TUDCA Research

TUDCA stands for tauroursodeoxycholic acid (a water-soluble bile acid). Here's some research associated with TUDCA:

LIVER

TUDCA improved palmitic acid induced hepatocellular lipotoxicity.
Nutr Metab (Lond). 2020 Jan 30;17:11
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6990600/

TUDCA reduced liver enzymes related to cholestasis (decreased bile flow) and cytolysis.
Hepatology. 1999 Feb;29(2):320-7.
https://pubmed.ncbi.nlm.nih.gov/9918924/

When TUDCA was administered, total fecal bile acid excretion increased markedly.
Hepatology. 1999 Feb;29(2):320-7.
https://pubmed.ncbi.nlm.nih.gov/9918905/

TUDCA stimulates bile flow increase by 250%. TUDCA also improves bile quality by increasing the amount of bile salts in bile.
Am J Physiol Gastrointest Liver Physiol 302: G1035–G1042, 2012.
https://pubmed.ncbi.nlm.nih.gov/22301109/

TUDCA stimulated secretion of ATP by isolated rat hepatocytes and produced measurable increases in ATP in the bile of isolated rat liver.
Biochem. J. (2001)358, 1±5
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1222025/

TUDCA inhibited fungicide induced ER (endoplasmic reticulum) stress and cytotoxicity in a dose-dependent manner. The apoptotic cell death and mitochondrial depolarization were also prevented by TUDCA in this study.
Cells. 2019 Sep 3;8(9):1023.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6770470/

TUDCA alleviates fatty liver by downregulating the expression of lipogenic genes and stopping fatty acid accumulation.
J Dairy Sci. 2019 Aug;102(8):7359-7370
https://pubmed.ncbi.nlm.nih.gov/31155263/

TUDCA helps to greatly diminish damage in liver hepatocytes from trauma induced hemorrhages when you take TUDCA right after injury.
Shock. 2019 Nov;52(5):e76-e84.
https://pubmed.ncbi.nlm.nih.gov/30499877/

TUDCA was shown to attenuate damage from hepatic ischemia reperfusion injury decreasing ER stress though down regulating pro inflammatory cytokines IRE1α/TRAF2/NF-κB pathway to suppress the function of kupffer cells.

Biomed Pharmacother. 2018 Oct;106:1271-1281
https://www.sciencedirect.com/science/article/abs/pii/S0753332217348722

TUDCA helps to restore the following back to normal: protein folding, cell apoptosis and fibronectin and collagen levels from cholestatic liver injury.

Cell Signal. 2018 Nov;51:72-85.
https://pubmed.ncbi.nlm.nih.gov/30044965/

TUDCA was shown to have liver healing benefits in severe liver injury caused by Acetaminophen (APAP) overdose. TUDCA helped to abolish APAP-induced inflammasomes activation, implying that ER stress acts as signaling event leading to the inflammasome activation in hepatocytes stimulated with APAP.

Biochem Pharmacol. 2018 Aug;154:278-292.
https://www.sciencedirect.com/science/article/abs/pii/S0006295218302004

TUDCA could attenuate lipid accumulation in Lis1 deficient hepatocytes in nonalcoholic fatty liver disease.

J Biol Chem. 2018 Apr 6;293(14):5160-5171.
https://pubmed.ncbi.nlm.nih.gov/29475944/

TUDCA increases healthy PUFA levels and alleviates cPLA2, COX, and PGE2 levels in liver tissue treated with tunicamycin.

Hum Exp Toxicol. 2018 Aug;37(8):803-816
https://pubmed.ncbi.nlm.nih.gov/29027487/

TUDCA attenuated liver pathological changes, reduced serum alanine aminotransferase and aspartate aminotransferase level, suppressed ROS activity, decreased TNF-a and IL-1β level, inhibited hepatocyte apoptosis induced, inhibited ER stress in liver in sleep apnea induced chronic intermittent hypoxia.

Exp Ther Med. 2017 Sep;14(3):2461-2468.
https://pubmed.ncbi.nlm.nih.gov/28962181/

TUDCA enhanced the bile acid transporter- and Nrf2-mediated adaptive response.

Free Radic Biol Med. 2017 Nov;112:24-35.
https://pubmed.ncbi.nlm.nih.gov/28688954/

TUDCA exerts a beneficial effect on liver fibrosis in a model of cholestatic liver disease, and suggest that this effect might, at least in part, be attributed to decreased hepatic UPR signaling and apoptotic cell death.

