Alvaro Puga

List of Publications by Year
in descending order

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Version: 2024-02-01

85	6,816	66343	60623
papers	citations	42 h-index	81 g-index

88	88	88	5858
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	REGULATION OF GENE EXPRESSION BY REACTIVE OXYGEN. Annual Review of Pharmacology and Toxicology, 1999, 39, 67-101.	9.4	980
2	The aryl hydrocarbon receptor cross-talks with multiple signal transduction pathways. Biochemical Pharmacology, 2009, 77, 713-722.	4.4	368
3	Aryl hydrocarbon receptor, cell cycle regulation, toxicity, and tumorigenesis. Journal of Cellular Biochemistry, 2005, 96, 1174-1184.	2.6	287
4	Aromatic Hydrocarbon Receptor Interaction with the Retinoblastoma Protein Potentiates Repression of E2F-dependent Transcription and Cell Cycle Arrest. Journal of Biological Chemistry, 2000, 275, 2943-2950.	3.4	273
5	Constitutive Activation of the Aromatic Hydrocarbon Receptor. Molecular and Cellular Biology, 1998, 18, 525-535.	2.3	216
6	The transcriptional signature of dioxin in human hepatoma HepG2 cells. Biochemical Pharmacology, 2000, 60, 1129-1142.	4.4	212
7	Dioxin Induces Expression of c-fosand c-junProto-Oncogenes and a Large Increase in Transcription Factor AP-1. DNA and Cell Biology, 1992, 11, 269-281.	1.9	174
8	Role of the aryl hydrocarbon receptor in cell cycle regulation. Chemico-Biological Interactions, 2002, 141, 117-130.	4.0	161
9	Ah receptor signals cross-talk with multiple developmental pathways. Biochemical Pharmacology, 2005, 69, 199-207.	4.4	158
10	Induction of cellular oxidative stress by aryl hydrocarbon receptor activation. Chemico-Biological Interactions, 2002, 141, 77-95.	4.0	155
11	Activation of mitogen-activated protein kinases (MAPKs) by aromatic hydrocarbons: role in the regulation of aryl hydrocarbon receptor (AHR) function. Biochemical Pharmacology, 2002, 64, 771-780.	4.4	154
12	The Aryl Hydrocarbon Receptor Functions as a Tumor Suppressor of Liver Carcinogenesis. Cancer Research, 2010, 70, 212-220.	0.9	154
13	Dioxin Causes a Sustained Oxidative Stress Response in the Mouse. Biochemical and Biophysical Research Communications, 1998, 253, 44-48.	2.1	144
14	Mitochondrial reactive oxygen production is dependent on the aromatic hydrocarbon receptor. Free Radical Biology and Medicine, 2002, 33, 1268-1278.	2.9	141
15	The Aryl Hydrocarbon Receptor Displaces p300 from E2F-dependent Promoters and Represses S Phase-specific Gene Expression. Journal of Biological Chemistry, 2004, 279, 29013-29022.	3.4	139
16	Chromium Cross-Links Histone Deacetylase 1-DNA Methyltransferase 1 Complexes to Chromatin, Inhibiting Histone-Remodeling Marks Critical for Transcriptional Activation. Molecular and Cellular Biology, 2007, 27, 7089-7101.	2.3	138
17	Dioxin induces transcription of fos and jun genes by ah receptor-dependent and -independent pathways. Toxicology and Applied Pharmacology, 1996, 141, 238-247.	2.8	122
18	Sustained Increase in Intracellular Free Calcium and Activation of Cyclooxygenase-2 Expression in Mouse Hepatoma Cells Treated with Dioxin. Biochemical Pharmacology, 1997, 54, 1287-1296.	4.4	120

