

B Paige Lawrence

List of Publications by Year in descending order

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Version: 2024-02-01

72
papers

2,636
citations

136950

32
h-index

206112

48
g-index

76
all docs

76
docs citations

76
times ranked

2896
citing authors

#	ARTICLE	IF	CITATIONS
1	The Aryl Hydrocarbon Receptor Modulates T Follicular Helper Cell Responses to Influenza Virus Infection in Mice. <i>Journal of Immunology</i> , 2022, 208, 2319-2330.	0.8	7
2	Dung biomass smoke exposure impairs resolution of inflammatory responses to influenza infection. <i>Toxicology and Applied Pharmacology</i> , 2022, 450, 116160.	2.8	4
3	Refinement of coding SNPs in the human aryl hydrocarbon receptor gene using ISNPranker: An integrative-SNP ranking web-tool. <i>Computational Biology and Chemistry</i> , 2021, 90, 107416.	2.3	2
4	DNA Methylation Patterns in CD4+ T Cells of Naïve and Influenza A Virus-Infected Mice Developmentally Exposed to an Aryl Hydrocarbon Receptor Ligand. <i>Environmental Health Perspectives</i> , 2021, 129, 017007.	6.0	5
5	Exposure to a mixture of 23 chemicals associated with unconventional oil and gas operations alters immune response to challenge in adult mice. <i>Journal of Immunotoxicology</i> , 2021, 18, 105-117.	1.7	1
6	Thyroid Disrupting Chemicals in Mixture Perturb Thymocyte Differentiation in <i>Xenopus laevis</i> Tadpoles. <i>Toxicological Sciences</i> , 2021, 181, 262-272.	3.1	8
7	Recovery scenario and immunity in COVID-19 disease: A new strategy to predict the potential of reinfection. <i>Journal of Advanced Research</i> , 2021, 31, 49-60.	9.5	27
8	The Aryl Hydrocarbon Receptor Modulates Murine Hematopoietic Stem Cell Homeostasis and Influences Lineage-Biased Stem and Progenitor Cells. <i>Stem Cells and Development</i> , 2021, 30, 970-980.	2.1	9
9	Environmental Lead Exposure and Influenza and Respiratory Syncytial Virus Diagnoses in Young Children: A Test-Negative Case-Control Study. <i>International Journal of Environmental Research and Public Health</i> , 2020, 17, 7625.	2.6	2
10	Early life exposures shape the CD4+ T cell transcriptome, influencing proliferation, differentiation, and mitochondrial dynamics later in life. <i>Scientific Reports</i> , 2019, 9, 11489.	3.3	6
11	Blood Lead Concentrations and Antibody Levels to Measles, Mumps, and Rubella among U.S. Children. <i>International Journal of Environmental Research and Public Health</i> , 2019, 16, 3035.	2.6	7
12	The Ancestral Environment Shapes Antiviral CD8+ T Cell Responses across Generations. <i>IScience</i> , 2019, 20, 168-183.	4.1	15
13	Developmental exposure to chemicals associated with unconventional oil and gas extraction alters immune homeostasis and viral immunity of the amphibian <i>Xenopus</i> . <i>Science of the Total Environment</i> , 2019, 671, 644-654.	8.0	15
14	Genome-Wide Transcriptional Analysis Reveals Novel AhR Targets That Regulate Dendritic Cell Function during Influenza A Virus Infection. <i>ImmunoHorizons</i> , 2019, 3, 219-235.	1.8	16
15	Environmental exposures are hidden modifiers of anti-viral immunity. <i>Current Opinion in Toxicology</i> , 2018, 10, 54-59.	5.0	11
16	Aryl hydrocarbon receptor signaling modulates antiviral immune responses: ligand metabolism rather than chemical source is the stronger predictor of outcome. <i>Scientific Reports</i> , 2018, 8, 1826.	3.3	37
17	Developmental Exposure to a Mixture of 23 Chemicals Associated With Unconventional Oil and Gas Operations Alters the Immune System of Mice. <i>Toxicological Sciences</i> , 2018, 163, 639-654.	3.1	12
18	Environmental cues received during development shape dendritic cell responses later in life. <i>PLoS ONE</i> , 2018, 13, e0207007.	2.5	11

