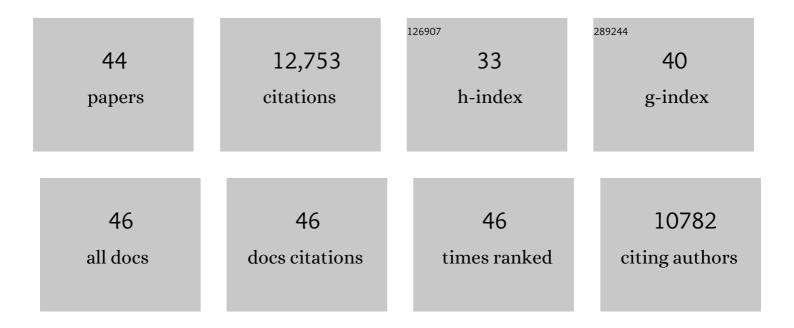
Wade V Welshons

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/12160703/publications.pdf Version: 2024-02-01



#	Article	IF	CITATIONS
1	Estrogen Agonists. , 2018, , 610-618.		0
2	ls it time to reassess current safety standards for glyphosate-based herbicides?. Journal of Epidemiology and Community Health, 2017, 71, 613-618.	3.7	146
3	Concerns over use of glyphosate-based herbicides and risks associated with exposures: a consensus statement. Environmental Health, 2016, 15, 19.	4.0	610
4	Manmade and natural oestrogens: opposite effects on assisted reproduction. Nature Reviews Endocrinology, 2016, 12, 251-252.	9.6	5
5	Holding Thermal Receipt Paper and Eating Food after Using Hand Sanitizer Results in High Serum Bioactive and Urine Total Levels of Bisphenol A (BPA). PLoS ONE, 2014, 9, e110509.	2.5	163
6	Evidence that bisphenol A (BPA) can be accurately measured without contamination in human serum and urine, and that BPA causes numerous hazards from multiple routes of exposure. Molecular and Cellular Endocrinology, 2014, 398, 101-113.	3.2	120
7	Should oral gavage be abandoned in toxicity testing of endocrine disruptors?. Environmental Health, 2014, 13, 46.	4.0	114
8	Bisphenol A (BPA) pharmacokinetics with daily oral bolus or continuous exposure via silastic capsules in pregnant rhesus monkeys: Relevance for human exposures. Reproductive Toxicology, 2014, 45, 105-116.	2.9	53
9	Metabolic disruption in male mice due to fetal exposure to low but not high doses of bisphenol A (BPA): Evidence for effects on body weight, food intake, adipocytes, leptin, adiponectin, insulin and glucose regulation. Reproductive Toxicology, 2013, 42, 256-268.	2.9	242
10	Regulatory decisions on endocrine disrupting chemicals should be based on the principles of endocrinology. Reproductive Toxicology, 2013, 38, 1-15.	2.9	172
11	Report of Very Low Real-World Exposure to Bisphenol A is Unwarranted Based on a Lack of Data and Flawed Assumptions. Toxicological Sciences, 2012, 125, 318-320.	3.1	16
12	Hormones and Endocrine-Disrupting Chemicals: Low-Dose Effects and Nonmonotonic Dose Responses. Endocrine Reviews, 2012, 33, 378-455.	20.1	2,413
13	Bisphenol A in Thermal Paper Receipts: Taylor et al. Respond. Environmental Health Perspectives, 2012, 120, .	6.0	0
14	Similarity of Bisphenol A Pharmacokinetics in Rhesus Monkeys and Mice: Relevance for Human Exposure. Environmental Health Perspectives, 2011, 119, 422-430.	6.0	242
15	Flawed Experimental Design Reveals the Need for Guidelines Requiring Appropriate Positive Controls in Endocrine Disruption Research. Toxicological Sciences, 2010, 115, 612-613.	3.1	72
16	Why Public Health Agencies Cannot Depend on Good Laboratory Practices as a Criterion for Selecting Data: The Case of Bisphenol A. Environmental Health Perspectives, 2009, 117, 309-315.	6.0	268
17	Bisphenol A Data in NHANES Suggest Longer than Expected Half-Life, Substantial Nonfood Exposure, or Both. Environmental Health Perspectives, 2009, 117, 784-789.	6.0	347
18	No effect of route of exposure (oral; subcutaneous injection) on plasma bisphenol A throughout 24h after administration in neonatal female mice. Reproductive Toxicology, 2008, 25, 169-176.	2.9	99

