frontiers | Frontiers in Pharmacology

TYPE Correction
PUBLISHED 08 September 2023
DOI 10.3389/fphar.2023.1274076

OPEN ACCESS

EDITED AND REVIEWED BY
Zhaoshi Bai,
The Affiliated Cancer Hospital of Nanjing Medical University, China

*CORRESPONDENCE
Nandita Mishra,
nanditamishra@am.amrita.edu

†These authors have contributed equally to this work and share first authorship

RECEIVED 07 August 2023
ACCEPTED 25 August 2023
PUBLISHED 08 September 2023

CITATION
Hanson S, Dharan A, V. JP, Pal S, Nair BG, Kar R and Mishra N (2023), Corrigendum: Paraptosis: a unique cell death mode for targeting cancer.
Front. Pharmacol. 14:1274076.
doi: 10.3389/fphar.2023.1274076

Corrigendum: Paraptosis: a unique cell death mode for targeting cancer

Sweata Hanson[1†], Aiswarya Dharan[1†], Jinsha P. V.[1], Sanjay Pal[1], Bipin G. Nair[1], Rekha Kar[2] and Nandita Mishra[1*]

[1]School of Biotechnology, Amrita Vishwa Vidyapeetham, Kollam, Kerala, India, [2]Department of Cell Systems and Anatomy, UT Health San Antonio, San Antonio, TX, United States

KEYWORDS

cancer, paraptosis, programmed cell death, alternative cell death, cancer therapy, apoptosis

A Corrigendum on
Paraptosis: a unique cell death mode for targeting cancer

by Hanson S, Dharan A, P. V. J, Pal S, Nair BG, Kar R and Mishra N (2023). Front. Pharmacol. 14: 1159409. doi: 10.3389/fphar.2023.1159409

In the published article, there was an error in Table 3 as published. The cell line type and the signalling pathway and mechanism attributed to the article by **Garrido-Armas et al. (2018)** was incorrect. The corrected Table 3 appear below.

The authors apologize for this error and state that this does not change the scientific conclusions of the article in any way. The original article has been updated.

Publisher's note

All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher.

TABLE 3 Paraptosis-inducing compounds against cancer cell lines.

Sl. No:	Compounds	Cell line type	Cell line	Signalling pathway and mechanism	References
1. Breast					
i)	Curcumin	Melanocyte	MDA-MB-434S	Inhibition of mitochondrial Na^+/Ca^{2+} exchanger (mNCX) and proteasome, pERK1/2↑, p-JNKs↑, Alix↓	Yoon et al. 2010 (2012)
		Epithelial	MDA-MB-231, HS578T		
ii)	Dimethoxy curcumin	Melanocyte	MDA-MB-434S	Proteasomal inhibition and ER stress, pERK1/2↑, p-JNKs ↑, Alix↓	Yoon et al. (2014a)
		Epithelial	MDA-MB-231, HS578T, MCF-7		
iii)	Celastrol	Melanocyte	MDA-MB-434S	Ca^{2+} overload, proteasomal inhibition via ER stress, pERK1/2↑, p-JNKs ↑, p-p38	Yoon et al. (2014)
		Epithelial	MCF-7		
iv)	15d-PGJ2	Epithelial	MDA-MB-231	Disruption of sulfhydryl homeostasis, ER stress, pERK1/2↑	Kar et al. (2009)
v)	Manumycin A	Epithelial	MDA-MB-231, BT-20	ER stress, accumulation of ubiquitinated proteins, p21↑, p27 ↑, PTEN ↑	Singha et al. (2013)
		Lymphoblast	HCC1937		
vi)	Withaferin A	Epithelial	MDA-MB-231, MCF-7	ER stress, ROS production, Alix↓	Ghosh et al. (2016)
vii)	Deoxyelephantopin derivative (DETD)	Epithelial	MDA-MB-231	Oxidative and ER stress, p-JNK↑	Shiau et al. (2017)
viii)	Chalcomoracin	Epithelial	MDA-MB-231	ROS production, PINK1 ↑, Alix ↓, p-ERK↑	Han et al. (2018)
ix)	6-Shogaol	Epithelial	MDA-MB-231	Proteasomal inhibition, ER stress	Nedungadi, et al. (2018)
x)	Plumbagin	Epithelial	MDA-MB-231	Disruption of sulfhydryl homeostasis and inhibition of proteasome	Binoy et al. (2019)
xi)	2′-hydroxy-retrochalcone	Epithelial	MDA-MB-231	Proteasomal dysfunction and ER stress	Nedungadi et al. (2021)
xii)	Indirubin-3′-monoxime (I3M)	Epithelial	MDA-MB-231	Proteasomal dysfunction and ER stress-mediated Ca^{2+} release.	Dilshara et al. (2021)
xiii)	Cannabinoids (C6 combination)	Epithelial	MDA-MB-231, MCF-7	ER stress (GRP78 increase)	Schoeman et al. (2020)
xiv)	Gambogic Acid	Epithelial	MDA-MB-453, MDA-MB-468, MDA-MB-435S	Disruption of thiol proteostasis	Seo et al. (2019)
		Melanocyte			

(Continued on following page)

10.3389/fphar.2023.1274076

TABLE 3 (*Continued*) Paraptosis-inducing compounds against cancer cell lines.

