Autophagy Inducers

Autophagy is the breakdown of unnecessary or dysfunctional cell components by lysosomes. This process can promote survival during cellular starvation by maintaining cellular energy levels. Autophagy is also a form of programmed cell death that can be initiated by processes such as development, differentiation, neurodegenerative diseases, stress, infection, and cancer¹. Although autophagy may improve survival of starved cells, it is also a tumor suppressor. Autophagy is induced by the presence of irregular proteins produced by chemotherapeutics; degradation of these proteins and organelles limits cell growth². Prolonged autophagy also leads to a high turnover rate of proteins and organelles which may kill cancer cells³. For a selection of compounds that induce autophagy across various disease models including cancer, fibrosis, and pathogenic infection, see the representative list below.

A0820 Acetyl-benzylisothiocyanate-L-cysteine

A4440 Allicin

A4441 Allicin (aqueous)

A6132 Apicidin

A6818 Arenobufagin

A7460 Asparaginase

A9710 AZD-2014

B1653 Benzyl Isothiocyanate

B1996 BEZ235

C0265 Carnosic Acid

C2947 Chlorpromazine Hydrochloride

C5654 Concanavalin A

D0375 Dasatinib Monohydrate

D4802 17-DMAG

E0813 Ecdysterone

E2003 Efavirenz

E6846 Erlotinib Monohydrochloride

E7357 Esomeprazole Potassium

E7356 Esomeprazole Magnesium Trihydrate

E7657 Etoposide

E7658 Etoposide phosphate

E8419 Everolimus

G3461 Ginsenoside F2

H9712 (E)-4-Hydroxytamoxifen

H9711 (Z)-4-Hydroxytamoxifen

H9716 (E,Z)-4-Hydroxytamoxifen

M1744 Melittin

M4454 MLN-4924

N3346 Nilotinib

O0400 Obatoclax

O6932 Oridonin

P3465 Piperine

P3561 Piperlongumine

P4492 PLX4720

P7000 PR-619

P7219 Pseudolaric acid B

P8167 Puromycin Aminonucleoside

P8168 Puromycin Dihydrochloride

P9200 PX-866

S0134 Saikosaponin D

S7603 Stavudine

S8044 R,S-Sulforaphane

T1968 Terpinen-4-ol

V0146 Valsartan

V0352 Vandetanib

W5727Wogonoside

Y4802 YM-201636

References:

- 1. Kobayashi S. Biol Pharm Bull. 2015;38(8):1098-103.
- 2. Mathew R, Karp CM, Beaudoin B, et al. Cell. 2009 Jun 12;137(6):1062-75.
- 3. Yang ZJ, Chee CE, Huang S, et al. Mol Cancer Ther. 2011 Sep;10(9):1533-41.







