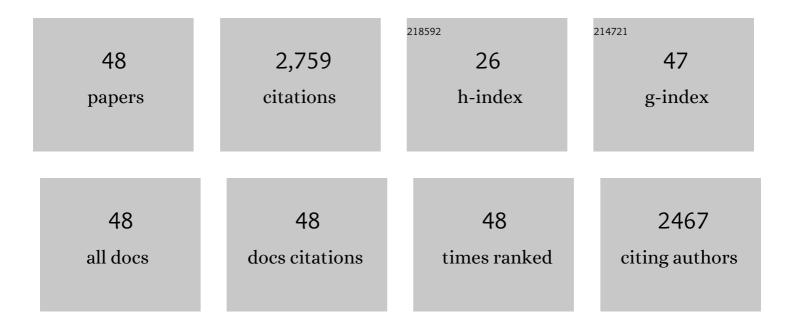
## Robert

## List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Roles of hepatic atypical protein kinase C hyperactivity and hyperinsulinemia in insulinâ€resistant forms of obesity and type 2 diabetes mellitus. MedComm, 2021, 2, 3-16.	3.1	5
2	Control of β-Site Amyloid Precursor Protein-Cleaving Enzyme-1 Expression by Protein Kinase C-λ/ι and Nuclear Factor κ-B. Current Alzheimer Research, 2021, 18, 941-955.	0.7	1
3	Atypical PKC controls β-secretase expression and thereby regulates production of Alzheimer plaque precursor Aβ in brain and insulin receptor degradation in liver Metabolism: Clinical and Experimental, 2020, 104, 154112.	1.5	1
4	Coordinated regulation of hepatic FoxO1, PGC-1α and SREBP-1c facilitates insulin action and resistance. Cellular Signalling, 2018, 43, 62-70.	1.7	23
5	Atypical PKC, PKCλ/ι, activates β-secretase and increases Aβ1–40/42 and phospho-tau in mouse brain and isolated neuronal cells, and may link hyperinsulinemia and other aPKC activators to development of pathological and memory abnormalities in Alzheimer's disease. Neurobiology of Aging, 2018, 61, 225-237.	1.5	18
6	Atypical PKC: therapeutic target for Alzheimer's?. Aging, 2018, 11, 13-14.	1.4	5
7	Deletion of Protein Kinase C λ in POMC Neurons Predisposes to Diet-Induced Obesity. Diabetes, 2017, 66, 920-934.	0.3	20
8	Brain Insulin Signaling Is Increased in Insulin-Resistant States and Decreases in FOXOs and PGC-1α and Increases in Aβ1–40/42 and Phospho-Tau May Abet Alzheimer Development. Diabetes, 2016, 65, 1892-1903.	0.3	72
9	Compensation for PKMζ in long-term potentiation and spatial long-term memory in mutant mice. ELife, 2016, 5, .	2.8	138
10	Hepatic insulin resistance in ob/ob mice involves increases in ceramide, aPKC activity, and selective impairment of Akt-dependent FoxO1 phosphorylation. Journal of Lipid Research, 2015, 56, 70-80.	2.0	48
11	BMI-related progression of atypical PKC-dependent aberrations in insulin signaling through IRS-1, Akt, FoxO1 and PGC-11± in livers of obese and type 2 diabetic humans. Metabolism: Clinical and Experimental, 2015, 64, 1454-1465.	1.5	22
12	Pharmacological TLR4 Inhibition Protects against Acute and Chronic Fat-Induced Insulin Resistance in Rats. PLoS ONE, 2015, 10, e0132575.	1.1	22
13	PKCλ Haploinsufficiency Prevents Diabetes by a Mechanism Involving Alterations in Hepatic Enzymes. Molecular Endocrinology, 2014, 28, 1097-1107.	3.7	10
14	Akt-Dependent Phosphorylation of Hepatic FoxO1 Is Compartmentalized on a WD40/ProF Scaffold and Is Selectively Inhibited by aPKC in Early Phases of Diet-Induced Obesity. Diabetes, 2014, 63, 2690-2701.	0.3	29
15	Requirements for Pseudosubstrate Arginine Residues during Autoinhibition and Phosphatidylinositol 3,4,5-(PO4)3-dependent Activation of Atypical PKC. Journal of Biological Chemistry, 2014, 289, 25021-25030.	1.6	27
16	Impairment of insulin-stimulated glucose transport and ERK activation by adipocyte-specific knockout of PKC-λ produces a phenotype characterized by diminished adiposity and enhanced insulin suppression of hepatic gluconeogenesis. Adipocyte, 2014, 3, 19-29.	1.3	10
17	Metformin action in human hepatocytes: coactivation of atypical protein kinase C alters 5′-AMP-activated protein kinase effects on lipogenic and gluconeogenic enzyme expression. Diabetologia, 2013, 56, 2507-2516.	2.9	18
18	Atypical protein kinase C in cardiometabolic abnormalities. Current Opinion in Lipidology, 2012, 23, 175-181.	1.2	9

