David V Serreze

List of Publications by Year in Descending Order

64 papers	2,791 citations	27 h-index	52 g-index
66 ext. papers	3,123 ext. citations	6.6 avg, IF	4.39 L-index

#	Paper	IF	Citations
64	Aberrant type 1 immunity drives susceptibility to mucosal fungal infections. *Science*, **2021**, 371,	33.3	31
63	Response to Comments on "Aberrant type 1 immunity drives susceptibility to mucosal fungal infections". *Science*, **2021**, 373, eabi8835	33.3	1
62	Decreased pancreatic acinar cell number in type 1 diabetes. *Diabetologia*, **2020**, 63, 1418-1423	10.3	25
61	The CD137 Ligand Is Important for Type 1 Diabetes Development but Dispensable for the Homeostasis of Disease-Suppressive CD137 FOXP3 Regulatory CD4 T Cells. *Journal of Immunology*, **2020**, 204, 2887-2899	5.3	2
60	CD70 Inversely Regulates Regulatory T Cells and Invariant NKT Cells and Modulates Type 1 Diabetes in NOD Mice. *Journal of Immunology*, **2020**, 205, 1763-1777	5.3	0
59	T Cells from NOD- Mice Target Both Pancreatic and Neuronal Tissue. *Journal of Immunology*, **2020**, 205, 2026-2038	5.3	1
58	Toll-Like Receptor 7 Is Required for Lacrimal Gland Autoimmunity and Type 1 Diabetes Development in Male Nonobese Diabetic Mice. *International Journal of Molecular Sciences*, **2020**, 21,	6.3	2
57	Combined congenic mapping and nuclease-based gene targeting for studying allele-specific effects of Tnfrsf9 within the Idd9.3 autoimmune diabetes locus. *Scientific Reports*, **2019**, 9, 4316	4.9	3
56	CD11c Cells Are Gatekeepers for Lymphocyte Trafficking to Infiltrated Islets During Type 1 Diabetes. *Frontiers in Immunology*, **2019**, 10, 99	8.4	14
55	Interleukin-27 Is Essential for Type 1 Diabetes Development and Sjögren Syndrome-like Inflammation. *Cell Reports*, **2019**, 29, 3073-3086.e5	10.6	17
54	Improved Murine MHC-Deficient HLA Transgenic NOD Mouse Models for Type 1 Diabetes Therapy Development. *Diabetes*, **2018**, 67, 923-935	0.9	7
53	HLA-B*39:06 Efficiently Mediates Type 1 Diabetes in a Mouse Model Incorporating Reduced Thymic Insulin Expression. *Journal of Immunology*, **2018**, 200, 3353-3363	5.3	11
52	A Hypermorphic Allele Contributes to Impaired Thymic Deletion of Autoreactive Diabetogenic CD8 T Cells in NOD Mice. *Journal of Immunology*, **2018**, 201, 1907-1917	5.3	8
51	Genetic and Small Molecule Disruption of the AID/RAD51 Axis Similarly Protects Nonobese Diabetic Mice from Type 1 Diabetes through Expansion of Regulatory B Lymphocytes. *Journal of Immunology*, **2017**, 198, 4255-4267	5.3	18
50	Interferon-Limits Diabetogenic CD8 T-Cell Effector Responses in Type 1 Diabetes. *Diabetes*, **2017**, 66, 710-721	0.9	14
49	Transient BAFF Blockade Inhibits Type 1 Diabetes Development in Nonobese Diabetic Mice by Enriching Immunoregulatory B Lymphocytes Sensitive to Deletion by Anti-CD20 Cotherapy. *Journal of Immunology*, **2017**, 199, 3757-3770	5.3	13
48	Autoimmune manifestations in aged mice arise from early-life immune dysregulation. *Science Translational Medicine*, **2016**, 8, 361ra137	17.5	27