Int J Mol Sci. 2017 Jan 20;18(1):214.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5297843/

TUDCA is safe and as efficacious as UDCA for the treatment of primary biliary cholangitis, and may be better to relieve symptoms than UDCA.
Medicine (Baltimore). 2016 Nov;95(47):e5391
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5134868/

Undernourishment in utero being causatively connected with the risk of NAFLD in later life because it triggers ER stress. When treated with TUDCA significantly improved hepatic steatosis and reduce ER stress.
Congenit Anom (Kyoto). 2017 Nov;57(6):178-183.
https://pubmed.ncbi.nlm.nih.gov/27859643/

The ER stress inhibitor TUDCA could partly relieve the liver damage, as indicated by the restoration of serum ALT and AST levels. Results demonstrated that ER stress may serve as an early warning mechanism of hepatotoxicity, and severe ER stress may lead to apoptosis.
J Toxicol Sci. 2016;41(6):745-756.
https://pubmed.ncbi.nlm.nih.gov/27853103/

We found that TUDCA reversed abnormal autophagy, reduced ER stress, and restored insulin sensitivity in the liver of obese mice and that glycolipid metabolism disorder was also improved via the restoration of defective hepatic autophagy.
Int J Endocrinol. 2015; 2015: 687938.
https://pubmed.ncbi.nlm.nih.gov/26681941/

Hepatoprotective and anti-inflammatory properties of TUDCA against LPS are independent of its ability to improve steatosis in Nonalcoholic fatty liver disease.
Cell Death Dis. 2015 Sep; 6(9): e1879.
https://pubmed.ncbi.nlm.nih.gov/26355342/

TUDCA attenuates hepatocarcinogenesis by suppressing carcinogen-induced ER stress-mediated cell death and inflammation without stimulating tumor progression.
Oncotarget. 2015 Sep 29; 6(29): 28011–28025.
https://europepmc.org/articles/pmc4695041/bin/oncotarget-06-28011-s001.pdf

TUDCA benefits the liver and might help fight liver cirrhosis. A group of people with liver cirrhosis and high liver enzymes took 750 milligrams (mg) of TUDCA for six months. During the treatment process, it was discovered that TUDCA will lower liver enzymes significantly when compared to before the people started taking it. Plus, supplementing with this bile acid was well tolerated and no patient complained of side effects.
J Huazhong Univ Sci Technolog Med Sci. 2013 Apr;33(2):189-194.
https://pubmed.ncbi.nlm.nih.gov/23592128/

An experiment on plants suggests that TUDCA can help to shield cells from stress linked to heavy metals. The studied plants had damage and ER stress after cadmium exposure. Then the researchers exposed them to TUDCA. TUDCA helped to lower the ER stress and damage from the heavy metal.
Plant Cell Physiol. 2016 Jun;57(6):1210-9.
https://academic.oup.com/pcp/article/57/6/1210/2461091

GLUTAMATE

Glutamate is an excitatory neurotransmitter in the CNS that regulates neuronal plasticity and induction of cell death. Cell death induced by glutamate may be involved in chronic neurodegenerative disorders, such as Alzheimer's Disease.
A significant reduction of glutamate-induced apoptosis of cortical neurons was observed in pretreatment with TUDCA.

Journal of Pharmacology and Experimental Therapeutics November 2004, 311 (2) 845-852.
https://jpet.aspetjournals.org/content/311/2/845.abstract

BRAIN

TUDCA shows a similar effect to intermittent fasting in terms of improving cognitive function.

Nat Commun. 2020 Feb 18;11(1):855.
https://pubmed.ncbi.nlm.nih.gov/32071312/

TUDCA rescued the abnormal memory and motor behaviors.

Cell Physiol Biochem. 2020 May 2;54(3):438-456
https://pubmed.ncbi.nlm.nih.gov/32357291/

TUDCA reduced neuronal loss in prion-infected cerebellar slice cultures. TUDCA treatment reduced astrocytosis and prolonged survival in prion-infected mice.

J Virol. 2015 Aug 1; 89(15): 7660–7672.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4505631/

TUDCA improved cerebral blood flow, reduced BBB permeability, inhibited the ER-stress mediated apoptosis, resulting in significantly improved neurological function in early brain injuries.

Brain Res. 2020 Jan 15;1727:146566.
https://pubmed.ncbi.nlm.nih.gov/31778648/

TUDCA crosses the blood-brain barrier

Clinical Neuropharmacology: January-February 2010 - Volume 33 - Issue 1 - p 17-21
https://pubmed.ncbi.nlm.nih.gov/19935406/

For Alzheimer's, TUDCA helps to stop DNA from breaking and your cells to live longer.
It can help prevent memory loss and keep your cognitive function running longer. TUDCA helps reduce the toxic effects of amyloid plaque in your brain as well.
(decreased Aβ production and inhibited the accumulation of Aβ deposits in the brain)

Trends Mol Med. 2008 Feb;14(2):54-62.
https://www.sciencedirect.com/science/article/abs/pii/S1471491408000026

TUDCA benefits people with Huntington's much the same way as Alzheimer's. In addition to protecting your DNA and cognitive function, TUDCA may prevent swelling in the mitochondria.