Sl. No:	Compounds	Cell line type	Cell line	Signalling pathway and mechanism	References
xv)	5,7-dibromo-8-(methoxymethoxy)-2-methylquinoline (HQ-11)	Epithelial	MDA-MB-231, MCF-7	ER stress, proteasomal inhibition, pERK↑	Ma et al. (2022)
xvi)	Glabridin	Epithelial	MDA-MB-231, MCF-7	ER stress, poly ubiquitinated protein accumulation, proteasome suppression, ROS production, MMP loss	Cui and Cui (2022)
xvii)	Isoxazole derivative of usnic acid	Epithelial	MDA-MB-231, MCF-7	ER stress, IP3R channel activation	Pyrczak-Felczykowska et al. (2022)
xviii)	Derivative of pyrazolo[3,4-*h*]quinoline scaffold (YRL1091)	Epithelial	MDA-MB-231, MCF-7	ER stress, accumulation of ubiquitinated proteins, ROS production, ERK↑, JNK↑, Alix↓	Nguyen et al. (2022)
xix)	Ginger extract	Epithelial	MDA-MB-231	ER stress, mitochondrial dysfunction, AIF translocation and DNA damage	Nedungadi et al. (2021)
xx)	Disulfiram oxy-derivatives	Epithelial	MCF-7	ER stress, mitochondrial damage, 20S proteasome inhibition and actin depolymerization at later stages	Solovieva et al. (2022)
2. Brain					
i)	Curcumin	Glioblastoma	A172	via microRNAs, AKT-Insulin, and p53-BCL2 networks, and AKT protein level reduction was confirmed	Garrido-Armas et al. (2018)
ii)	Ophiobolin A	Pleomorphicastrocytoid, Neuronal, Fibroblast, Fibroblast) Fibroblast	U373-MG, U251N, U251MG, A172	ER stress, NAC inhibition, decrease of BKCa channel	Bury et al. (2013)
			T98G		
iii)	Oligomeric Procyanidins	Epithelial	U-87	Extracellular Ca^{2+} influx, pERK1/2↑, p-p38 ↑	(Zhang et al., 2010)
iv)	Paclitaxel	Epithelial	U-87	CHX has no effect, MEK, p38 and JNK pathways are not involved	Sun et al. (2010)
v)	Yessotoxin	Muscle cells from intracranial tumor	BC3H1	ER and mitochondrial swelling, p-JNK↑	Korsnes et al. (2011)
vi)	1-Desulfo Yessotoxin	Muscle cells from intracranial tumor	BC3H1	ER and mitochondrial swelling, p-p38↑	Korsnes et al. (2013)
vii)	Xanthohumol	Epithelial	SH-SY5Y	ER stress and LC3B upregulation, p38 ↑	Mi et al. (2017)

(Continued on following page)

10.3389/fphar.2023.1274076

TABLE 3 (*Continued*) Paraptosis-inducing compounds against cancer cell lines.

