

# Irene E Zohn

## List of Publications by Year in descending order

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35  
papers

1,491  
citations

394421

19  
h-index

377865

34  
g-index

36  
all docs

36  
docs citations

36  
times ranked

2218  
citing authors

#	ARTICLE	IF	CITATIONS
1	Model organisms and mechanisms of gene–environment interactions in structural birth defects. <i>Genesis</i> , 2021, 59, e23461.	1.6	0
2	Hsp90 and complex birth defects: A plausible mechanism for the interaction of genes and environment. <i>Neuroscience Letters</i> , 2020, 716, 134680.	2.1	2
3	Variations in maternal vitamin A intake modifies phenotypes in a mouse model of 22q11.2 deletion syndrome. <i>Birth Defects Research</i> , 2020, 112, 1194-1208.	1.5	7
4	Persistent Feeding and Swallowing Deficits in a Mouse Model of 22q11.2 Deletion Syndrome. <i>Frontiers in Neurology</i> , 2020, 11, 4.	2.4	22
5	Suckling, Feeding, and Swallowing: Behaviors, Circuits, and Targets for Neurodevelopmental Pathology. <i>Annual Review of Neuroscience</i> , 2020, 43, 315-336.	10.7	26
6	Mouse Models of Neural Tube Defects. <i>Advances in Experimental Medicine and Biology</i> , 2020, 1236, 39-64.	1.6	6
7	The ubiquitin ligase HECTD1 promotes retinoic acid signaling required for development of the aortic arch. <i>DMM Disease Models and Mechanisms</i> , 2019, 12, .	2.4	12
8	Reduced maternal vitamin A status increases the incidence of normal aortic arch variants. <i>Genesis</i> , 2019, 57, e23326.	1.6	2
9	Supt20 is required for development of the axial skeleton. <i>Developmental Biology</i> , 2017, 421, 245-257.	2.0	4
10	High levels of iron supplementation prevents neural tube defects in the <i>Fpn1</i> mouse model. <i>Birth Defects Research</i> , 2017, 109, 81-91.	1.5	9
11	Papers from the Ninth International Conference on Neural Tube Defects. <i>Birth Defects Research</i> , 2017, 109, 65-67.	1.5	2
12	Prevention of neural tube defects in <i>Lrp2</i> mutant mouse embryos by folic acid supplementation. <i>Birth Defects Research</i> , 2017, 109, 16-26.	1.5	16
13	Mechanism for generation of left isomerism in <i>Ccdc40</i> mutant embryos. <i>PLoS ONE</i> , 2017, 12, e0171180.	2.5	4
14	Abnormal labyrinthine zone in the <i>Hectd1</i> -null placenta. <i>Placenta</i> , 2016, 38, 16-23.	1.5	7
15	Hard to swallow: Developmental biological insights into pediatric dysphagia. <i>Developmental Biology</i> , 2016, 409, 329-342.	2.0	39
16	Neural Tube Defects. , 2015, , 697-721.		1
17	Dysphagia and disrupted cranial nerve development in a mouse model of DiGeorge/22q11 Deletion Syndrome. <i>DMM Disease Models and Mechanisms</i> , 2014, 7, 245-57.	2.4	42
18	<i>Hectd1</i> is required for development of the junctional zone of the placenta. <i>Developmental Biology</i> , 2014, 392, 368-380.	2.0	30

#	ARTICLE	IF	CITATIONS
19	HectD1 E3 Ligase Modifies Adenomatous Polyposis Coli (APC) with Polyubiquitin to Promote the APC-Axin Interaction. <i>Journal of Biological Chemistry</i> , 2013, 288, 3753-3767.	3.4	58
20	An Explant Assay for Assessing Cellular Behavior of the Cranial Mesenchyme. <i>Journal of Visualized Experiments</i> , 2013, , .	0.3	2
21	Hectd1 regulates intracellular localization and secretion of Hsp90 to control cellular behavior of the cranial mesenchyme. <i>Journal of Cell Biology</i> , 2012, 196, 789-800.	5.2	66
22	Does the cranial mesenchyme contribute to neural fold elevation during neurulation?. <i>Birth Defects Research Part A: Clinical and Molecular Teratology</i> , 2012, 94, 841-848.	1.6	10
23	Mouse as a model for multifactorial inheritance of neural tube defects. <i>Birth Defects Research Part C: Embryo Today Reviews</i> , 2012, 96, 193-205.	3.6	26
24	The coiled-coil domain containing protein CCDC40 is essential for motile cilia function and left-right axis formation. <i>Nature Genetics</i> , 2011, 43, 79-84.	21.4	292
25	Transformation by a nucleotide-activated P2Y receptor is mediated by activation of G $\alpha$ i, G $\alpha$ q and Rho-dependent signaling pathways. <i>Journal of Molecular Signaling</i> , 2010, 5, 11.	0.5	9
26	The visceral yolk sac endoderm provides for absorption of nutrients to the embryo during neurulation. <i>Birth Defects Research Part A: Clinical and Molecular Teratology</i> , 2010, 88, 593-600.	1.6	95
27	Chapter 1 Modeling Neural Tube Defects in the Mouse. <i>Current Topics in Developmental Biology</i> , 2008, 84, 1-35.	2.2	28
28	The flatiron mutation in mouse ferroportin acts as a dominant negative to cause ferroportin disease. <i>Blood</i> , 2007, 109, 4174-4180.	1.4	93
29	The Hectd1 ubiquitin ligase is required for development of the head mesenchyme and neural tube closure. <i>Developmental Biology</i> , 2007, 306, 208-221.	2.0	63
30	p38 and a p38-Interacting Protein Are Critical for Downregulation of E-Cadherin during Mouse Gastrulation. <i>Cell</i> , 2006, 125, 957-969.	28.9	217
31	Using genomewide mutagenesis screens to identify the genes required for neural tube closure in the mouse. <i>Birth Defects Research Part A: Clinical and Molecular Teratology</i> , 2005, 73, 583-590.	1.6	51
32	Cell polarity pathways converge and extend to regulate neural tube closure. <i>Trends in Cell Biology</i> , 2003, 13, 451-454.	7.9	25
33	Rho GTPase-dependent transformation by G protein-coupled receptors. <i>Oncogene</i> , 2001, 20, 1547-1555.	5.9	77
34	G2A is an oncogenic G protein-coupled receptor. <i>Oncogene</i> , 2000, 19, 3866-3877.	5.9	71
35	Mas Oncogene Signaling and Transformation Require the Small GTP-Binding Protein Rac. <i>Molecular and Cellular Biology</i> , 1998, 18, 1225-1235.	2.3	73