Proc Natl Acad Sci U S A. 2002 Aug 6; 99(16): 10671–10676.
https://pubmed.ncbi.nlm.nih.gov/12149470/

In this rat study they were looking how TUDCA interacted with 3-nitropropionic acid (3-NP) which exhibit symptomatology and neuropathology reminiscent of Huntington's Disease. What the researchers found is that when TUDCA when given to 3-NP treated rats they exhibited an 80% reduction in apoptosis and in lesion volumes associated with 3-NP administration. This is the first demonstration of how a bile acid can be delivered to the brain and function as a neuroprotectant and thus may offer potential therapeutic benefit in the treatment of certain neurodegenerative diseases.

Exp Neurol. 2001 Oct;171(2):351-60.
https://pubmed.ncbi.nlm.nih.gov/11573988/

Parkinson's disease is characterized by cell death and mitochondrial dysfunction. TUDCA can increase how long your brain cells live and help your mitochondria to work better. It also can decrease the number of dopamine cells you lose over time.

Mol Neurobiol. 2012 Oct;46(2):475-86.
https://pubmed.ncbi.nlm.nih.gov/22773138/

TUDCA can help protect your brain from the effects of a stroke. Brain damage was reduced by 50% because of TUDCA

Proc Natl Acad Sci U S A. 2003 May 13;100(10):6087-92. Epub 2003 Apr 29.
https://pubmed.ncbi.nlm.nih.gov/12721362/

TUDCA helps to prevent the polarization of astrocytes and pro-inflammatory microglia cells and ameliorates neurodegeneration in MS.

J Clin Invest. 2020 Mar 17. pii: 129401.
https://pubmed.ncbi.nlm.nih.gov/32182223/

TUDCA exerts its neuroprotective role in a Parkinson's Disease, by up-regulation of mitochondrial turnover a as neuroprotective mechanism.

Biochim Biophys Acta. 2017 Sep ;1863(9):2171-2181.
https://www.sciencedirect.com/science/article/pii/S0925443917301990

ER stress peaked at 72 h after TBI and that TUDCA abolished ER stress and inhibited p-PERK, p-eIF2a, ATF4, Pten, Caspase-12 and CHOP expression levels. TUDCA also improved neurological function and alleviated brain edema.

Front Cell Neurosci. 2017;11:193.
https://www.frontiersin.org/articles/10.3389/fncel.2017.00193/full

TUDCA is neuroprotective from lipopolysaccharide (LPS) an endotoxin that can trigger different neurological disease. It was shown that TUDCA prevented neuroinflammation, decreased LPS-induced apoptosis, and meliorated synaptic plasticity impairments by increasing BDNF. TUDCA treatment decreases the effects of Parkinson's Disease, most of all negatively modulates neuroinflammation. Additionally, results from cellular models using microglia corroborate TUDCA modulation of ANXA1 synthesis, linking inhibition of neuroinflammation and neuroprotection by TUDCA.

Mol Cell Neurosci. 2019 Apr;96:1-9
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6720125/

TUDCA can alleviate the acute cerebral infarction-induced neurological impairment through mitigating lipid peroxidation and inflammatory response and reducing apoptosis, regulating the Nrf2 signaling pathway.

Eur Rev Med Pharmacol Sci. 2019 Jan;23(1):343-351.
https://pubmed.ncbi.nlm.nih.gov/30657576/

TUDCA was shown to work like an Anti-depressant because of it's ability to inhibit neuroinflammatory response by down regulating proinflammatory cytokines induced by lipopolysaccharides such as interleukin-6 and Tumor necrosis factor in the brain.

Pharmacology. 2019;103(1-2):93-100.
https://pubmed.ncbi.nlm.nih.gov/30517939/

Administration of TUDCA reduced muscle denervation in ALS.

Mol Ther. 2019 Jan 2;27(1):87-101
https://pubmed.ncbi.nlm.nih.gov/30446391/

TUDCA reversed the changes in the levels of synaptic plasticity proteins after anesthesia use.

Front Psychiatry. 2018 Aug 2;9:332.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6083612/

TUDCA was also effective in the prevention of typical parkinsonian symptoms such as spontaneous activity, ability to initiate movement and tremors. TUDCA prevented MPTP-induced decrease of dopaminergic fibers and ATP levels, mitochondrial dysfunction and neuroinflammation.

Mol Neurobiol. 2018 Dec;55(12):9139-9155
https://pubmed.ncbi.nlm.nih.gov/29651747/

TUDCA was shown to have benefits for depression through the attenuation of neuroinflammation, as TUDCA treatment (200 mg/kg) normalized the levels of tumor necrosis factor-α and interleukin-6 in both hippocampus and prefrontal cortex.

Fundam Clin Pharmacol. 2018 Aug;32(4):363-377
https://pubmed.ncbi.nlm.nih.gov/29578616/

TUDCA significantly activates the neuronal autophagic expression in rats with acute spinal cord injury to inhibit the apoptosis of nerve cells; therefore, it has a protective effect on neurons.

