## Flavia Cerrato

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Microdeletions in the human H19 DMR result in loss of IGF2 imprinting and Beckwith-Wiedemann syndrome. Nature Genetics, 2004, 36, 958-960.	21.4	261
2	Hypomethylation at multiple maternally methylated imprinted regions including PLAGL1 and GNAS loci in Beckwith–Wiedemann syndrome. European Journal of Human Genetics, 2009, 17, 611-619.	2.8	194
3	The KCNQ1OT1 imprinting control region and non-coding RNA: new properties derived from the study of Beckwith–Wiedemann syndrome and Silver–Russell syndrome cases. Human Molecular Genetics, 2012, 21, 10-25.	2.9	135
4	Mechanisms causing imprinting defects in familial Beckwith–Wiedemann syndrome with Wilms' tumour. Human Molecular Genetics, 2007, 16, 254-264.	2.9	100
5	Distinct Methylation Changes at the IGF2-H19 Locus in Congenital Growth Disorders and Cancer. PLoS ONE, 2008, 3, e1849.	2.5	93
6	The molecular function and clinical phenotype of partial deletions of the IGF2/H19 imprinting control region depends on the spatial arrangement of the remaining CTCF-binding sites. Human Molecular Genetics, 2013, 22, 544-557.	2.9	78
7	Different mechanisms cause imprinting defects at the IGF2/H19 locus in Beckwith-Wiedemann syndrome and Wilms' tumour. Human Molecular Genetics, 2008, 17, 1427-1435.	2.9	76
8	Role of histone acetylation and DNA methylation in the maintenance of the imprinted expression of the <i>H19</i> and <i>Igf2</i> genes. FEBS Letters, 1999, 458, 45-50.	2.8	73
9	The two-domain hypothesis in Beckwith–Wiedemann syndrome: autonomous imprinting of the telomeric domain of the distal chromosome 7 cluster. Human Molecular Genetics, 2005, 14, 503-511.	2.9	63
10	Inherited and Sporadic Epimutations at the <i>IGF2-H19</i> Locus in Beckwith-Wiedemann Syndrome and Wilms’ Tumor. Endocrine Development, 2009, 14, 1-9.	1.3	48
11	Loss-of-function maternal-effect mutations of PADI6 are associated with familial and sporadic Beckwith-Wiedemann syndrome with multi-locus imprinting disturbance. Clinical Epigenetics, 2020, 12, 139.	4.1	40
12	Transcription alterations of KCNQ1 associated with imprinted methylation defects in the Beckwith–Wiedemann locus. Genetics in Medicine, 2019, 21, 1808-1820.	2.4	38
13	A splicing mutation of the HMGA2 gene is associated with Silver–Russell syndrome phenotype. Journal of Human Genetics, 2015, 60, 287-293.	2.3	33
14	A novel microdeletion in the IGF2/H19 imprinting centre region defines a recurrent mutation mechanism in familial Beckwith–Wiedemann syndrome. European Journal of Medical Genetics, 2011, 54, e451-e454.	1.3	30
15	Paternal deletion of the 11p15.5 centromeric-imprinting control region is associated with alteration of imprinted gene expression and recurrent severe intrauterine growth restriction. Journal of Medical Genetics, 2013, 50, 99-103.	3.2	29
16	The H19 endodermal enhancer is required for Igf2 activation and tumor formation in experimental liver carcinogenesis. Oncogene, 2000, 19, 6376-6385.	5.9	28
17	Humanized <i>H19/lgf2</i> locus reveals diverged imprinting mechanism between mouse and human and reflects Silver–Russell syndrome phenotypes. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 10938-10943.	7.1	28
18	DNA Methylation in the Diagnosis of Monogenic Diseases. Genes, 2020, 11, 355.	2.4	28

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19	ls ZFP57 binding to H19/IGF2:IG-DMR affected in Silver-Russell syndrome?. Clinical Epigenetics, 2018, 10, 23.	4.1	25
20	Origins of DNA methylation defects in Wilms tumors. Cancer Letters, 2019, 457, 119-128.	7.2	23
21	The phenotypic variations of multi-locus imprinting disturbances associated with maternal-effect variants of NLRP5 range from overt imprinting disorder to apparently healthy phenotype. Clinical Epigenetics, 2019, 11, 190.	4.1	22
22	Paternal imprints can be established on the maternal Igf2-H19 locus without altering replication timing of DNA. Human Molecular Genetics, 2003, 12, 3123-3132.	2.9	19
23	Looking for CDKN1C enhancers. European Journal of Human Genetics, 2014, 22, 442-443.	2.8	19
24	Reply to "Microdeletion and IGF2 loss of imprinting in a cascade causing Beckwith-Wiedemann syndrome with Wilms' tumor". Nature Genetics, 2005, 37, 786-787.	21.4	18
25	Tissue-specific and mosaic imprinting defects underlie opposite congenital growth disorders in mice. PLoS Genetics, 2018, 14, e1007243.	3.5	13
26	Developmentally regulated functions of the H19 differentially methylated domain. Human Molecular Genetics, 2003, 13, 353-361.	2.9	11
27	The number of the CTCF binding sites of the <i>H19/IGF2</i> :IG-DMR correlates with DNA methylation and expression imprinting in a humanized mouse model. Human Molecular Genetics, 2021, 30, 1509-1520.	2.9	10
28	A novel large deletion of the ICR1 region including H19 and putative enhancer elements. BMC Medical Genetics, 2015, 16, 30.	2.1	9
29	Two maternal duplications involving the CDKN1C gene are associated with contrasting growth phenotypes. Clinical Epigenetics, 2016, 8, 69.	4.1	9
30	Novel genetic variants of KHDC3L and other members of the subcortical maternal complex associated with Beckwith–Wiedemann syndrome or Pseudohypoparathyroidism 1B and multi-locus imprinting disturbances. Clinical Epigenetics, 2022, 14, .	4.1	7
31	Both Epimutations and Chromosome Aberrations Affect Multiple Imprinted Loci in Aggressive Wilms Tumors. Cancers, 2020, 12, 3411.	3.7	6
32	Mosaic Segmental and Whole-Chromosome Upd(11)mat in Silver-Russell Syndrome. Genes, 2021, 12, 581.	2.4	5
33	Variable Expressivity of the Beckwith-Wiedemann Syndrome in Four Pedigrees Segregating Loss-of-Function Variants of CDKN1C. Genes, 2021, 12, 706.	2.4	2