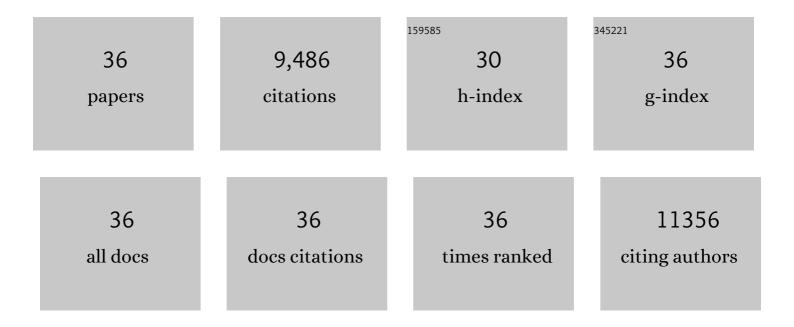
Chuxia Deng

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Generation of Smurf2 Conditional Knockout Mice. International Journal of Biological Sciences, 2018, 14, 542-548.	6.4	2
2	Bombesin-like receptor 3 (Brs3) expression in glutamatergic, but not GABAergic, neurons is required for regulation of energy metabolism. Molecular Metabolism, 2017, 6, 1540-1550.	6.5	15
3	Mutant activated FGFR3 impairs endochondral bone growth by preventing SOX9 downregulation in differentiating chondrocytes. Human Molecular Genetics, 2015, 24, 1764-1773.	2.9	33
4	Defective FGF signaling causes coloboma formation and disrupts retinal neurogenesis. Cell Research, 2013, 23, 254-273.	12.0	36
5	TGF-Î ² and BMP Signaling in Osteoblast Differentiation and Bone Formation. International Journal of Biological Sciences, 2012, 8, 272-288.	6.4	1,354
6	Myocardial deletion of <i>Smad4 </i> using a novel α skeletal muscle actin Cre recombinase transgenic mouse causes misalignment of the cardiac outflow tract. International Journal of Biological Sciences, 2010, 6, 546-555.	6.4	25
7	Generation of <i>Fgfr3 </i> Conditional Knockout Mice. International Journal of Biological Sciences, 2010, 6, 327-332.	6.4	38
8	A Pro253Arg mutation in fibroblast growth factor receptor 2 (Fgfr2) causes skeleton malformation mimicking human Apert syndrome by affecting both chondrogenesis and osteogenesis. Bone, 2008, 42, 631-643.	2.9	124
9	Docking of Axonal Mitochondria by Syntaphilin Controls Their Mobility and Affects Short-Term Facilitation. Cell, 2008, 132, 137-148.	28.9	497
10	Smad3 Signal Transducer Regulates Skin Inflammation and Specific IgE Response in Murine Model of Atopic Dermatitis. Journal of Investigative Dermatology, 2007, 127, 1923-1929.	0.7	34
11	Ewing sarcoma gene EWS is essential for meiosis and B lymphocyte development. Journal of Clinical Investigation, 2007, 117, 1314-1323.	8.2	135
12	A critical role for β cell M3 muscarinic acetylcholine receptors in regulating insulin release and blood glucose homeostasis in vivo. Cell Metabolism, 2006, 3, 449-461.	16.2	246
13	Identification of the control region for tissue-specific imprinting of the stimulatory G protein α-subunit. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 5513-5518.	7.1	97
14	Alternative <i>Gnas</i> gene products have opposite effects on glucose and lipid metabolism. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 7386-7391.	7.1	174
15	Smad3 Knockout Mice Exhibit a Resistance to Skin Chemical Carcinogenesis. Cancer Research, 2004, 64, 7836-7845.	0.9	77
16	Defective lysosomal targeting of activated fibroblast growth factor receptor 3 in achondroplasia. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 609-614.	7.1	110
17	HBsAg andHBx knocked into thep21 locus causes hepatocellular carcinoma in mice. Hepatology, 2004, 39, 318-324.	7.3	129
18	Smad3 is required for enamel biomineralization. Biochemical and Biophysical Research Communications, 2003, 305, 684-690.	2.1	31

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19	Mice Lacking Smad3 Are Protected Against Cutaneous Injury Induced by Ionizing Radiation. American Journal of Pathology, 2002, 160, 1057-1068.	3.8	274
20	Receptorâ€regulated and inhibitory Smads are critical in regulating transforming growth factorβ–mediated Meckel's cartilage development. Developmental Dynamics, 2002, 224, 69-78.	1.8	67
21	Conditional knockout of theSmad1 gene. Genesis, 2002, 32, 76-79.	1.6	42
22	Overexpression of Smad2 Reveals Its Concerted Action with Smad4 in Regulating TGF- β-Mediated Epidermal Homeostasis. Developmental Biology, 2001, 236, 181-194.	2.0	60
23	A Ser365->Cys mutation of fibroblast growth factor receptor 3 in mouse downregulates Ihh/PTHrP signals and causes severe achondroplasia. Human Molecular Genetics, 2001, 10, 457-465.	2.9	146
24	Antagonistic Effects of Smad2 VersusSmad7 Are Sensitive to Their Expression Level during Tooth Development. Journal of Biological Chemistry, 2001, 276, 44163-44172.	3.4	30
25	Functional Characterization of Transforming Growth Factor β Signaling in Smad2- and Smad3-deficient Fibroblasts. Journal of Biological Chemistry, 2001, 276, 19945-19953.	3.4	367
26	Functional Collaboration between Different Cyclin-Dependent Kinase Inhibitors Suppresses Tumor Growth with Distinct Tissue Specificity. Molecular and Cellular Biology, 2000, 20, 6147-6158.	2.3	276
27	Lack of Obesity and Normal Response to Fasting and Thyroid Hormone in Mice Lacking Uncoupling Protein-3. Journal of Biological Chemistry, 2000, 275, 16251-16257.	3.4	342
28	Mice lacking Smad3 show accelerated wound healing and an impaired local inflammatory response. Nature Cell Biology, 1999, 1, 260-266.	10.3	842
29	Differential regulation of p21 by p53 and Rb in cellular response to oxidative stress. Molecular Carcinogenesis, 1999, 24, 15-24.	2.7	61
30	Differential regulation of p21 by p53 and Rb in cellular response to oxidative stress. Molecular Carcinogenesis, 1999, 24, 15-24.	2.7	2
31	A genetic model of substrate deprivation therapy for a glycosphingolipid storage disorder. Journal of Clinical Investigation, 1999, 103, 497-505.	8.2	153
32	Involvement of p21 and p27 in the regulation of CDK activity and cell cycle progression in the regenerating liver. Oncogene, 1998, 16, 2141-2150.	5.9	176
33	Fibroblast Growth Factor Receptor-1 (FGFR-1) Is Essential for Normal Neural Tube and Limb Development. Developmental Biology, 1997, 185, 42-54.	2.0	145
34	Activation of Statl by mutant fibroblast growth-factor receptor in thanatophoric dysplasia type II dwarfism. Nature, 1997, 386, 288-292.	27.8	310
35	Fibroblast Growth Factor Receptor 3 Is a Negative Regulator of Bone Growth. Cell, 1996, 84, 911-921.	28.9	1,014
36	Mice Lacking p21CIP1/WAF1 undergo normal development, but are defective in G1 checkpoint control. Cell, 1995, 82, 675-684.	28.9	2,022