Robert

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19	Correction of metabolic abnormalities in a rodent model of obesity, metabolic syndrome, and type 2 diabetes mellitus by inhibitors of hepatic protein kinase C–ι. Metabolism: Clinical and Experimental, 2012, 61, 459-469.	1.5	32
20	The critical role of atypical protein kinase C in activating hepatic SREBP-1c and NFκB in obesity. Journal of Lipid Research, 2009, 50, 1133-1145.	2.0	50
21	Atypical Protein Kinase C Activity in the Hypothalamus Is Required for Lipopolysaccharide-Mediated Sickness Responses. Endocrinology, 2009, 150, 5362-5372.	1.4	22
22	The Irs1 Branch of the Insulin Signaling Cascade Plays a Dominant Role in Hepatic Nutrient Homeostasis. Molecular and Cellular Biology, 2009, 29, 5070-5083.	1.1	132
23	Muscle-specific knockout of PKC-λ impairs glucose transport and induces metabolic and diabetic syndromes. Journal of Clinical Investigation, 2007, 117, 2289-2301.	3.9	140
24	Repletion of Atypical Protein Kinase C following RNA Interference-mediated Depletion Restores Insulin-stimulated Glucose Transport. Journal of Biological Chemistry, 2006, 281, 17466-17473.	1.6	51
25	Insulin-Sensitive Protein Kinases (Atypical Protein Kinase C and Protein Kinase B/Akt): Actions and Defects in Obesity and Type II Diabetes. Experimental Biology and Medicine, 2005, 230, 593-605.	1.1	164
26	Tissue-Specific Differences in Activation of Atypical Protein Kinase C and Protein Kinase B in Muscle, Liver, and Adipocytes of Insulin Receptor Substrate-1 Knockout Mice. Molecular Endocrinology, 2004, 18, 2513-2521.	3.7	36
27	Activation of Protein Kinase C-Â by Insulin and Phosphatidylinositol-3,4,5-(PO4)3 Is Defective in Muscle in Type 2 Diabetes and Impaired Glucose Tolerance: Amelioration by Rosiglitazone and Exercise. Diabetes, 2003, 52, 1926-1934.	0.3	153
28	Skeletal Muscle Insulin Resistance in Obesity-Associated Type 2 Diabetes in Monkeys Is Linked to a Defect in Insulin Activation of Protein Kinase C-Â/Â/Â. Diabetes, 2002, 51, 2936-2943.	0.3	74
29	Sorbitol activates atypical protein kinase C and GLUT4 glucose transporter translocation/glucose transport through proline-rich tyrosine kinase-2, the extracellular signal-regulated kinase pathway and phospholipase D. Biochemical Journal, 2002, 362, 665.	1.7	30
30	Sorbitol activates atypical protein kinase C and GLUT4 glucose transporter translocation/glucose transport through proline-rich tyrosine kinase-2, the extracellular signal-regulated kinase pathway and phospholipase D. Biochemical Journal, 2002, 362, 665-674.	1.7	48
31	Cbl, IRS-1, and IRS-2 Mediate Effects of Rosiglitazone on PI3K, PKC-Â, and Glucose Transport in 3T3/L1 Adipocytes. Endocrinology, 2002, 143, 1705-1716.	1.4	6
32	Insulin and PIP3 Activate PKC-ζ by Mechanisms That Are Both Dependent and Independent of Phosphorylation of Activation Loop (T410) and Autophosphorylation (T560) Sites. Biochemistry, 2001, 40, 249-255.	1.2	123
33	Glucose Activates Protein Kinase C-ζ/λ through Proline-rich Tyrosine Kinase-2, Extracellular Signal-regulated Kinase, and Phospholipase D. Journal of Biological Chemistry, 2001, 276, 35537-35545.	1.6	63
34	Effects of Adenoviral Gene Transfer of Wild-Type, Constitutively Active, and Kinase-Defective Protein Kinase C-Â on Insulin-Stimulated Glucose Transport in L6 Myotubes. Endocrinology, 2000, 141, 4120-4127.	1.4	31
35	Insulin Activates Protein Kinases C-ζ and C-λ by an Autophosphorylation-dependent Mechanism and Stimulates Their Translocation to GLUT4 Vesicles and Other Membrane Fractions in Rat Adipocytes. Journal of Biological Chemistry, 1999, 274, 25308-25316.	1.6	190
36	Evidence for Involvement of Protein Kinase C (PKC)-ζ and Noninvolvement of Diacylglycerol-Sensitive PKCs in Insulin-Stimulated Glucose Transport in L6 Myotubes*. Endocrinology, 1997, 138, 4721-4731.	1.4	211