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19	Ten nucleotide differences, five of which cause amino acid changes, are associated with the Ah receptor locus polymorphism of C57BL/6 and DBA/2 mice. Pharmacogenetics and Genomics, 1993, 3, 312-321.	5.7	114
20	Human AH locus polymorphism and cancer: inducibility of CYP1A1 and other genes by combustion products and dioxin. Pharmacogenetics and Genomics, 1991, 1, 68-78.	5.7	111
21	HDAC1 bound to the Cyp1a1 promoter blocks histone acetylation associated with Ah receptor-mediated trans-activation. Biochimica Et Biophysica Acta Gene Regulatory Mechanisms, 2007, 1769, 569-578.	2.4	111
22	Dioxin Exposure Is an Environmental Risk Factor for Ischemic Heart Disease. Cardiovascular Toxicology, 2001, 1, 285-298.	2.7	110
23	The Aryl Hydrocarbon Receptor Binds to E2F1 and Inhibits E2F1-induced Apoptosis. Molecular Biology of the Cell, 2008, 19, 3263-3271.	2.1	110
24	Activation of transcription factors activator protein-1 and nuclear factor-Î¹B by 2,3,7,8-Tetrachlorodibenzo-p-dioxin. Biochemical Pharmacology, 2000, 59, 997-1005.	4.4	100
25	Ligand-Independent Regulation of Transforming Growth Factor Î²1 Expression and Cell Cycle Progression by the Aryl Hydrocarbon Receptor. Molecular and Cellular Biology, 2007, 27, 6127-6139.	2.3	96
26	Chromium Inhibits Transcription from Polycyclic Aromatic Hydrocarbon-inducible Promoters by Blocking the Release of Histone Deacetylase and Preventing the Binding of p300 to Chromatin. Journal of Biological Chemistry, 2004, 279, 4110-4119.	3.4	95
27	Expression of genes in the TGF-Î² signaling pathway is significantly deregulated in smooth muscle cells from aorta of aryl hydrocarbon receptor knockout mice. Toxicology and Applied Pharmacology, 2004, 194, 79-89.	2.8	93
28	Genomewide Analysis of Aryl Hydrocarbon Receptor Binding Targets Reveals an Extensive Array of Gene Clusters that Control Morphogenetic and Developmental Programs. Environmental Health Perspectives, 2009, 117, 1139-1146.	6.0	90
29	2,3,7,8-Tetrachlorodibenzo-p-dioxin Blocks Androgen-Dependent Cell Proliferation of LNCaP Cells through Modulation of pRB Phosphorylation. Molecular Pharmacology, 2004, 66, 502-511.	2.3	85
30	A Critical Role For MAP Kinases in the Control of Ah Receptor Complex Activity. Toxicological Sciences, 2004, 82, 80-87.	3.1	72
31	Restoration of retinoblastoma mediated signaling to Cdk2 results in cell cycle arrest. Oncogene, 2000, 19, 1857-1867.	5.9	69
32	Fitting a xenobiotic receptor into cell homeostasis: How the dioxin receptor interacts with TGFÎ² signaling. Biochemical Pharmacology, 2009, 77, 700-712.	4.4	67
33	The MurineCyp1a-1Gene Negatively Regulates Its Own Transcription and that of Other Members of the Aromatic Hydrocarbon-Responsive [Ah] Gene Battery. Molecular Endocrinology, 1990, 4, 1773-1781.	3.7	58
34	Role of the aryl hydrocarbon receptor in cell cycle regulation. Toxicology, 2002, 181-182, 171-177.	4.2	56
35	Disruption of dioxin-inducible phase I and phase II gene expression patterns by cadmium, chromium, and arsenic. Molecular Carcinogenesis, 2000, 28, 225-235.	2.7	55
36	Arsenite-Induced Aryl Hydrocarbon Receptor Nuclear Translocation Results in Additive Induction of Phase I Genes and Synergistic Induction of Phase II Genes. Molecular Pharmacology, 2005, 68, 336-346.	2.3	55