#	Article	IF	CITATIONS
19	Low Phytoestrogen Levels in Feed Increase Fetal Serum Estradiol Resulting in the "Fetal Estrogenization Syndrome―and Obesity in CD-1 Mice. Environmental Health Perspectives, 2008, 116, 322-328.	6.0	91
20	Human exposure to bisphenol A (BPA). Reproductive Toxicology, 2007, 24, 139-177.	2.9	2,344
21	Estradiol and Bisphenol A Stimulate Androgen Receptor and Estrogen Receptor Gene Expression in Fetal Mouse Prostate Mesenchyme Cells. Environmental Health Perspectives, 2007, 115, 902-908.	6.0	119
22	Large effects from small exposures. II. The importance of positive controls in low-dose research on bisphenol A. Environmental Research, 2006, 100, 50-76.	7.5	226
23	Estrogen receptors in membrane lipid rafts and signal transduction in breast cancer. Molecular and Cellular Endocrinology, 2006, 246, 91-100.	3.2	92
24	Large Effects from Small Exposures. III. Endocrine Mechanisms Mediating Effects of Bisphenol A at Levels of Human Exposure. Endocrinology, 2006, 147, s56-s69.	2.8	829
25	Implications for human health of the extensive bisphenol A literature showing adverse effects at low doses: A response to attempts to mislead the public. Toxicology, 2005, 212, 244-252.	4.2	48
26	The importance of appropriate controls, animal feed, and animal models in interpreting results from low-dose studies of bisphenol A. Birth Defects Research Part A: Clinical and Molecular Teratology, 2005, 73, 140-145.	1.6	59
27	Large effects from small exposures. I. Mechanisms for endocrine-disrupting chemicals with estrogenic activity Environmental Health Perspectives, 2003, 111, 994-1006.	6.0	770
28	Bisphenol A is released from used polycarbonate animal cages into water at room temperature Environmental Health Perspectives, 2003, 111, 1180-1187.	6.0	261
29	Low-dose bioactivity of xenoestrogens in animals: fetal exposure to low doses of methoxychlor and other xenoestrogens increases adult prostate size in mice. Toxicology and Industrial Health, 1999, 15, 12-25.	1.4	140
30	Developmental effects of estrogenic chemicals are predicted by an in vitro assay incorporating modification of cell uptake by serum. Journal of Steroid Biochemistry and Molecular Biology, 1999, 69, 343-357.	2.5	68
31	A Physiologically Based Approach To the Study of Bisphenol a and Other Estrogenic Chemicals On the Size of Reproductive Organs, Daily Sperm Production, and Behavior. Toxicology and Industrial Health, 1998, 14, 239-260.	1.4	708
32	[The Importance of Protocol Design and Data Reporting to Research on Endocrine Disruption]: Response. Environmental Health Perspectives, 1998, 106, A316.	6.0	4
33	Lithium-stimulated proliferation and alteration of phosphoinositide metabolites in MCF-7 human breast cancer cells. Journal of Cellular Physiology, 1995, 165, 134-144.	4.1	33
34	Relationship of growth stimulated by lithium, estradiol, and EGF to phospholipase C activity in MCF-7 human breast cancer cells. Breast Cancer Research and Treatment, 1995, 34, 265-277.	2.5	13
35	Nuclear vs translocating steroid receptor models and the excluded middle. Endocrine, 1995, 3, 1-4.	2.2	8
36	pH-Dependent Cytotoxicity of Contaminants of Phenol Red for MCF-7 Breast Cancer Cells*. Endocrinology, 1991, 129, 3321-3330.	2.8	36

WADE V WELSHONS

#	Article	IF	CITATIONS
37	A Sensitive Bioassay for Detection of Dietary Estrogens in Animal Feeds. Journal of Veterinary Diagnostic Investigation, 1990, 2, 268-273.	1.1	63
38	Hormone Receptor Assays: Clinical Usefulness in the Management of Carcinoma of the Breast. CRC Critical Reviews in Clinical Laboratory Sciences, 1988, 26, 97-152.	1.0	54
39	Adaptation of estrogen-dependent MCF-7 cells to low estrogen (phenol red-free) culture. European Journal of Cancer & Clinical Oncology, 1987, 23, 1935-1939.	0.7	110
40	Estrogen Receptors as Nuclear Proteins. Advances in Experimental Medicine and Biology, 1987, 230, 13-29.	1.6	1
41	Nuclear Location of Estrogen Receptors. , 1986, , 97-147.		2
42	Evolution of a Model of Estrogen Action. , 1986, 42, 297-329.		35
43	THE RAT PITUITARY ESTROGEN RECEPTOR: ROLE OF THE NUCLEAR RECEPTOR IN THE REGULATION OF TRANSCRIPTION OF THE PROLACTIN GENE AND THE NUCLEAR LOCALIZATION OF THE UNOCCUPIED RECEPTOR. , 1985, , 539-562.		4
44	Nuclear localization of unoccupied oestrogen receptors. Nature, 1984, 307, 747-749.	27.8	928