Sl. No:	Compounds	Cell line type	Cell line	Signalling pathway and mechanism	References
3. Blood					
i)	Honokiol	Lymphoblast	K562	ROS generation ROS generation, ER stress, LC3 upregulation, mTOR and MAPK activated	Liu et al. (2021), Wang et al. (2013)
		Promyelocyte	NB4		
ii)	Xanthohumol	Promyeloblast	HL-60	ER stress and LC3B upregulation, p38 ↑	Mi et al. (2017)
iii)	Iturin lipopeptide	Lymphoblast	K562	LC3B and p62 upregulation	Zhao et al. (2019)
iv)	Brassinin	Lymphoblast	K562	ROS production, mitochondrial damage, ER stress, and activation of MAPK	Yang et al. (2023)
		Lymphoblast-like	KBM5, LAMA84, and KCL22		
4. Cervical					
i)	Celastrol	Epithelial	HeLa	Proteasome inhibition, Mitochondrial Ca^{2+} overload, pERK1/2↑, p-JNKs ↑, p-p38 ↑	Wang et al. (2012)
ii)	Cyclosporin A	Epithelial	HeLa, SiHa	LC3 upregulation, Cyclophilin B↓, Alix↓	Ram and Ramakrishna (2014)
iii)	8-p-Hydroxybenzoyl tovarol	Epithelial	HeLa	Bip, CHOP, IRE1α and XBP1 upregulation	Zhang et al. (2015)
iv)	Seleno-DL-Cystine	Epithelial	HeLa	Bip and CHOP polyubiquitination upregulation, ROS generation	Wallenberg et al. (2014)
v)	Paclitaxel	Epithelial	HeLa	CHX has no effect, MEK, p38 and JNK are not involved	Sun et al. (2010)
vi)	Wheat germ Agglutinin	Epithelial	HeLa, SiHa, CaSKi	Autophagy-linked FYVE (Alfy) protein inhibition, ER stress, LC3B upregulation	Tsai et al. (2017)
vii)	2′-hydroxy-retrochalcone	Epithelial	HeLa	Proteasomal dysfunction, ER stress, LC3 upregulation	Nedungadi et al. (2021)
5. Thyroid					
i)	Tunicamycin	Epithelial	8505C, CAL62, FRO cell lines	Bip, CHOP, p-PERK and IRE1 upregulation	Kim et al. (2014)
6. Liver					
i)	Hesperidin	Epithelial	HepG2	Mitochondrial dysfunction and Ca^{2+} overload, p-ERK↑	Yumnam et al., 2016)

(Continued on following page)

10.3389/fphar.2023.1274076

TABLE 3 (*Continued*) Paraptosis-inducing compounds against cancer cell lines.

Sl. No:	Compounds	Cell line type	Cell line	Signalling pathway and mechanism	References
ii)	Cis-Nerolidol	Epithelial	HepG2/C3 A	ER stress, increased activity of cytochrome P450 enzymes	Biazi et al. (2017)
iii)	Gambogic Acid	Epithelial; diffusely spreading cells	SNU-449	Proteasomal inhibition and ER stress, ROS independent-mitochondrial depolarization	Seo et al. (2019)
7. Colon					
i)	Curcumin	Epithelial	HCT116	Proteasome inhibition ROS, Mitochondrial Ca^{2+} overload, LC3 upregulation, pERK1/2↑, p-JNKs↑, Alix↓	Lee et al. (2015)
ii)	Celastrol	Epithelial	DLD-1, RKO	Proteasome inhibition, Mitochondrial Ca^{2+} overload, pERK1/2↑, p-JNKs ↑, p-p38 ↑	Yoon et al. (2014)
iii)	15d-PGJ2	Epithelial	HCT116	Disruption of sulfhydryl homeostasis LC3 upregulation, pERK1/2↑	Kar et al. (2009)
iv)	Ginsenoside Rh2	Epithelial	HCT116, SW480	p53 activation, activation of death by antioxidants	Li et al. (2011), Wan et al. (2018)
v)	Protopanaxadiol	Epithelial	HCT116, SW480	Death acceleration by inhibiting ROS generation, NF-κB activated	Wang et al. (2013)
vi)	ɣ-Tocotrienol	Epithelial	SW620 and HCT-8	Wnt signals↓ (β-catenin, cyclin D, c-Jun)	Zhang et al. (2011) (2013)
	δ-Tocotrienol	Epithelial	SW620	Wnt signals↓ (β-catenin, cyclin D, c-Jun)	
vii)	Iturin A-like lipopeptides	Epithelial	Caco-2	ER stress, ROS generation, Ca^{2+} ↑	Zhao et al. (2019)
viii)	Loperamide	Epithelial	DLD-1, SW-480, SW-620, HCT116	ER stress, Ca^{2+} imbalance and CHOP↑	Kim et al. (2019)
ix)	Purified resin glycoside fraction (Pharbitidis Semen)	Epithelial	HT-29 and HCT-116	Chloride intracellular channel-1 activation and intracellular Cl^{-}↑, MAPK activation	Zhu et al. (2019)
8. Prostate					
i)	Curcumin	Epithelial	PC-3M	Proteasome inhibition ROS, Mitochondrial Ca^{2+} overload, LC3 upregulation, pERK1/2↑, p-JNKs↑, Alix↓	Lee et al. (2015)

(Continued on following page)

10.3389/fphar.2023.1274076

TABLE 3 (*Continued*) Paraptosis-inducing compounds against cancer cell lines.

