

# Nancy I Kerkvliet

## List of Publications by Year in descending order

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37  
papers

2,334  
citations

236925

25  
h-index

330143

37  
g-index

40  
all docs

40  
docs citations

40  
times ranked

2009  
citing authors

#	ARTICLE	IF	CITATIONS
1	Cutting Edge: Activation of the Aryl Hydrocarbon Receptor by 2,3,7,8-Tetrachlorodibenzo-p-dioxin Generates a Population of CD4+CD25+ Cells with Characteristics of Regulatory T Cells. <i>Journal of Immunology</i> , 2005, 175, 4184-4188.	0.8	200
2	Recent advances in understanding the mechanisms of TCDD immunotoxicity. <i>International Immunopharmacology</i> , 2002, 2, 277-291.	3.8	192
3	Dioxin and immune regulation. <i>Annals of the New York Academy of Sciences</i> , 2010, 1183, 25-37.	3.8	161
4	AHR-mediated immunomodulation: The role of altered gene transcription. <i>Biochemical Pharmacology</i> , 2009, 77, 746-760.	4.4	156
5	Aryl Hydrocarbon Receptor-Deficient Mice Generate Normal Immune Responses to Model Antigens and Are Resistant to TCDD-Induced Immune Suppression. <i>Toxicology and Applied Pharmacology</i> , 2001, 171, 157-164.	2.8	148
6	Activation of aryl hydrocarbon receptor by TCDD prevents diabetes in NOD mice and increases Foxp3 <sup>+</sup> T cells in pancreatic lymph nodes. <i>Immunotherapy</i> , 2009, 1, 539-547.	2.0	139
7	Modeling of the Aryl Hydrocarbon Receptor (AhR) Ligand Binding Domain and Its Utility in Virtual Ligand Screening to Predict New AhR Ligands. <i>Journal of Medicinal Chemistry</i> , 2009, 52, 5635-5641.	6.4	120
8	T Lymphocytes Are Direct, Aryl Hydrocarbon Receptor (AhR)-Dependent Targets of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD): AhR Expression in Both CD4+ and CD8+ T Cells Is Necessary for Full Suppression of a Cytotoxic T Lymphocyte Response by TCDD. <i>Toxicology and Applied Pharmacology</i> , 2002, 185, 146-152.	2.8	103
9	TCDD, FICZ, and Other High Affinity AhR Ligands Dose-Dependently Determine the Fate of CD4+ T Cell Differentiation. <i>Toxicological Sciences</i> , 2018, 161, 310-320.	3.1	101
10	The Anti-Inflammatory Drug Leflunomide Is an Agonist of the Aryl Hydrocarbon Receptor. <i>PLoS ONE</i> , 2010, 5, e13128.	2.5	99
11	Functional Characterization and Gene Expression Analysis of CD4+CD25+ Regulatory T Cells Generated in Mice Treated with 2,3,7,8-Tetrachlorodibenzo-p-dioxin. <i>Journal of Immunology</i> , 2008, 181, 2382-2391.	0.8	92
12	2,3,7,8-Tetrachlorodibenzo-p-dioxin Affects the Number and Function of Murine Splenic Dendritic Cells and Their Expression of Accessory Molecules. <i>Toxicology and Applied Pharmacology</i> , 2001, 171, 117-125.	2.8	78
13	2,3,7,8-Tetrachlorodibenzo-p-dioxin Suppresses Tumor Necrosis Factor- $\alpha$ and Anti-CD40-Induced Activation of NF- $\kappa$ B/Rel in Dendritic Cells: p50 Homodimer Activation Is Not Affected. <i>Molecular Pharmacology</i> , 2002, 62, 722-728.	2.3	69
14	The Aryl Hydrocarbon Receptor Mediates Leflunomide-Induced Growth Inhibition of Melanoma Cells. <i>PLoS ONE</i> , 2012, 7, e40926.	2.5	64
15	A Structural Switch between Agonist and Antagonist Bound Conformations for a Ligand-Optimized Model of the Human Aryl Hydrocarbon Receptor Ligand Binding Domain. <i>Biology</i> , 2014, 3, 645-669.	2.8	45
16	Distribution and Behavior of the Ah Receptor in Murine T Lymphocytes. <i>Toxicology and Applied Pharmacology</i> , 1996, 138, 275-284.	2.8	44
17	Benzimidazoisquinolines: A New Class of Rapidly Metabolized Aryl Hydrocarbon Receptor (AhR) Ligands that Induce AhR-Dependent Tregs and Prevent Murine Graft-Versus-Host Disease. <i>PLoS ONE</i> , 2014, 9, e88726.	2.5	43
18	Suppression of cytotoxic T lymphocyte activity by 2,3,7,8-tetrachlorodibenzo-p-dioxin occurs in vivo, but not in vitro, and is independent of corticosterone elevation. <i>Toxicology</i> , 1995, 97, 105-112.	4.2	41

