## List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/4876046/publications.pdf Version: 2024-02-01



WAN LEE

#	Article	IF	CITATIONS
1	MiR-320-3p Regulates the Proliferation and Differentiation of Myogenic Progenitor Cells by Modulating Actin Remodeling. International Journal of Molecular Sciences, 2022, 23, 801.	4.1	9
2	Twinfilin-1 is an essential regulator of myogenic differentiation through the modulation of YAP in C2C12 myoblasts. Biochemical and Biophysical Research Communications, 2022, 599, 17-23.	2.1	7
3	MiR-141-3p regulates myogenic differentiation in C2C12 myoblasts via CFL2-YAP-mediated mechanotransduction. BMB Reports, 2022, 55, 104-109.	2.4	5
4	MiR-183-5p Induced by Saturated Fatty Acids Hinders Insulin Signaling by Downregulating IRS-1 in Hepatocytes. International Journal of Molecular Sciences, 2022, 23, 2979.	4.1	3
5	MiR-141-3p regulates myogenic differentiation in C2C12 myoblasts via CFL2-YAP-mediated mechanotransduction BMB Reports, 2022, , .	2.4	Ο
6	Kank1 Is Essential for Myogenic Differentiation by Regulating Actin Remodeling and Cell Proliferation in C2C12 Progenitor Cells. Cells, 2022, 11, 2030.	4.1	7
7	Palmitic Acid-Induced miR-429-3p Impairs Myoblast Differentiation by Downregulating CFL2. International Journal of Molecular Sciences, 2021, 22, 10972.	4.1	6
8	Role of MiR-325-3p in the Regulation of CFL2 and Myogenic Differentiation of C2C12 Myoblasts. Cells, 2021, 10, 2725.	4.1	8
9	CFL2 is an essential mediator for myogenic differentiation in C2C12 myoblasts. Biochemical and Biophysical Research Communications, 2020, 533, 710-716.	2.1	19
10	MiR-96-5p Induced by Palmitic Acid Suppresses the Myogenic Differentiation of C2C12 Myoblasts by Targeting FHL1. International Journal of Molecular Sciences, 2020, 21, 9445.	4.1	12
11	MiR-183-5p induced by saturated fatty acids regulates the myogenic differentiation by directly targeting FHL1 in C2C12 myoblasts. BMB Reports, 2020, 53, 605-610.	2.4	13
12	Alteration of mitochondrial DNA content modulates antioxidant enzyme expressions and oxidative stress in myoblasts. Korean Journal of Physiology and Pharmacology, 2019, 23, 519.	1.2	5
13	Mitochondrial dysfunction reduces the activity of KIR2.1 K <sup>+</sup> channel in myoblasts <i>via</i> impaired oxidative phosphorylation. Korean Journal of Physiology and Pharmacology, 2018, 22, 697.	1.2	2
14	Saturated fatty acids-induced miR-424–5p aggravates insulin resistance via targeting insulin receptor in hepatocytes. Biochemical and Biophysical Research Communications, 2018, 503, 1587-1593.	2.1	28
15	Exosome-derived microRNAs in cancer metabolism: possible implications in cancer diagnostics and therapy. Experimental and Molecular Medicine, 2017, 49, e285-e285.	7.7	169
16	Dataset on the identification of differentially expressed genes by annealing control primer-based PCR in mitochondrial DNA-depleted myocytes. Data in Brief, 2017, 11, 266-272.	1.0	0
17	Data on the expression of PEPCK in HepG2 hepatocytes transfected with miR-195. Data in Brief, 2017, 15, 747-751.	1.0	5
18	Data on the decreased expression of FOXO1 by miR-1271 in HepG2 hepatocytes. Data in Brief, 2017, 15, 800-804.	1.0	4