Eur Rev Med Pharmacol Sci. 2018 Feb;22(4):1133-1141.
https://pubmed.ncbi.nlm.nih.gov/29509267/

TUDCA has shown promising results in spinal cord injuries to support recovery through suppressing inflammatory cytokine, decreased LPS stimulated inflammation, and suppressing TNF-a.

Sci Rep. 2018 Feb 16;8(1):3176.
https://pubmed.ncbi.nlm.nih.gov/29453346/

In Marinesco-Sjögren syndrome (MSS) people given TUDCA were found to have reduces ER stress-induced death of MSS patient-derived cells.

J Neurol Sci. 2018 Feb 15;385:49-56.
https://pubmed.ncbi.nlm.nih.gov/29406913/

TUDCA administration can improve motor function and reduce secondary injury and lesion area after spinal cord injury.

Cell Stress Chaperones. 2018 Jul;23(4):551-560.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6045539/

Inhibition of ER stress with TUDCA suppresses restraint stress-induced seizure susceptibility.

Neurochem Int. 2017 Nov;110:25-37.
https://pubmed.ncbi.nlm.nih.gov/28887093/

Results indicate that TUDCA may attenuate early brain injury via Akt pathway activation, deceasing inflammation, and brain inflammation.

Front Cell Neurosci. 2017 Jul 6;11:193.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5498474/

TUDCA exerts its neuroprotective role in a parkin-dependent manner. The results point to the up-regulation of mitochondrial turnover by TUDCA as a novel neuroprotective mechanism of this molecule, and contribute to the validation of TUDCA clinical application in Parkinson's Disease.

Exp Neurol. 2017 Sep;295:77-87.
https://pubmed.ncbi.nlm.nih.gov/28552716/

TUDCA abolishes unfolded protein response activation, which results in improvement of axonal degeneration and its associated locomotor impairment in neurodegenerative disorders

Acta Neuropathol. 2017 Feb;133(2):283-301
https://pubmed.ncbi.nlm.nih.gov/28004277/

TUDCA can inhibit the expression of caspase-12 in rat neurons after spinal cord injury, reduce cell apoptosis, and exert neuroprotective effects on rat secondary nerve injuries after a spinal cord injury.

Int J Clin Exp Pathol. 2015 Dec 1;8(12):15871-8.
https://pubmed.ncbi.nlm.nih.gov/26884858/

TUDCA inhibits multiple proteins involved in apoptosis and upregulates cell survival pathways. TUDCA exhibits anti-inflammatory effects in models of neuroinflammation and attenuates neuronal loss in chronic neurodegenerative diseases.

Neurocrit Care. 2016 Aug;25(1):153-66. (Review Study)
https://pubmed.ncbi.nlm.nih.gov/26759227/

Studies show that TUDCA may act as a potent protector of brain cells. In one study, researchers gave TUDCA to animals with brain plaque and memory loss. After six months, they found that the amount of brain plaque was significantly reduced. TUDCA not only helped remove the plaque, but also helped prevent its production. The researchers also observed that the animals' memory notably improved

Mol Neurobiol. 2012 Jun;45(3):440-54.
https://pubmed.ncbi.nlm.nih.gov/22438081/

TUDCA administration, either before or after MPTP, significantly reduced the swimming latency, improved gait quality, and decreased foot dragging. Importantly, TUDCA was also effective in the prevention of typical parkinsonian symptoms such as spontaneous activity, ability to initiate movement and tremors. Accordingly, TUDCA prevented MPTP-induced decrease of dopaminergic fibers and ATP levels, mitochondrial dysfunction and neuroinflammation.

Mol Neurobiol. 2018 Dec;55(12):9139-9155.
https://pubmed.ncbi.nlm.nih.gov/29651747/

In a lab study, animals with brain injury from a stroke took TUDCA one hour after it occurred. After seven days, their brains had a 50% decrease of harmed cells from the stroke. Those are noteworthy results considering the animals only received one dosage

Global Advances in Health and Medicine May 1, 2014 Volume: 3 issue: 3, page(s): 58-69
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4030606/

CARDIOVASCULAR

TUDCA sharply reduces the number of cells that die during a heart attack. It can also help to heal your cells that were damaged from a heart attack.

Am J Chin Med. 2007;35(2):279-95.
https://pubmed.ncbi.nlm.nih.gov/17436368/

TUDCA when given after a heart attack has been shown to shut down enzymes that cause improper protein folding, minimize scaring, and helps to have less myocardial dysfunction.

J Thorac Dis. 2018 Sep;10(9):5283-5297.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6196212/

High glucose levels damage your endothelial cells. These are the cells that line the inside of all your organs and blood vessels. This damage increases your chance of cardiovascular disease. TUDCA helps protect these special cells that line your heart and arteries. It reduces oxidative stress. It prevents your endothelial cells from becoming dysfunctional in the presence of too much glucose.