47	B-lymphocytes expressing an Ig specificity recognizing the pancreatic β-cell autoantigen peripherin are potent contributors to type 1 diabetes development in NOD mice. *Diabetes*, **2016**, 65, 1977-1987	0.9	14
46	Bridging Mice to Men: Using HLA Transgenic Mice to Enhance the Future Prediction and Prevention of Autoimmune Type 1 Diabetes in Humans. *Methods in Molecular Biology*, **2016**, 1438, 137-51	1.4	7
45	The Presence and Preferential Activation of Regulatory T Cells Diminish Adoptive Transfer of Autoimmune Diabetes by Polyclonal Nonobese Diabetic (NOD) T Cell Effectors into NSG versus NOD-scid Mice. *Journal of Immunology*, **2015**, 195, 3011-9	5.3	10
44	Genetic Analysis of Substrain Divergence in Non-Obese Diabetic (NOD) Mice. *G3: Genes, Genomes, Genetics*, **2015**, 5, 771-5	3.2	15
43	Compensatory mechanisms allow undersized anchor-deficient class I MHC ligands to mediate pathogenic autoreactive T cell responses. *Journal of Immunology*, **2014**, 193, 2135-46	5.3	18
42	In vivo detection of peripherin-specific autoreactive B cells during type 1 diabetes pathogenesis. *Journal of Immunology*, **2014**, 192, 3080-90	5.3	15
41	Subcongenic analyses reveal complex interactions between distal chromosome 4 genes controlling diabetogenic B cells and CD4 T cells in nonobese diabetic mice. *Journal of Immunology*, **2012**, 189, 1406-17	5.3	11
40	Mouse models for the study of autoimmune type 1 diabetes: a NOD to similarities and differences to human disease. *Seminars in Immunopathology*, **2011**, 33, 67-87	12	137
39	Calcium insufficiency accelerates type 1 diabetes in vitamin D receptor-deficient nonobese diabetic (NOD) mice. *Endocrinology*, **2011**, 152, 4620-9	4.8	11
38	Unmasking genes in a type 1 diabetes-resistant mouse strain that enhances pathogenic CD8 T-cell responses. *Diabetes*, **2011**, 60, 1354-9	0.9	2
37	Loss of intra-islet CD20 expression may complicate efficacy of B-cell-directed type 1 diabetes therapies. *Diabetes*, **2011**, 60, 2914-21	0.9	55
36	Prevention of "Humanized" diabetogenic CD8 T-cell responses in HLA-transgenic NOD mice by a multipeptide coupled-cell approach. *Diabetes*, **2011**, 60, 1229-36	0.9	30
35	Invariant natural killer T-cell control of type 1 diabetes: a dendritic cell genetic decision of a silver bullet or Russian roulette. *Diabetes*, **2010**, 59, 423-32	0.9	23
34	A recombinant heavy chain antibody approach blocks ART2 mediated deletion of an iNKT cell population that upon activation inhibits autoimmune diabetes. *Journal of Autoimmunity*, **2010**, 34, 145-54	15.5	27
33	Bridging mice to men: using HLA transgenic mice to enhance the future prediction and prevention of autoimmune type 1 diabetes in humans. *Methods in Molecular Biology*, **2010**, 602, 119-34	1.4	6
32	Rapid identification of MHC class I-restricted antigens relevant to autoimmune diabetes using retrogenic T cells. *Journal of Immunological Methods*, **2008**, 335, 106-15	2.5	8
31	Through regulation of TCR expression levels, an Idd7 region gene(s) interactively contributes to the impaired thymic deletion of autoreactive diabetogenic CD8+ T cells in nonobese diabetic mice. *Journal of Immunology*, **2008**, 180, 3250-9	5.3	18
30	Idd9/11 genetic locus regulates diabetogenic activity of CD4 T-cells in nonobese diabetic (NOD) mice. *Diabetes*, **2008**, 57, 3273-80	0.9	19