Sl. No:	Compounds	Cell line type	Cell line	Signalling pathway and mechanism	References
ii)	15d-PGJ2	Epithelial	DU145	Disruption of sulfhydryl homeostasis LC3 upregulation, pERK1/2↑	Kar et al. (2009)
iii)	Benzo[a]quinolizidine analogs	Epithelial	PC3	ER stress and LC3B upregulation	Zheng et al. 2016)
iv)	Chalcomoracin	Epithelial	LNCaP, PC-3	ROS generation, ER stress, PINK1 ↑, Alix ↓, p-ERK↑	Han et al, 2018)
v)	δ-Tocotrienol	Epithelial	CRPC cells —DU145, PC-3	ER stress, LC3 and p62 upregulation, p-JNK ↑, p-p38 ↑	Fontana et al. (2020)
9. Ovarian					
i)	Morusin	Epithelial	A2780, HO-8910, SKOV3	Ca^{2+} overload, ROS generation and loss of mitochondrial membrane potential	Xue et al. (2018)
ii)	Elaiophylin	Epithelial	SKOV3, OVCAR8, UWB1.289, SW626	ER stress, SHP2/SOS1/MAPK↑	Li et al. (2022)
10. Lung					
(i)	Cyclosporin A	Epithelial	A549	LC3 upregulation, Cyclophilin B↓, Alix↓	Ram and Ramakrishna (2014)
ii)	Paclitaxel	Epithelial	A549	CHX has no effect, MEK, p38 and JNK are not involved	Guo et al. (2010)
		Epithelial	ASTC-a-1		
iii)	6-Shogaol	Epithelial	A549	Proteasome inhibition, ER stress, ROS generation, LC3 upregulation	Nedungadi et al. (2018)
iv)	Hinokitiol copper complex	Epithelial	A549	Proteasome inhibition, ER stress	Chen et al. (2017)
v)	Chalcomoracin	Epithelial	H460	ER stress, MAPK activation	Han et al. (2018)
		Epithelial	A549		
		Adenocyte	PC-9		
vi)	Paris Saponin II (PSII)	Epithelial	NCI-H460	ER stress, JNK pathway activation	Man et al. (2020)
		Epithelial	NCI-H520		
vii)	Prenylated bibenzyls (*Radula constricta*)	Epithelial	A549, NCI-H1299	ROS elevation and loss in mitochondrial membrane potential	Zhang et al. (2019)

(Continued on following page)

10.3389/fphar.2023.1274076

TABLE 3 (*Continued*) Paraptosis-inducing compounds against cancer cell lines.

Sl. No:	Compounds	Cell line type	Cell line	Signalling pathway and mechanism	References
viii)	Gambogic Acid	Epithelial	NCI-H460	Proteasomal inhibition and ER stress, ROS independent-mitochondrial depolarization	Seo et al. (2019)
ix)	Epimedokoreanin B	Epithelial	A549, NCL-H292	ER stress, autophagosome accumulation, ROS production, loss of MMP, UPR signaling	Zheng et al. (2022)
x)	DHW-221	Epithelial	A549	ER stress, PI3K/mTOR inhibition, MAPK activation	Liu et al. (2022)
xi)	Ginger extract	Epithelial	A549	ER stress, mitochondrial dysfunction, AIF translocation and DNA damage	Nedungadi et al. (2021)
11. Skin					
i)	Cyclosporin A	Keratinocyte	HaCaT	LC3 upregulation, Cyclophilin B↓, Alix↓	(Ram and Ramakrishna (2014)
ii)	δ-tocotrienol	Epithelial	A375	Ca^{2+} overload and ROS generation, MAPK activation	Raimondi et al. (2021)
12. Bone					
i)	Cyclosporin A	Epithelial	U2OS, Saos-2	LC3 upregulation, Cyclophilin B↓, Alix↓	Ram and Ramakrishna (2014)
13. Kidney and Bladder					
i)	Jolkinolide B	Epithelial	T24, UM-UC-3, T24/CDDP	ROS-mediated ER stress, MAPK and ERK activation	Sang et al. (2021)
14. Oral					
i)	Isorhamnetin (3′-Methoxy-3,4′,5,7-tetrahydroxyflavone)	Epithelial	HSC-3, HSC-4, PE/CA-PJ15	↑ROS generation, ERK/MAPK	Chen et al. (2021)
15. Pancreas					
i)	Gambogic Acid	Epithelial	BxPC-3	Proteasomal inhibition and ER stress, ROS- independent mitochondrial depolarization	Seo et al. (2019)
16. Stomach					
i)	Gambogic Acid	Epithelial	SNU-668 (gastric cancer)	Proteasomal inhibition and ER stress, ROS independent-mitochondrial depolarization	Seo et al. 2019)

10.3389/fphar.2023.1274076