Clin Sci (Lond). 2016 Nov 1; 130(21): 1881–1888.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5507333/

Treatment of obese mice with TUDCA led to a decrease in cardiac hypertrophy, decreased hypertension, and normalization of cardiac contractility.

J Mol Cell Cardiol. 2011 Jan; 50(1): 107–116.
https://www.jmmc-online.com/article/S0022-2828(10)00402-5/fulltext

TUDCA markedly decreased systolic blood pressure in the hypertensive animals.

Mol Biol Rep. 2020 Mar;47(3):2243-2252.
https://pubmed.ncbi.nlm.nih.gov/32072406/

TUDCA mitigated the Met syndrome-induced cardiovascular complications through up-regulating survival markers and downregulating ER and oxidative stress markers.

J Mol Cell Cardiol. 2020 Apr 18;143:15-25.
https://www.x-mol.com/paper/1252055045706244096?recommend

TUDCA significantly improved vasodilation by helping to regulate calcium levels which decrease as we age because level of Calreticulin decrease as we age.

Am J Physiol Heart Circ Physiol. 2020 May 1;318(5):
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7346535/

TUDCA use may ameliorate cardiac contractility through improving endoplasmic reticulum stress-associated calcium mishandling, apoptosis, inflammation and fibrosis, thereby offering therapeutic potential in hypertension-induced cardiac dysfunction.

J Pharm Pharmacol. 2019 Dec;71(12):1809-1821.
https://pubmed.ncbi.nlm.nih.gov/31579948/

TUDCA improved endothelium-dependent dilation in diabetic mesenteric arteries, without affecting endothelium-independent dilation. TUDCA also reduced arterial stiffness and was associated with reductions in ER stress markers in aortic and perivascular adipose tissue.

J Vasc Res. 2017 ;54(5):280-287
https://pubmed.ncbi.nlm.nih.gov/28930750/

Hemorrhagic shock will decrease blood levels of TUDCA leading to liver injury and with supplementation of TUDCA it attenuated the liver injury.

Shock. 2020 Feb;53(2):217-222.
https://pubmed.ncbi.nlm.nih.gov/30998645/

TUDCA inhibited dedifferentiation of vascular smooth muscle cells by decreasing ER stress and reduced in-stent restenosis, possibly through downregulation of the improper protein folding.

Cardiovasc Drugs Ther. 2019 Feb;33(1):25-33.
https://pubmed.ncbi.nlm.nih.gov/30663009/

Taking TUDCA after getting off a statin medication can help to improve muscle endurance by reducing muscle inflammation caused by the ER stress from the statin medications use.

Biochem Biophys Res Commun. 2019 Jan 15;508(3):857-863.
https://pubmed.ncbi.nlm.nih.gov/30528737/

When looking at the negative effect of cardiovascular disease induced by obesity and diabetes TUDCA was shown to be a strategy for prevention and treatment because of the ability of TUDCA to help reduce inflammation, reduce ROS, and improve insulin sensitivity.

Front Pharmacol. 2018 Oct 26;9:1226.
https://pubmed.ncbi.nlm.nih.gov/30416448/

TUDCA-mediated inhibition on H2O2-induced oxidative stress and cardiomyocytes apoptosis was through suppressing ER stress, and TUDCA possesses the potential to be developed as therapeutic tool in clinical use for cardiovascular diseases.

Dose Response. 2018 Jul 18;16(3):1559325818782631
https://pubmed.ncbi.nlm.nih.gov/30038553/

In heart attacks it was shown that TUDCA significantly decrease the infarct size, reduced cell apoptosis and the inflammation as indicated by the reduction in macrophages and neutrophil infiltration.

Pflugers Arch. 2018 Mar;470(3):471-480.
https://pubmed.ncbi.nlm.nih.gov/29288332/

TUDCA in treatment of heart diseases related to transverse aortic constriction help recovery by reduced cardiac hypertrophy, reducing myocardial fibrosis and collagen deposition, reduced cardiac apoptosis.

PLoS One. 2017 Apr 20;12(4):e0176071.
https://pubmed.ncbi.nlm.nih.gov/28426781/

TUDCA attenuates angiotensin II induced Abdominal aortic aneurysm formation by inhibiting ER stress mediated apoptosis.

Eur J Vasc Endovasc Surg. 2017 Mar;53(3):337-345.
https://pubmed.ncbi.nlm.nih.gov/27889204/

There was a decrease in systolic blood pressure in the hypertensive group treated with TUDCA. TUDCA an ER stress inhibitor, normalized myogenic responses and endothelium-dependent relaxation in hypertensive group. This study showed the potential of TUDCA use for hypertension.

Sci Rep. 2016 Aug 23;6:31925.
https://pubmed.ncbi.nlm.nih.gov/27550383/

Blocking the activation of the transiently activated UPR pathway by TUDCA prevented cardiac fibrosis, and improved prognosis. These findings offer a window for additional interventions that can preserve heart function.