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19	Influence of 2,3,7,8-Tetrachlorodibenzo-p-dioxin on the Antigen-Presenting Activity of Dendritic Cells. <i>Toxicological Sciences</i> , 2003, 72, 103-112.	3.1	37
20	2,3,7,8-Tetrachlorodibenzo-p-dioxin Alters the Differentiation of Alloreactive CD8 <sup>+</sup> T Cells Toward a Regulatory T Cell Phenotype by a Mechanism that is Dependent on Aryl Hydrocarbon Receptor in CD4 <sup>+</sup> T Cells. <i>Journal of Immunotoxicology</i> , 2008, 5, 81-91.	1.7	37
21	Activation of the Aryl Hydrocarbon Receptor by 10-Cl-BBQ Prevents Insulinitis and Effector T Cell Development Independently of Foxp3 <sup>+</sup> Regulatory T Cells in Nonobese Diabetic Mice. <i>Journal of Immunology</i> , 2016, 196, 264-273.	0.8	37
22	Early Consequences of 2,3,7,8-Tetrachlorodibenzo-p-dioxin Exposure on the Activation and Survival of Antigen-Specific T Cells. <i>Toxicological Sciences</i> , 2004, 82, 129-142.	3.1	31
23	Aryl Hydrocarbon Receptor-Mediated Perturbations in Gene Expression during Early Stages of CD4 <sup>+</sup> T-cell Differentiation. <i>Frontiers in Immunology</i> , 2012, 3, 223.	4.8	28
24	Functional alterations in CD11b <sup>+</sup> Gr-1 <sup>+</sup> cells in mice injected with allogeneic tumor cells and treated with 2,3,7,8-tetrachlorodibenzo-p-dioxin. <i>International Immunopharmacology</i> , 2003, 3, 553-570.	3.8	27
25	AhR activation increases IL-2 production by alloreactive CD4 <sup>+</sup> T cells initiating the differentiation of mucosal homing Tim3 <sup>+</sup> Lag3 <sup>+</sup> Tr1 cells. <i>European Journal of Immunology</i> , 2017, 47, 1989-2001.	2.9	26
26	Is chronic AhR activation by rapidly metabolized ligands safe for the treatment of immune-mediated diseases?. <i>Current Opinion in Toxicology</i> , 2017, 2, 72-78.	5.0	25
27	2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD) Directly Enhances the Maturation and Apoptosis of Dendritic Cells In Vitro. <i>Journal of Immunotoxicology</i> , 2005, 1, 159-166.	1.7	24
28	CTL Hyporesponsiveness Induced by 2,3,7,8-Tetrachlorodibenzo-p-dioxin: Role of Cytokines and Apoptosis. <i>Toxicology and Applied Pharmacology</i> , 2000, 166, 214-221.	2.8	22
29	Î³-Glutamyltranspeptidase knockout mice as a model for understanding the consequences of diminished glutathione on T cell-dependent immune responses. <i>European Journal of Immunology</i> , 2000, 30, 1902-1910.	2.9	20
30	Dietary Indole-3-Carbinol Activates AhR in the Gut, Alters Th17-Microbe Interactions, and Exacerbates Insulinitis in NOD Mice. <i>Frontiers in Immunology</i> , 2020, 11, 606441.	4.8	19
31	Expression of constitutively-active aryl hydrocarbon receptor in T-cells enhances the down-regulation of CD62L, but does not alter expression of CD25 or suppress the allogeneic CTL response. <i>Journal of Immunotoxicology</i> , 2009, 6, 194-203.	1.7	17
32	Discovery and Mechanistic Characterization of a Select Modulator of AhR-regulated Transcription (SMAhRT) with Anti-cancer Effects. <i>Apoptosis: an International Journal on Programmed Cell Death</i> , 2021, 26, 307-322.	4.9	17
33	The aryl hydrocarbon receptor is required for induction of p21 <sup>cip1/waf1</sup> expression and growth inhibition by SU5416 in hepatoma cells. <i>Oncotarget</i> , 2017, 8, 25211-25225.	1.8	15
34	Identification of a Raloxifene Analog That Promotes AhR-Mediated Apoptosis in Cancer Cells. <i>Biology</i> , 2017, 6, 41.	2.8	13
35	Suppression of Acute Graft-Versus-Host Response by TCDD Is Independent of the CTLA-4-IFN-Î³-IDO pathway. <i>Toxicological Sciences</i> , 2013, 135, 81-90.	3.1	11
36	Hydroville Curriculum Project: A Successful Toxicology Outreach Program for High School Teachers and Students in Oregon. <i>Comments on Modern Biology Part B, Comments on Toxicology</i> , 2002, 8, 209-217.	0.2	2

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37	Immunotoxicology of Dioxins and Related Chemicals. , 2005, , 299-328.		0