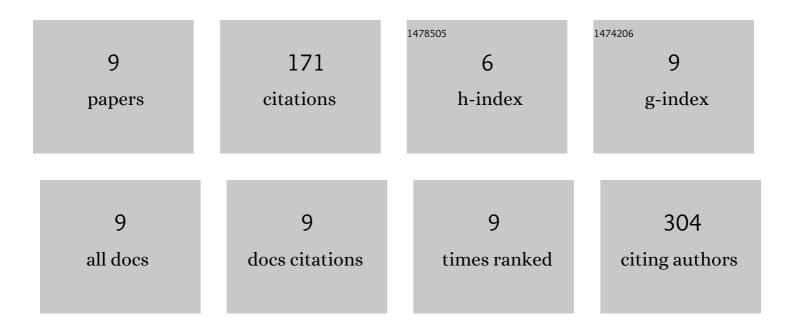
## Kavitha Swaminathan

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/11754394/publications.pdf Version: 2024-02-01



#	Article	IF	CITATIONS
1	The effects of changes in glutathione levels through exogenous agents on intracellular cysteine content and protein adduct formation in chronic alcohol-treated VL17A cells. Toxicology Mechanisms and Methods, 2017, 27, 128-135.	2.7	3
2	GSH protects against oxidative stress and toxicity in VL-17A cells exposed to high glucose. European Journal of Nutrition, 2015, 54, 223-234.	3.9	12
3	Modulation of GSH with exogenous agents leads to changes in glyoxalase 1 enzyme activity in VL-17A cells exposed to chronic alcohol plus high glucose. Food and Function, 2014, 5, 345-358.	4.6	2
4	Inhibition of CYP2E1 leads to decreased malondialdehyde–acetaldehyde adduct formation in VL-17A cells under chronic alcohol exposure. Life Sciences, 2013, 92, 325-336.	4.3	22
5	Chronic ethanol and high glucose inducible CYP2E1 mediated oxidative stress leads to greater cellular injury in VL-17A cells: a potential mechanism for liver injury due to chronic alcohol consumption and hyperglycemia. Toxicology Research, 2013, 2, 245.	2.1	1
6	Increased oxidative stress and toxicity in ADH and CYP2E1 overexpressing human hepatoma VL-17A cells exposed to high glucose. Integrative Biology (United Kingdom), 2012, 4, 550.	1.3	17
7	In Vitro Evidence for Chronic Alcohol and High Glucose Mediated Increased Oxidative Stress and Hepatotoxicity. Alcoholism: Clinical and Experimental Research, 2012, 36, 1004-1012.	2.4	21
8	Elevated glutathione level does not protect against chronic alcohol mediated apoptosis in recombinant human hepatoma cell line VL-17A over-expressing alcohol metabolizing enzymes – Alcohol dehydrogenase and Cytochrome P450 2E1. Toxicology in Vitro, 2011, 25, 969-978.	2.4	11
9	Apoptosis in HepG2 cells exposed to high glucose. Toxicology in Vitro, 2010, 24, 387-396.	2.4	82