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37	Ah Receptor Activation by Dioxin Disrupts Activin, BMP, and WNT Signals During the Early Differentiation of Mouse Embryonic Stem Cells and Inhibits Cardiomyocyte Functions. Toxicological Sciences, 2016, 149, 346-357.	3.1	54
38	Sex- and tissue-specific methylome changes in brains of mice perinatally exposed to lead. NeuroToxicology, 2015, 46, 92-100.	3.0	52
39	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
40	Butylhydroquinone Protects Cells Genetically Deficient in Glutathione Biosynthesis from Arsenite-Induced Apoptosis Without Significantly Changing Their Prooxidant Status. Toxicological Sciences, 2005, 87, 365-384.	3.1	50
41	Different Global Gene Expression Profiles in Benzo[<I>a</I>]Pyrene- and Dioxin-Treated Vascular Smooth Muscle Cells of AHR-Knockout and Wild-Type Mice. Cardiovascular Toxicology, 2004, 4, 47-74.	2.7	49
42	Induction of Oxidative Stress Responses by Dioxin and other Ligands of the Aryl Hydrocarbon Receptor. Dose-Response, 2005, 3, dose-response.0.	1.6	46
43	Disruption of Aryl Hydrocarbon Receptor Homeostatic Levels during Embryonic Stem Cell Differentiation Alters Expression of Homeobox Transcription Factors that Control Cardiomyogenesis. Environmental Health Perspectives, 2013, 121, 1334-1343.	6.0	45
44	Disruption of Ah Receptor Signaling during Mouse Development Leads to Abnormal Cardiac Structure and Function in the Adult. PLoS ONE, 2015, 10, e0142440.	2.5	42
45	Repression of the Aryl Hydrocarbon Receptor Is Required to Maintain Mitotic Progression and Prevent Loss of Pluripotency of Embryonic Stem Cells. Stem Cells, 2016, 34, 2825-2839.	3.2	40
46	Dioxin Exposure Disrupts the Differentiation of Mouse Embryonic Stem Cells into Cardiomyocytes. Toxicological Sciences, 2010, 115, 225-237.	3.1	38
47	Ah Receptor Signaling Controls the Expression of Cardiac Development and Homeostasis Genes. Toxicological Sciences, 2015, 147, 425-435.	3.1	38
48	Recruitment of CREB1 and Histone Deacetylase 2 (HDAC2) to the Mouse Ltbp-1 Promoter Regulates its Constitutive Expression in a Dioxin Receptor-dependent Manner. Journal of Molecular Biology, 2008, 380, 1-16.	4.2	36
49	The aryl hydrocarbon receptor at the crossroads of multiple signaling pathways. Exs, 2009, 99, 231-257.	1.4	35
50	Pluripotency factors and Polycomb Group proteins repress aryl hydrocarbon receptor expression in murine embryonic stem cells. Stem Cell Research, 2014, 12, 296-308.	0.7	35
51	Stable Expression of MouseCyp1a1and HumanCYP1A2cDNAs Transfected into Mouse Hepatoma Cells Lacking Detectable P450 Enzyme Activity. DNA and Cell Biology, 1990, 9, 425-436.	1.9	33
52	Regulation of Mouse Ah Receptor (Ahr) Gene Basal Expression by Members of the Sp Family of Transcription Factors. DNA and Cell Biology, 1998, 17, 811-822.	1.9	31
53	Long-term exposure to low-concentrations of Cr(VI) induce DNA damage and disrupt the transcriptional response to benzo[a]pyrene. Toxicology, 2014, 316, 14-24.	4.2	31
54	Perspectives on the Potential Involvement of the Ah Receptor-Dioxin Axis in Cardiovascular Disease. Toxicological Sciences, 2011, 120, 256-261.	3.1	29