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19	Conditional deletion of Ahr alters gene expression profiles in hematopoietic stem cells. PLoS ONE, 2018, 13, e0206407.	2.5	18
20	Water Contaminants Associated With Unconventional Oil and Gas Extraction Cause Immunotoxicity to Amphibian Tadpoles. Toxicological Sciences, 2018, 166, 39-50.	3.1	21
21	Long term effects of carbaryl exposure on antiviral immune responses in <i>Xenopus laevis</i> . Chemosphere, 2017, 170, 169-175.	8.2	21
22	A Birth Cohort Study of Maternal and Infant Serum PCB-153 and DDE Concentrations and Responses to Infant Tuberculosis Vaccination. Environmental Health Perspectives, 2016, 124, 813-821.	6.0	36
23	The Oxygen Environment at Birth Specifies the Population of Alveolar Epithelial Stem Cells in the Adult Lung. Stem Cells, 2016, 34, 1396-1406.	3.2	28
24	Demographic, Reproductive, and Dietary Determinants of Perfluorooctane Sulfonic (PFOS) and Perfluorooctanoic Acid (PFOA) Concentrations in Human Colostrum. Environmental Science & Technology, 2016, 50, 7152-7162.	10.0	19
25	Influence of Early-Life Environmental Exposures on Immune Function Across the Life Span. , 2016, , 21-54.		1
26	Developmental Activation of the AHR Increases Effector CD4 ⁺ T Cells and Exacerbates Symptoms in Autoimmune Disease-Prone <i>Gnaq</i> ^{+/+} Mice. Toxicological Sciences, 2015, 148, 555-566.	3.1	19
27	Cumulative neonatal oxygen exposure predicts response of adult mice infected with influenza A virus. Pediatric Pulmonology, 2015, 50, 222-230.	2.0	17
28	Influenza A virus-dependent remodeling of pulmonary clock function in a mouse model of COPD. Scientific Reports, 2015, 5, 9927.	3.3	63
29	Neonatal hyperoxia leads to persistent alterations in NK responses to influenza A virus infection. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2015, 308, L76-L85.	2.9	13
30	Activation of the aryl hydrocarbon receptor during development enhances the pulmonary CD4 ⁺ T-cell response to viral infection. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2015, 309, L305-L313.	2.9	19
31	Linking the Aryl Hydrocarbon Receptor with Altered DNA Methylation Patterns and Developmentally Induced Aberrant Antiviral CD8 ⁺ T Cell Responses. Journal of Immunology, 2015, 194, 4446-4457.	0.8	51
32	Differential Consequences of Two Distinct AhR Ligands on Innate and Adaptive Immune Responses to Influenza A Virus. Toxicological Sciences, 2014, 137, 324-334.	3.1	50
33	Effects of Developmental Activation of the AhR on CD4 ⁺ T-Cell Responses to Influenza Virus Infection in Adult Mice. Environmental Health Perspectives, 2014, 122, 1201-1208.	6.0	29
34	Neonatal oxygen exposure alters airway hyperresponsiveness but not the response to allergen challenge in adult mice. Pediatric Allergy and Immunology, 2014, 25, 180-186.	2.6	23
35	Sex-specific enhanced behavioral toxicity induced by maternal exposure to a mixture of low dose endocrine-disrupting chemicals. NeuroToxicology, 2014, 45, 121-130.	3.0	70
36	New insights into the role of the aryl hydrocarbon receptor in the function of CD11c ⁺ cells during respiratory viral infection. European Journal of Immunology, 2014, 44, 1685-1698.	2.9	27