#	Article	IF	CITATIONS
19	Data on the expression and insulin-stimulated phosphorylation of IRS-1 by miR-96 in L6-GLUT4myc myocytes. Data in Brief, 2017, 15, 728-732.	1.0	4
20	Data on the effect of miR-15b on the expression of INSR in murine C2C12 myocytes. Data in Brief, 2017, 15, 882-886.	1.0	0
21	MicroRNA expression analysis in the liver of high fat diet-induced obese mice. Data in Brief, 2016, 9, 1155-1159.	1.0	11
22	Data for differentially expressed microRNAs in saturated fatty acid palmitate-treated HepG2 cells. Data in Brief, 2016, 9, 996-999.	1.0	3
23	MiR-1271 upregulated by saturated fatty acid palmitate provokes impaired insulin signaling by repressing INSR and IRS-1 expression in HepG2 cells. Biochemical and Biophysical Research Communications, 2016, 478, 1786-1791.	2.1	28
24	Induction of miR-96 by Dietary Saturated Fatty Acids Exacerbates Hepatic Insulin Resistance through the Suppression of INSR and IRS-1. PLoS ONE, 2016, 11, e0169039.	2.5	60
25	C1q tumor necrosis factor α-related protein isoform 5 attenuates palmitate-induced DNA fragmentation in myocytes through an AMPK-dependent mechanism. Data in Brief, 2015, 5, 770-774.	1.0	4
26	Obesityâ€induced miRâ€15b is linked causally to the development of insulin resistance through the repression of the insulin receptor in hepatocytes. Molecular Nutrition and Food Research, 2015, 59, 2303-2314.	3.3	77
27	Sfrp2 is a transcriptional target of SREBP-1 in mouse chondrogenic cells. Molecular and Cellular Biochemistry, 2015, 406, 163-171.	3.1	1
28	MicroRNA-126 Suppresses Mesothelioma Malignancy by Targeting IRS1 and Interfering with the Mitochondrial Function. Antioxidants and Redox Signaling, 2014, 21, 2109-2125.	5.4	85
29	Saturated fatty acidâ€induced miRâ€195 impairs insulin signaling and glycogen metabolism in HepC2 cells. FEBS Letters, 2014, 588, 3939-3946.	2.8	74
30	CTRP5 ameliorates palmitate-induced apoptosis and insulin resistance through activation of AMPK and fatty acid oxidation. Biochemical and Biophysical Research Communications, 2014, 452, 715-721.	2.1	22
31	Induction of miRâ€29a by saturated fatty acids impairs insulin signaling and glucose uptake through translational repression of IRSâ€1 in myocytes. FEBS Letters, 2014, 588, 2170-2176.	2.8	97
32	The induction of miR-96 by mitochondrial dysfunction causes impaired glycogen synthesis through translational repression of IRS-1 in SK-Hep1 cells. Biochemical and Biophysical Research Communications, 2013, 434, 503-508.	2.1	35
33	Implications of microRNAs in the pathogenesis of diabetes. Archives of Pharmacal Research, 2013, 36, 154-166.	6.3	37
34	Regulation of the transcriptional activation of CTRP3 in chondrocytes by c-Jun. Molecular and Cellular Biochemistry, 2012, 368, 111-117.	3.1	5
35	Effects of Aerobic Exercise Training on C1q Tumor Necrosis Factor α-Related Protein Isoform 5 (Myonectin): Association with Insulin Resistance and Mitochondrial DNA Density in Women. Journal of Clinical Endocrinology and Metabolism, 2012, 97, E88-E93.	3.6	41
36	Ets-2 is involved in transcriptional regulation of C1qTNF-related protein 5 in muscle cells. Molecular Biology Reports, 2012, 39, 9445-9451.	2.3	3

#	Article	IF	CITATIONS
37	Depletion of Mitochondrial DNA Stabilizes C1qTNF-Related Protein 6 mRNA in Muscle Cells. Journal of Korean Medical Science, 2012, 27, 465.	2.5	8