PLoS One. 2016; 11(7): e0159682.
https://pubmed.ncbi.nlm.nih.gov/27441395/

TUDCA's beneficial effects of ER stress inhibition during exposure to intermittent hypoxia suggest a potential new therapeutic approach to improve myocardial susceptibility to tissue damage in obstructive sleep apnea patients.

Int J Cardiol. 2016 May 1;210:45-53.
https://pubmed.ncbi.nlm.nih.gov/26922713/

TUDCA was shown to reduce cardiomyocyte apoptosis in the hearts of nephrectomized mice. The study concluded it was through TUDCA's ability to reduce ER stress, down regulating harmful signaling proteins as to why it can be a viable treatment for uremic cardiomyopathy or other cardiac diseases.

Cell Physiol Biochem. 2016;38(1):141-52.
https://pubmed.ncbi.nlm.nih.gov/26765262/

When your heart is under too much pressure, it can enlarge and possibly malfunction. One study on animals suggests that TUDCA can decrease heart enlargement due to the effects of high blood pressure. TUDCA may also help to prevent scar tissue formation in the heart from the pressure.

PLoS One. 2017 Apr 20;12(4):e0176071.
https://pubmed.ncbi.nlm.nih.gov/28426781/

High blood sugar levels can make the cells lining the heart dysfunctional because of endoplasmic reticulum stress and can contribute to heart disease. A study on 12 healthy people suggests that TUDCA may help stabilize these cells. The people consumed a sugar drink and then TUDCA one hour later. The TUDCA seemed to prevent the cells lining the heart from becoming faulty.

Clin Sci (Lond). 2016 Nov 1;130(21):1881-8.
https://pubmed.ncbi.nlm.nih.gov/27503949/

DIABETES

Several reports confirm the ability of TUDCA to improve the hyperglycemia associated with both types of diabetes.

Science. 2006 Aug 25; 313(5790): 1137–1140.
https://pubmed.ncbi.nlm.nih.gov/16931765/

TUDCA improved systemic glucose homeostasis in animals placed on a high-fat diet.

FEBS Lett. 2011 Feb 4; 585(3): 539–544.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3133948/

TUDCA improved metabolic and neurodevelopmental deficits and reversed leptin resistance in the offspring of obese mice.

PLoS Biol. 2020 Mar 12;18(3):e3000296
https://pubmed.ncbi.nlm.nih.gov/32163401/

TUDCA improved diabetes induced severe albuminuria and podocyte injury in the Kidneys.
Biochem Biophys Res Commun. 2020 Apr 30;525(2):319-325.
https://pubmed.ncbi.nlm.nih.gov/32089264/

TUDCA treatment recovered islet volume, serum insulin level, and abdominal fat storage.
Int J Mol Sci. 2019 Oct 25;20(21):5317.
https://pubmed.ncbi.nlm.nih.gov/31731478/

TUDCA remarkably diminishes the aggravated diabetic neuropathy features caused by ERp44 depletion which can then cause improper protein folding .
Biochem Biophys Res Commun. 2018 Oct 12;504(4):921-926.
https://pubmed.ncbi.nlm.nih.gov/30224065/

TUDCA may be helpful to counteract obesity-induced hyperinsulinemia through increasing insulin clearance.
Sci Rep. 2017 Nov 1;7(1):14876.
https://pubmed.ncbi.nlm.nih.gov/29093479/

With TUDCA taken for 6 weeks it reduced blood pressure (135±4 versus 151±4 mm Hg), albumin excretion, ER and oxidative stress, and glomerular injury, while increasing glomerular filtration rate in hypertensive-diabetic kidneys.
Hypertension. 2017 May;69(5):879-891
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5395247/

TUDCA was found to help regulate fatty acid breakdown and also to help regulate insulin resistance.
Exp Biol Med (Maywood). 2017 Feb; 242(4): 441–447.
https://pubmed.ncbi.nlm.nih.gov/27811171/

For eight weeks, being treated with TUDCA at 250 mg/kg twice a day, diabetic mice had significantly reduced blood glucose, albuminuria, and attenuated renal histopathology. TUDCA shows potential as a therapeutic target for the prevention and treatment of Diabetic Neuropathy.
Nutrients. 2016 Oct; 8(10): 589.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5083977/

Study suggest that hyperglycaemia-induced endothelial dysfunction can be mitigated by oral administration of TUDCA, thus supporting the hypothesis that ER stress may contribute to endothelial dysfunction during postprandial hyperglycaemia.
Clin Sci (Lond). 2016 Nov 1;130(21):1881-8.
https://pubmed.ncbi.nlm.nih.gov/27503949/

TUDCA in pancreatic β-cells increases insulin secretion only at high glucose concentrations by a mechanism that is mediated by the cAMP/PKA/CREB pathway.
Metabolism. 2016 Mar;65(3):54-63.
https://pubmed.ncbi.nlm.nih.gov/26892516/

Animal studies indicate that TUDCA may help lower blood sugar. Type 1 diabetic animals received TUDCA for 24 days. After only 15 days, tests showed that their fasting blood sugar levels had lowered by 43%. In this study, researchers hypothesize the animals had more insulin because their pancreas cells started to produce insulin again.