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37	Negative effects of low dose atrazine exposure on the development of effective immunity to FV3 in <i>Xenopus laevis</i> . <i>Developmental and Comparative Immunology</i> , 2014, 47, 52-58.	2.3	32
38	New insights into the aryl hydrocarbon receptor as a modulator of host responses to infection. <i>Seminars in Immunopathology</i> , 2013, 35, 615-626.	6.1	53
39	Neither direct nor developmental exposure to bisphenol A alters the severity of experimental inflammatory colitis in mice. <i>Journal of Immunotoxicology</i> , 2013, 10, 334-340.	1.7	15
40	Novel Cellular Targets of AhR Underlie Alterations in Neutrophilic Inflammation and Inducible Nitric Oxide Synthase Expression during Influenza Virus Infection. <i>Journal of Immunology</i> , 2013, 190, 659-668.	0.8	45
41	Neonatal hyperoxia alters the host response to influenza A virus infection in adult mice through multiple pathways. <i>American Journal of Physiology - Lung Cellular and Molecular Physiology</i> , 2013, 305, L282-L290.	2.9	44
42	The Effects of Maternal Exposure to Bisphenol A on Allergic Lung Inflammation into Adulthood. <i>Toxicological Sciences</i> , 2012, 130, 82-93.	3.1	90
43	Lung development and the host response to influenza A virus are altered by different doses of neonatal oxygen in mice. <i>American Journal of Physiology - Lung Cellular and Molecular Physiology</i> , 2012, 302, L1078-L1087.	2.9	39
44	You AhR what you eat?. <i>Nature Immunology</i> , 2012, 13, 117-119.	14.5	17
45	Memory CD8 ⁺ T Cells Are Sufficient To Alleviate Impaired Host Resistance to Influenza A Virus Infection Caused by Neonatal Oxygen Supplementation. <i>Vaccine Journal</i> , 2012, 19, 1432-1441.	3.1	18
46	Neonatal Oxygen Increases Sensitivity to Influenza A Virus Infection in Adult Mice by Suppressing Epithelial Expression of Ear1. <i>American Journal of Pathology</i> , 2012, 181, 441-451.	3.8	37
47	Developmental Exposure to Bisphenol A Modulates Innate but Not Adaptive Immune Responses to Influenza A Virus Infection. <i>PLoS ONE</i> , 2012, 7, e38448.	2.5	59
48	Environmental toxicants and the developing immune system: A missing link in the global battle against infectious disease?. <i>Reproductive Toxicology</i> , 2011, 31, 327-336.	2.9	102
49	Aryl hydrocarbon receptor activation during pregnancy, and in adult nulliparous mice, delays the subsequent development of DMBA-induced mammary tumors. <i>International Journal of Cancer</i> , 2011, 128, 1509-1523.	5.1	30
50	Activation of the Aryl Hydrocarbon Receptor During Pregnancy in the Mouse Alters Mammary Development Through Direct Effects on Stromal and Epithelial Tissues1. <i>Biology of Reproduction</i> , 2011, 84, 1094-1102.	2.7	19
51	Aryl Hydrocarbon Receptor Activation Reduces Dendritic Cell Function during Influenza Virus Infection. <i>Toxicological Sciences</i> , 2010, 116, 514-522.	3.1	66
52	Activation of the Aryl Hydrocarbon Receptor during Different Critical Windows in Pregnancy Alters Mammary Epithelial Cell Proliferation and Differentiation. <i>Toxicological Sciences</i> , 2009, 111, 151-162.	3.1	29
53	The aryl hydrocarbon receptor is a modulator of anti-viral immunity. <i>Biochemical Pharmacology</i> , 2009, 77, 642-653.	4.4	70
54	TCDD exposure disrupts mammary epithelial cell differentiation and function. <i>Reproductive Toxicology</i> , 2009, 28, 11-17.	2.9	24