38	The Induction of MicroRNA Targeting IRS-1 Is Involved in the Development of Insulin Resistance under Conditions of Mitochondrial Dysfunction in Hepatocytes. PLoS ONE, 2011, 6, e17343.	2.5	127
39	Comparison of laparoscopic versus open radical nephrectomy for large renal tumors: a retrospective analysis of multiâ€center results. BJU International, 2011, 107, 817-821.	2.5	48
40	ldentification of the Target Proteins of Rosiglitazone in 3T3-L1 Adipocytes through Proteomic Analysis of Cytosolic and Secreted Proteins. Molecules and Cells, 2011, 31, 239-246.	2.6	26
41	C1qTNF-Related Protein-6 Increases the Expression of Interleukin-10 in Macrophages. Molecules and Cells, 2010, 30, 59-64.	2.6	50
42	C1qTNFâ€related proteinâ€6 mediates fatty acid oxidation via the activation of the AMPâ€activated protein kinase. FEBS Letters, 2010, 584, 968-972.	2.8	43
43	Corrigendum to "C1qTNF-related protein-6 mediates fatty acid oxidation via the activation of the AMP-activated protein kinase―[FEBS Lett. 584 (2010) 968-972]. FEBS Letters, 2010, 584, 2491-2491.	2.8	1
44	Role of hepatocyte nuclear factorâ€4α in transcriptional regulation of C1qTNFâ€related protein 5 in the liver. FEBS Letters, 2010, 584, 3080-3084.	2.8	8
45	C1q Tumor Necrosis Factor α-related Protein Isoform 5 Is Increased in Mitochondrial DNA-depleted Myocytes and Activates AMP-activated Protein Kinase. Journal of Biological Chemistry, 2009, 284, 27780-27789.	3.4	93
46	Dangnyohwan improves glucose utilization and reduces insulin resistance by increasing the adipocyte-specific GLUT4 expression in Otsuka Long-Evans Tokushima Fatty rats. Journal of Ethnopharmacology, 2008, 115, 473-482.	4.1	15
47	Combination gene therapy using multidrug resistance (MDR1) gene shRNA and herpes simplex virus-thymidine kinase. Cancer Letters, 2008, 261, 205-214.	7.2	24
48	Depletion of mitochondrial DNA up-regulates the expression of MDR1 gene via an increase in mRNA stability. Experimental and Molecular Medicine, 2008, 40, 109.	7.7	52
49	Involvement of Vesicular H+-ATPase in Insulin-Stimulated Glucose Transport in 3T3-F442A Adipocytes. Endocrine Journal, 2007, 54, 733-743.	1.6	18
50	The depletion of cellular mitochondrial DNA causes insulin resistance through the alteration of insulin receptor substrate-1 in rat myocytes. Diabetes Research and Clinical Practice, 2007, 77, S165-S171.	2.8	22
51	Genetic risk for metabolic syndrome: examination of candidate gene polymorphisms related to lipid metabolism in Japanese people. Journal of Medical Genetics, 2007, 45, 22-28.	3.2	52
52	Proteomic analysis of cellular change involved in mitochondria-to-nucleus communication in L6â€GLUT4myc myocytes. Proteomics, 2006, 6, 1210-1222.	2.2	12
53	Implication of phosphorylation of the myosin II regulatory light chain in insulin-stimulated GLUT4 translocation in 3T3-F442A adipocytes. Experimental and Molecular Medicine, 2006, 38, 180-189.	7.7	28
54	O-GlcNAc modification on IRS-1 and Akt2 by PUGNAc inhibits their phosphorylation and induces insulin resistance in rat primary adipocytes. Experimental and Molecular Medicine, 2005, 37, 220-229.	7.7	126