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55	Distinct Contributions of JNK and p38 to Chromium Cytotoxicity and Inhibition of Murine Embryonic Stem Cell Differentiation. Environmental Health Perspectives, 2009, 117, 1124-1130.	6.0	28
56	Lead Induces Similar Gene Expression Changes in Brains of Gestationally Exposed Adult Mice and in Neurons Differentiated from Mouse Embryonic Stem Cells. PLoS ONE, 2013, 8, e80558.	2.5	28
57	Repression of Ah receptor and induction of transforming growth factor-Î² genes in DEN-induced mouse liver tumors. Toxicology, 2008, 246, 242-247.	4.2	27
58	Distinct Signaling Properties of Mitogen-activated Protein Kinase Kinases 4 (MKK4) and 7 (MKK7) in Embryonic Stem Cell (ESC) Differentiation. Journal of Biological Chemistry, 2012, 287, 2787-2797.	3.4	24
59	Aromatic hydrocarbon receptor polymorphism: development of new methods to correlate genotype with phenotype.. Environmental Health Perspectives, 1998, 106, 421-426.	6.0	22
60	Gene Expression Profiles of Mouse Aorta and Cultured Vascular Smooth Muscle Cells Differ Widely, Yet Show Common Responses to Dioxin Exposure. Cardiovascular Toxicology, 2004, 4, 385-404.	2.7	21
61	The Ah Receptor Recruits IKKÎ± to Its Target Binding Motifs to Phosphorylate Serine-10 in Histone H3 Required for Transcriptional Activation. Toxicological Sciences, 2014, 139, 121-132.	3.1	21
62	Chromium disrupts chromatin organization and CTCF access to its cognate sites in promoters of differentially expressed genes. Epigenetics, 2018, 13, 363-375.	2.7	21
63	Long-term exposure to hexavalent chromium inhibits expression of tumor suppressor genes in cultured cells and in mice. Journal of Trace Elements in Medicine and Biology, 2012, 26, 188-191.	3.0	20
64	Does the aryl hydrocarbon receptor regulate pluripotency?. Current Opinion in Toxicology, 2017, 2, 1-7.	5.0	20
65	Aromatic Hydrocarbon Receptor Polymorphism: Development of New Methods to Correlate Genotype with Phenotype. Environmental Health Perspectives, 1998, 106, 421.	6.0	18
66	Trout CYP1A3 Gene: Recognition of Fish DNA Motifs by Mouse Regulatory Proteins. Marine Biotechnology, 1999, 1, 155-166.	2.4	17
67	Loss of NR2E3 represses AHR by LSD1 reprogramming, is associated with poor prognosis in liver cancer. Scientific Reports, 2017, 7, 10662.	3.3	17
68	Regulation of a long noncoding RNA MALAT1 by aryl hydrocarbon receptor in pancreatic cancer cells and tissues. Biochemical and Biophysical Research Communications, 2020, 532, 563-569.	2.1	14
69	Converging Roles of the Aryl Hydrocarbon Receptor in Early Embryonic Development, Maintenance of Stemness, and Tissue Repair. Toxicological Sciences, 2021, 182, 1-9.	3.1	13
70	Hexavalent chromium disrupts chromatin architecture. Seminars in Cancer Biology, 2021, 76, 54-60.	9.6	13
71	Long-term Coexposure to Hexavalent Chromium and B[a]P Causes Tissue-Specific Differential Biological Effects in Liver and Gastrointestinal Tract of Mice. Toxicological Sciences, 2015, 146, 52-64.	3.1	12
72	Gene-Environment Interactions Target Mitogen-activated Protein 3 Kinase 1 (MAP3K1) Signaling in Eyelid Morphogenesis. Journal of Biological Chemistry, 2015, 290, 19770-19779.	3.4	10

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73	Formaldehyde-Assisted Isolation of Regulatory Elements (FAIRE) Analysis Uncovers Broad Changes in Chromatin Structure Resulting from Hexavalent Chromium Exposure. PLoS ONE, 2014, 9, e97849.	2.5	9
74	Ah receptor expression in cardiomyocytes protects adult female mice from heart dysfunction induced by TCDD exposure. Toxicology, 2016, 355-356, 9-20.	4.2	8
75	Dioxin Disrupts Dynamic DNA Methylation Patterns in Genes That Govern Cardiomyocyte Maturation. Toxicological Sciences, 2020, 178, 325-337.	3.1	7
76	Aryl Hydrocarbon Receptor Ablation in Cardiomyocytes Protects Male Mice From Heart Dysfunction Induced by NKX2.5 Haploinsufficiency. Toxicological Sciences, 2017, 160, 74-82.	3.1	5
77	Chromium exposure disrupts chromatin architecture upsetting the mechanisms that regulate transcription. Experimental Biology and Medicine, 2019, 244, 752-757.	2.4	5
78	Prenatal exposure to PCBs in Cyp1a2 knockâ€out mice interferes with F 1 fertility, impairs longâ€term potentiation, reduces acoustic startle and impairs conditioned freezing contextual memory with minimal transgenerational effects. Journal of Applied Toxicology, 2019, 39, 603-621.	2.8	4
79	Hexavalent chromium promotes differential binding of CTCF to its cognate sites in Euchromatin. Epigenetics, 2021, 16, 1-16.	2.7	3
80	Biochemical Responses to Dioxins: Which Genes? Which Endpoints?. , 2005, , 533-558.		1
81	Prenatal and early postnatal lead exposure in mice: neuroimaging findings. Quantitative Imaging in Medicine and Surgery, 2015, 5, 511-8.	2.0	1
82	Aryl Hydrocarbon Receptor. , 2016, , 1-15.		1
83	Aryl Hydrocarbon Receptor. , 2018, , 437-451.		1
84	Developmental and lifelong dioxin exposure induces measurable changes in cardiac structure and function in adulthood. Scientific Reports, 2021, 11, 10378.	3.3	0
85	Molecular Signatures of Dioxin Toxicity. , 2003, , .		0