29	Interleukin-2 gene variation impairs regulatory T cell function and causes autoimmunity. *Nature Genetics*, **2007**, 39, 329-37	36.3	306
28	"Humanized" HLA transgenic NOD mice to identify pancreatic beta cell autoantigens of potential clinical relevance to type 1 diabetes. *Annals of the New York Academy of Sciences*, **2007**, 1103, 103-11	6.5	16
27	In vivo cytotoxicity of insulin-specific CD8+ T-cells in HLA-A*0201 transgenic NOD mice. *Diabetes*, **2007**, 56, 2551-60	0.9	42
26	Cellular expression requirements for inhibition of type 1 diabetes by a dominantly protective major histocompatibility complex haplotype. *Diabetes*, **2007**, 56, 424-30	0.9	12
25	Invasion of the killer BSs in type 1 diabetes. *Frontiers in Bioscience - Landmark*, **2007**, 12, 2183-93	2.8	8
24	HLA-A*0201-restricted T cells from humanized NOD mice recognize autoantigens of potential clinical relevance to type 1 diabetes. *Journal of Immunology*, **2006**, 176, 3257-65	5.3	94
23	Partial versus full allogeneic hemopoietic chimerization is a preferential means to inhibit type 1 diabetes as the latter induces generalized immunosuppression. *Journal of Immunology*, **2006**, 177, 6675-84	5.3	20
22	Genes within the Idd5 and Idd9/11 diabetes susceptibility loci affect the pathogenic activity of B cells in nonobese diabetic mice. *Journal of Immunology*, **2006**, 177, 7033-41	5.3	28
21	The good turned ugly: immunopathogenic basis for diabetogenic CD8+ T cells in NOD mice. *Immunological Reviews*, **2005**, 204, 250-63	11.3	65
20	Major histocompatibility complex-linked diabetes susceptibility in NOD/Lt mice: subcongenic analysis localizes a component of Idd16 at the H2-D end of the diabetogenic H2(g7) complex. *Diabetes*, **2005**, 54, 1603-6	0.9	29
19	Diabetes acceleration or prevention by a coxsackievirus B4 infection: critical requirements for both interleukin-4 and gamma interferon. *Journal of Virology*, **2005**, 79, 1045-52	6.6	57
18	Activated NKT cells inhibit autoimmune diabetes through tolerogenic recruitment of dendritic cells to pancreatic lymph nodes. *Journal of Immunology*, **2005**, 174, 1196-204	5.3	118
17	Requirement for both H-2Db and H-2Kd for the induction of diabetes by the promiscuous CD8+ T cell clonotype AI4. *Journal of Immunology*, **2004**, 173, 2530-41	5.3	24
16	Individual nonobese diabetic mice exhibit unique patterns of CD8+ T cell reactivity to three islet antigens, including the newly identified widely expressed dystrophia myotonica kinase. *Journal of Immunology*, **2004**, 173, 6727-34	5.3	103
15	Enhanced pathogenicity of diabetogenic T cells escaping a non-MHC gene-controlled near death experience. *Journal of Immunology*, **2004**, 173, 3791-800	5.3	38
14	B cell selection defects underlie the development of diabetogenic APCs in nonobese diabetic mice. *Journal of Immunology*, **2004**, 172, 5086-94	5.3	65
13	MHC class II molecules play a role in the selection of autoreactive class I-restricted CD8 T cells that are essential contributors to type 1 diabetes development in nonobese diabetic mice. *Journal of Immunology*, **2004**, 172, 871-9	5.3	35
12	Paralytic autoimmune myositis develops in nonobese diabetic mice made Th1 cytokine-deficient by expression of an IFN-gamma receptor beta-chain transgene. *Journal of Immunology*, **2003**, 170, 2742-9	5.3	7

11	Genetic disassociation of autoimmunity and resistance to costimulation blockade-induced transplantation tolerance in nonobese diabetic mice. *Journal of Immunology*, **2003**, 171, 185-95	5.3	58
10	Identification of the beta cell antigen targeted by a prevalent population of pathogenic CD8+ T cells in autoimmune diabetes. *Proceedings of the National Academy of Sciences of the United States of America*, **2003**, 100, 8384-8	11.5	313
9	Functional evidence for the mediation of diabetogenic T cell responses by HLA-A2.1 MHC class I molecules through transgenic expression in NOD mice. *Proceedings of the National Academy of Sciences of the United States of America*, **2002**, 99, 13753-8	11.5	62
8	During the early prediabetic period in NOD mice, the pathogenic CD8(+) T-cell population comprises multiple antigenic specificities. *Clinical Immunology*, **2002**, 105, 332-41	9	35
7	The preferential ability of B lymphocytes to act as diabetogenic APC in NOD mice depends on expression of self-antigen-specific immunoglobulin receptors. *European Journal of Immunology*, **2002**, 32, 3657-66	6.1	109
6	Inhibition of autoimmune diabetes in nonobese diabetic mice by transgenic restoration of H2-E MHC class II expression: additive, but unequal, involvement of multiple APC subtypes. *Journal of Immunology*, **2001**, 167, 2404-10	5.3	27
5	Th1 to Th2 cytokine shifts in nonobese diabetic mice: sometimes an outcome, rather than the cause, of diabetes resistance elicited by immunostimulation. *Journal of Immunology*, **2001**, 166, 1352-9	5.3	149
4	Identification of a CD8 T cell that can independently mediate autoimmune diabetes development in the complete absence of CD4 T cell helper functions. *Journal of Immunology*, **2000**, 164, 3913-8	5.3	125
3	Emv30null NOD-scid mice. An improved host for adoptive transfer of autoimmune diabetes and growth of human lymphohematopoietic cells. *Diabetes*, **1995**, 44, 1392-8	0.9	50
2	Immunostimulation circumvents diabetes in NOD/Lt mice. *Journal of Autoimmunity*, **1989**, 2, 759-76	15.5	174
1	Human CD8+ T-cells Require Glycolysis to Elicit Effector Function		1