#	Article	IF	CITATIONS
55	Depletion of Mitochondrial DNA Causes Impaired Glucose Utilization and Insulin Resistance in L6 GLUT4myc Myocytes. Journal of Biological Chemistry, 2005, 280, 9855-9864.	3.4	59
56	Calorie restriction improves whole-body glucose disposal and insulin resistance in association with the increased adipocyte-specific GLUT4 expression in Otsuka Long–Evans Tokushima Fatty rats. Archives of Biochemistry and Biophysics, 2005, 436, 276-284.	3.0	56
57	EHD2 Interacts with the Insulin-Responsive Glucose Transporter (GLUT4) in Rat Adipocytes and May Participate in Insulin-Induced GLUT4 Recruitmentâ€. Biochemistry, 2004, 43, 7552-7562.	2.5	33
58	N-Acetylated α-linked acidic dipeptidase expressed in rat adipocytes is localized in the insulin-responsive glucose transporter (GLUT4) intracellular compartments and involved in the insulin-stimulated GLUT4 recruitment. Archives of Biochemistry and Biophysics, 2004, 424, 11-22.	3.0	0
59	Cadmium induces impaired glucose tolerance in rat by down-regulating GLUT4 expression in adipocytes. Archives of Biochemistry and Biophysics, 2003, 413, 213-220.	3.0	90
60	Transient Changes in Four GLUT4 Compartments in Rat Adipocytes during the Transition, Insulin-Stimulated To Basal: Implications for the GLUT4 Trafficking Pathwayâ€. Biochemistry, 2002, 41, 14364-14371.	2.5	10
61	Protein kinase C-ζ phosphorylates insulin-responsive aminopeptidase in vitro at Ser-80 and Ser-91. Archives of Biochemistry and Biophysics, 2002, 403, 71-82.	3.0	16
62	The hepatocyte glucose-6-phosphatase subcomponent T3: its relationship to GLUT2. Biochimica Et Biophysica Acta - Biomembranes, 2002, 1564, 198-206.	2.6	8
63	Association of Carboxyl Esterase with Facilitative Glucose Transporter Isoform 4 (GLUT4) Intracellular Compartments in Rat Adipocytes and Its Possible Role in Insulin-induced GLUT4 Recruitment. Journal of Biological Chemistry, 2000, 275, 10041-10046.	3.4	9
64	Characterization and partial purification of liver glucose transporter GLUT2. Biochimica Et Biophysica Acta - Biomembranes, 2000, 1466, 379-389.	2.6	11
65	Modulation of GLUT4 and GLUT1 Recycling by Insulin in Rat Adipocytes:Â Kinetic Analysis Based on the Involvement of Multiple Intracellular Compartmentsâ€. Biochemistry, 2000, 39, 9358-9366.	2.5	28
66	Separation and Partial Characterization of Three Distinct Intracellular GLUT4 Compartments in Rat Adipocytes. Journal of Biological Chemistry, 1999, 274, 37755-37762.	3.4	33
67	Glucose transporters and insulin action: Some insights into diabetes management. Archives of Pharmacal Research, 1999, 22, 329-334.	6.3	8
68	Cloning of anl-3-Hydroxyacyl-CoA Dehydrogenase That Interacts with the GLUT4 C-Terminus. Archives of Biochemistry and Biophysics, 1999, 363, 323-332.	3.0	16
69	A Synthetic Peptide Corresponding to the GLUT4 C-terminal Cytoplasmic Domain Causes Insulin-like Glucose Transport Stimulation and GLUT4 Recruitment in Rat Adipocytes. Journal of Biological Chemistry, 1997, 272, 21427-21431.	3.4	26
70	A Myosin-Derived Peptide C109 Binds to GLUT4-Vesicles and Inhibits the Insulin-Induced Glucose Transport Stimulation and GLUT4 Recruitment in Rat Adipocytes. Biochemical and Biophysical Research Communications, 1997, 240, 409-414.	2.1	10
71	GLUT1 Transmembrane Glucose Pathway. Journal of Biological Chemistry, 1996, 271, 5225-5230.	3.4	19