Robert

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37	Protein Kinase C-ζ as a Downstream Effector of Phosphatidylinositol 3-Kinase during Insulin Stimulation in Rat Adipocytes. Journal of Biological Chemistry, 1997, 272, 30075-30082.	1.6	406
38	Insulin activates myelin basic protein (p42 MAP) kinase by a protein kinase C-independent pathway in rat adipocytes. FEBS Letters, 1993, 333, 287-290.	1.3	20
39	Protein kinase C downregulation?. Nature, 1992, 360, 305-305.	13.7	10
40	Protein kinase C(19-31) pseudosubstrate inhibition of insulin action in rat adipocytes. FEBS Letters, 1991, 282, 139-142.	1.3	8
41	Potential role of phospholipidâ€signaling systems in insulin action and states of clinical insulin resistance. Diabetes/metabolism Reviews, 1989, 5, 455-474.	0.4	23
42	Glucose-induced synthesis of diacylglycerol de novo is associated with translocation (activation) of protein kinase C in rat adipocytes. FEBS Letters, 1989, 249, 234-238.	1.3	46
43	Studies of in vivo phosphorylated proteins in BC3H-1 myocytes suggest that protein kinase C is involved in insulin action. FEBS Letters, 1989, 244, 177-180.	1.3	16
44	Insulin stimulates the translocation of protein kinase C in rat adipocytes. FEBS Letters, 1989, 257, 337-340.	1.3	68
45	Insulin but not phorbol ester treatment increases phosphorylation of vinculin by protein kinase C in BC3H-1 myocytes. FEBS Letters, 1987, 214, 122-126.	1.3	26
46	Human chorionic gonadotropin activates the inositol 1,4,5-trisphosphate-Ca2+ intracellular signalling system in bovine luteal cells. FEBS Letters, 1986, 208, 287-291.	1.3	27
47	Comparison of Effects of Adrenocorticotropin and Lys-γ <sub>3</sub> Melanocyte-Stimulating Hormone on Steroidogenesis, Adenosine 3′,5′-Monophosphate Production, and Phospholipid Metabolism in Rat Adrenal Fasciculata-Reticularis Cells in Vitro*. Endocrinology, 1983, 112, 129-132.	1.4	50
48	Adrenocorticotropin and Adenosine 3′,5′-Monophosphate Stimulatede NovoSynthesis of Adrenal Phosphatidic Acid by a Cycloheximide-Sensitive, Ca++-Dependent Mechanism*. Endocrinology, 1981, 109, 1895-1901.	1.4	25