Front. Physiol., 15 May 2019
https://pubmed.ncbi.nlm.nih.gov/31156448/

DIET & DIGESTIVE TRACT

TUDCA attenuates the progression of high-fat diet-induced non-alcoholic fatty liver disease in mice by improving gut inflammation, improving intestinal barrier function, decreasing intestinal fat transport and modulating gut microbiome composition.

British Journal of Pharmacology 2018 Feb; 175(3): 469–484.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5773980/

Lipogenesis-associated factors induced by high fructose were inhibited by TUDCA.

Chin Med J (Engl). 2018 Oct 5;131(19):2310-2319
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6166463/

Intermittent fasting raises blood levels of TUDCA to help with protection from diabetic retinopathy.

Diabetes. 2018 Sep;67(9):1867-1879.
https://pubmed.ncbi.nlm.nih.gov/29712667/

TUDCA helped to alleviates adipose tissue inflammation in obesity by inhibit ER stress, and thereby modify metabolic dysfunction.

Sci Rep. 2016; 6: 27486.
https://pubmed.ncbi.nlm.nih.gov/27271106/

DIGESTIVE TRACT

Caspase-3 activation is an early marker of ulcerative colitis that is prevented by TUDCA treatment. TUDCA also acted as a gut barrier protector.

Lab Invest. 2014 Dec;94(12):1419-30.
https://pubmed.ncbi.nlm.nih.gov/25310532/

TUDCA reduced mortality rates, prolonged survival times, significantly diminished intestinal damage, and inhibited intestinal inflammation in the mouse model of Neonatal necrotizing enterocolitis.

Int Immunopharmacol. 2019 Sep;74:105665s
https://pubmed.ncbi.nlm.nih.gov/31254957/

TUDCA suppresses NF-κB signaling and ameliorates colitis-associated tumorigenesis, suggesting that TUDCA could be a potential treatment for colitis-associated cancer.

J Gastroenterol Hepatol. 2019 Mar;34(3):544-551.
https://pubmed.ncbi.nlm.nih.gov/30378164/

Oral administration of TUDCA helped to restore the barrier function of the intestinal tract in colonic epithelial cells in the colitis.

Inflamm Bowel Dis. 2017 Dec;23(12):2121-2133.
https://pubmed.ncbi.nlm.nih.gov/29084077/

TUDCA protects bile acid homeostasis under inflammatory conditions and suppresses Crohn's disease ileitis.

Lab Invest. 2017 May;97(5):519-529.
https://pubmed.ncbi.nlm.nih.gov/28165466/

TUDCA reduce the severity of colitis and ameliorate colitis-associated fecal dysbiosis at the phylum level.

Appl Environ Microbiol. 2017 Mar 17;83(7):e02766-16.
https://pubmed.ncbi.nlm.nih.gov/28115375/

TUDCA, a compound capable of restoring Toll-interacting protein cellular function by effectively alleviate gut inflammation and can potently be used to help colitis.

Sci Rep. 2016 Oct 5;6:34672
https://pubmed.ncbi.nlm.nih.gov/27703259/

When TUDCA was given to colitis group it was shown that the colonic tissue levels of IL-1β, IFN-γ and TNF-α were significantly reduced and helped to greatly lessen the GI symptoms.

Int Immunopharmacol. 2016 Jul;36:271-276.
https://pubmed.ncbi.nlm.nih.gov/27179450/

Studies done on human cells in an environment that mimicked gut inflammation show that TUDCA helps to reduce the inflammation that contributes to these diseases. When researchers exposed cells to toxins that should trigger inflammation, TUDCA helped to reduce the amount of inflammatory molecules they produced.

Br J Pharmacol. 2018 Feb;175(3):469-484.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5773980/

Mineral Balance

TUDCA restored hepatic, renal, and serum Mg^{2+} levels and renal and serum Ca^{2+} levels to normal values in type 1 diabetes.

Biol Trace Elem Res. 2019 Jun;189(2):501-510.
https://pubmed.ncbi.nlm.nih.gov/30171596/

TUDCA significantly increased intracellular free zinc. Intracellular free Zn^{2+} may be involved in the protective action of ER stress inhibition on the mPTP opening.

Biol Trace Elem Res. 2016 Nov;174(1):189-197.
https://pubmed.ncbi.nlm.nih.gov/27106542/

STOMACH

TUDCA protected the gastric mucosa. TUDCA may play a role in treating gastritis associated with bile reflux.

