Irene E Zohn

List of Publications by Year
in descending order

35 papers	1,491 citations	393982 19 h-index	377514 34 g-index
36 all docs	36 docs citations	36 times ranked	2218 citing authors

#	Article	IF	Citations
1	The coiled-coil domain containing protein CCDC40 is essential for motile cilia function and left-right axis formation. Nature Genetics, 2011, 43, 79-84.	9.4	292
2	p38 and a p38-Interacting Protein Are Critical for Downregulation of E-Cadherin during Mouse Gastrulation. Cell, 2006, 125, 957-969.	13.5	217
3	The visceral yolk sac endoderm provides for absorption of nutrients to the embryo during neurulation. Birth Defects Research Part A: Clinical and Molecular Teratology, 2010, 88, 593-600.	1.6	95
4	The flatiron mutation in mouse ferroportin acts as a dominant negative to cause ferroportin disease. Blood, 2007, 109, 4174-4180.	0.6	93
5	Rho GTPase-dependent transformation by G protein-coupled receptors. Oncogene, 2001, 20, 1547-1555.	2.6	77
6	Mas Oncogene Signaling and Transformation Require the Small GTP-Binding Protein Rac. Molecular and Cellular Biology, 1998, 18, 1225-1235.	1.1	73
7	G2A is an oncogenic G protein-coupled receptor. Oncogene, 2000, 19, 3866-3877.	2.6	71
8	Hectd1 regulates intracellular localization and secretion of Hsp90 to control cellular behavior of the cranial mesenchyme. Journal of Cell Biology, 2012, 196, 789-800.	2.3	66
9	The Hectd1 ubiquitin ligase is required for development of the head mesenchyme and neural tube closure. Developmental Biology, 2007, 306, 208-221.	0.9	63
10	HectD1 E3 Ligase Modifies Adenomatous Polyposis Coli (APC) with Polyubiquitin to Promote the APC-Axin Interaction. Journal of Biological Chemistry, 2013, 288, 3753-3767.	1.6	58
11	Using genomewide mutagenesis screens to identify the genes required for neural tube closure in the mouse. Birth Defects Research Part A: Clinical and Molecular Teratology, 2005, 73, 583-590.	1.6	51
12	Dysphagia and disrupted cranial nerve development in a mouse model of DiGeorge/22q11 Deletion Syndrome. DMM Disease Models and Mechanisms, 2014, 7, 245-57.	1.2	42
13	Hard to swallow: Developmental biological insights into pediatric dysphagia. Developmental Biology, 2016, 409, 329-342.	0.9	39
14	Hectd1 is required for development of the junctional zone of the placenta. Developmental Biology, 2014, 392, 368-380.	0.9	30
15	Chapter 1 Modeling Neural Tube Defects in the Mouse. Current Topics in Developmental Biology, 2008, 84, 1-35.	1.0	28
16	Mouse as a model for multifactorial inheritance of neural tube defects. Birth Defects Research Part C: Embryo Today Reviews, 2012, 96, 193-205.	3.6	26
17	Suckling, Feeding, and Swallowing: Behaviors, Circuits, and Targets for Neurodevelopmental Pathology. Annual Review of Neuroscience, 2020, 43, 315-336.	5.0	26
18	Cell polarity pathways converge and extend to regulate neural tube closure. Trends in Cell Biology, 2003, 13, 451-454.	3.6	25

#	Article	IF	Citations
19	Persistent Feeding and Swallowing Deficits in a Mouse Model of 22q11.2 Deletion Syndrome. Frontiers in Neurology, 2020, 11, 4.	1.1	22
20	Prevention of neural tube defects in <i>Lrp2</i> mutant mouse embryos by folic acid supplementation. Birth Defects Research, 2017, 109, 16-26.	0.8	16
21	The ubiquitin ligase HECTD1 promotes retinoic acid signaling required for development of the aortic arch. DMM Disease Models and Mechanisms, 2019, 12, .	1.2	12
22	Does the cranial mesenchyme contribute to neural fold elevation during neurulation?. Birth Defects Research Part A: Clinical and Molecular Teratology, 2012, 94, 841-848.	1.6	10
23	Transformation by a nucleotide-activated P2Y receptor is mediated by activation of GÎ±i, GÎ±q and Rho-dependent signaling pathways. Journal of Molecular Signaling, 2010, 5, 11.	0.5	9
24	High levels of iron supplementation prevents neural tube defects in the <i>Fpn1^{ffe}</i> mouse model. Birth Defects Research, 2017, 109, 81-91.	0.8	9
25	Abnormal labyrinthine zone in the Hectd1 -null placenta. Placenta, 2016, 38, 16-23.	0.7	7
26	Variations in maternal vitamin A intake modifies phenotypes in a mouse model of 22q11.2 deletion syndrome. Birth Defects Research, 2020, 112, 1194-1208.	0.8	7
27	Mouse Models of Neural Tube Defects. Advances in Experimental Medicine and Biology, 2020, 1236, 39-64.	0.8	6
28	Supt20 is required for development of the axial skeleton. Developmental Biology, 2017, 421, 245-257.	0.9	4
29	Mechanism for generation of left isomerism in Ccdc40 mutant embryos. PLoS ONE, 2017, 12, e0171180.	1.1	4
30	An Explant Assay for Assessing Cellular Behavior of the Cranial Mesenchyme. Journal of Visualized Experiments, 2013, , .	0.2	2
31	Papers from the Ninth International Conference on Neural Tube Defects. Birth Defects Research, 2017, 109, 65-67.	0.8	2
32	Reduced maternal vitamin A status increases the incidence of normal aortic arch variants. Genesis, 2019, 57, e23326.	0.8	2
33	Hsp90 and complex birth defects: A plausible mechanism for the interaction of genes and environment. Neuroscience Letters, 2020, 716, 134680.	1.0	2
34	Neural Tube Defects. , 2015, , 697-721.		1
35	Model organisms and mechanisms of geneâ€"environment interactions in structural birth defects. Genesis, 2021, 59, e23461.	0.8	0