject of 2018 research [9, 51, 131, 135–137]. The immune system in MS is severely deregulated in the balance between pro- and anti-inflammatory cells, and this affects both the innate and the adaptive immune system, causing the severe clinical features of MS. A novel treatment currently used and studied for MS is dimethyl fumarate (DMF), characterized by neuroprotective and immunomodulatory features that are able to restore the immune balance and the tolerogenic capacity of the immune system in particular in MS patients affected by the relapsing-remitting form [135]. It has been possible to obtain new therapies on MS and its specific forms thanks to the continuous research on this topic, which is demonstrated for example by the interplay between Th17 cells and gamma-delta T cells in animal models of experimental autoimmune encephalomyelitis (EAE) which is the animal model for MS [9]. These results demonstrate that it may be possible to use current anti-IL-17 therapies (approved for psoriatic disease) also in MS patients with the relapsing-remitting form and other T cell mediated autoimmune diseases.

What to Expect in 2019

Once again, we are particularly excited by the available and growing evidence on the pathogenesis of autoimmune diseases which appear more relevant than ever to being implied in the clinical management of patients in a truly translational fashion and the quality of the research continues to grow with more elegant solutions expected to be reported in different areas based on translational research. We welcome a more strict relationship between basic and clinical science which will allow a more rapid advancement of the understanding and treatment of autoimmune diseases.

Compliance with Ethical Standards

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

Ethical Approval and Informed Consent No ethical approval for the local IRB was needed for this review article.

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