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55	The Aryl Hydrocarbon Receptor Affects Distinct Tissue Compartments during Ontogeny of the Immune System. <i>Toxicological Sciences</i> , 2008, 102, 160-170.	3.1	59
56	Aryl Hydrocarbon Receptor Targets Pathways Extrinsic to Bone Marrow Cells to Enhance Neutrophil Recruitment during Influenza Virus Infection. <i>Toxicological Sciences</i> , 2008, 102, 89-99.	3.1	36
57	Neonatal Hyperoxia Enhances the Inflammatory Response in Adult Mice Infected with Influenza A Virus. <i>American Journal of Respiratory and Critical Care Medicine</i> , 2008, 177, 1103-1110.	5.6	110
58	Activation of the aryl hydrocarbon receptor is essential for mediating the anti-inflammatory effects of a novel low-molecular-weight compound. <i>Blood</i> , 2008, 112, 1158-1165.	1.4	96
59	Aryl Hydrocarbon Receptor Activation during Influenza Virus Infection Unveils a Novel Pathway of IFN- β Production by Phagocytic Cells. <i>Journal of Immunology</i> , 2007, 179, 247-255.	0.8	59
60	Environmental Toxins as Modulators of Antiviral Immune Responses. <i>Viral Immunology</i> , 2007, 20, 231-242.	1.3	13
61	Protection against Lethal Challenge with <i>Streptococcus pneumoniae</i> Is Conferred by Aryl Hydrocarbon Receptor Activation but Is Not Associated with an Enhanced Inflammatory Response. <i>Infection and Immunity</i> , 2006, 74, 5679-5686.	2.2	33
62	Aryl Hydrocarbon Receptor Activation Impairs the Priming but Not the Recall of Influenza Virus-Specific CD8+ T Cells in the Lung. <i>Journal of Immunology</i> , 2006, 177, 5819-5828.	0.8	66
63	A Dose-Response Study of the Effects of Prenatal and Lactational Exposure to TCDD on the Immune Response to Influenza A Virus. <i>Journal of Toxicology and Environmental Health - Part A: Current Issues</i> , 2006, 69, 445-463.	2.3	37
64	Increased mortality associated with TCDD exposure in mice infected with influenza A virus is not due to severity of lung injury or alterations in Clara cell protein content. <i>Chemico-Biological Interactions</i> , 2005, 155, 181-190.	4.0	15
65	Activation of the aryl hydrocarbon receptor increases pulmonary neutrophilia and diminishes host resistance to influenza A virus. <i>American Journal of Physiology - Lung Cellular and Molecular Physiology</i> , 2005, 289, L111-L124.	2.9	75
66	A Novel Effect of Dioxin: Exposure during Pregnancy Severely Impairs Mammary Gland Differentiation. <i>Toxicological Sciences</i> , 2004, 78, 248-257.	3.1	101
67	Activation of the Aryl Hydrocarbon Receptor Diminishes the Memory Response to Homotypic Influenza Virus Infection but Does Not Impair Host Resistance. <i>Toxicological Sciences</i> , 2004, 79, 304-314.	3.1	39
68	Developmental Exposure to the Potent Aryl Hydrocarbon Receptor Agonist 2,3,7,8-Tetrachlorodibenzo-p-Dioxin Impairs the Cell-Mediated Immune Response to Infection with Influenza A Virus, but Enhances Elements of Innate Immunity. <i>Journal of Immunotoxicology</i> , 2004, 1, 103-112.	1.7	39
69	Fewer CTL, not enhanced NK cells, are sufficient for viral clearance from the lungs of immunocompromised mice. <i>Cellular Immunology</i> , 2003, 226, 54-64.	3.0	44
70	T cell receptor transgenic mice provide novel insights into understanding cellular targets of TCDD: suppression of antibody production, but not the response of CD8+ T cells, during infection with influenza virus. <i>Toxicology and Applied Pharmacology</i> , 2003, 192, 275-286.	2.8	14
71	Examining the relationship between impaired host resistance and altered immune function in mice treated with TCDD. <i>Toxicology</i> , 2003, 188, 15-28.	4.2	56
72	Exposure to 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) Renders Influenza Virus-Specific CD8+ T Cells Hyporesponsive to Antigen. <i>Toxicological Sciences</i> , 2003, 74, 74-84.	3.1	36