Arch Physiol Biochem. 2002 Jul;110(3):197-202
https://pubmed.ncbi.nlm.nih.gov/12221520/

KIDNEY

TUDCA is a nephroprotectant after ischemia/reperfusion-induced acute kidney injury and stabilizes unfolded protein response pathways.

Basic Clin Pharmacol Toxicol. 2012;111(1):14–23
https://pubmed.ncbi.nlm.nih.gov/22212133/

TUDCA enhanced the mitochondrial function, improved the functional recovery, including kidney recovery, blood perfusion ratio, and vessel formation of mesenchymal stem cells in chronic kidney disease.

Redox Biol. 2019 Apr;22:101144.
https://europepmc.org/article/pmc/pmc6383184

TUDCA-treated chronic kidney disease (CKD) Mesenchymal stem cells (hMSCs) increased anti-oxidant enzyme activities through the upregulation of the cellular prion protein (PrPC) expression. Upregulated PrPC expression in cells protected against CKD-mediated ER stress and apoptosis. TUDCA-treated CKD-hMSCs suppressed ROS generation and ER stress in the hippocampus. These results indicate that TUDCA-treated CKD-hMSCs prevent the CKD-mediated cell death by inhibiting ER stress.

Int. J. Mol. Sci. 2019, 20(3), 613
https://www.mdpi.com/1422-0067/20/3/629

TUDCA acted as a preventive tool against chronic high salt induced renal damage.

Acta Physiol (Oxf). 2019 May;226(1):e13227.
https://pubmed.ncbi.nlm.nih.gov/30501003/

Treatment of TUDCA not only attenuated proteinuria and kidney histological changes, but also ameliorated podocyte and glomeruli injury in diabetic mice.

Sci Rep. 2017 Mar 23;7(1):323.
https://pubmed.ncbi.nlm.nih.gov/28336924/

TUDCA may help protect the kidneys and cells, however studies on animals suggest that TUDCA reduces kidney inflammation due to high sodium levels. It may also help protect kidney tissue from damage by preventing kidney cell death.

Acta Physiol (Oxf). 2019 May;226(1):e13227.
https://pubmed.ncbi.nlm.nih.gov/30501003/

THYROID

TUDCA helps increase your thyroid function. It helps upregulate energy in your cells. Brown adipose tissue is more metabolically active than white. TUDCA can double the amount of brown adipose tissue! TUDCA increases the amount of energy you use up and helps you use insulin better.

FEBS Lett. 2011 Feb 4; 585(3): 539–544.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3133948/

Hormones

TUDCA inhibits testosterone-induced RAGE expression and AGE accumulation in PCOS.

Endocrinology. 2020 Feb 1;161(2).
https://pubmed.ncbi.nlm.nih.gov/32053721/

TUDCA has been shown to decrease Endoplasmic Reticulum stress induced by high oxidative stress in granulosa cells in ovaries with endometrioma mediating apoptosis of these cells, leading to ovarian dysfunction in patients with endometriosis

Mol Hum Reprod. 2020 Jan 1;26(1):40-52.
https://pubmed.ncbi.nlm.nih.gov/31869409/

TUDCA alleviated Endoplasmic Reticulum stress in Adrenocortical Carcinoma (ACC) and induced autophagy, thereby inhibiting ACC cell apoptosis.

Oncol Lett. 2019 Dec; 18(6): 6475–6482.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6888259/

In PCOS taking TUDCA was found to rebalance proper testosterone levels by decreasing the inflammation in the Granulose cells of antral follicle

Endocrinology. 2019 Jan 1;160(1):119-132.
https://academic.oup.com/endo/article/160/1/119/5171869

TUDCA was shown to prevent aggregating of misfolded proteins, alleviate ER stress, and hinder UPR-mediated cell death in ovarian cortex during aging and senescence therefore represents the important cellular mechanism linked to tissue stability and homeostasis.

Cell Tissue Res. 2018 Dec;374(3):643-652
https://pubmed.ncbi.nlm.nih.gov/30066106/

TUDCA was shown to decreased interstitial fibrosis and collagen deposition in ovaries, accompanied by a reduction in TGF-β1 expression in granulosa cells in female with PCOS.

Sci Rep. 2017 Sep 7;7(1):10824.
https://pubmed.ncbi.nlm.nih.gov/28883502/

Pregnancy

TUDCA reduced DNA damage and ER stress in developing embryos.

Mol Reprod Dev. 2020 Jan;87(1):161-173
https://pubmed.ncbi.nlm.nih.gov/31793725/

TUDCA supplementation during IVC increases the developmental competency of bovine in vitro-derived embryos. Additionally, we found that the presence of TUDCA in IVC medium improves the cryo-tolerance of bovine embryos

Theriogenology. 2020 Jan 15;142:131-137
https://pubmed.ncbi.nlm.nih.gov/31593880/

TUDCA during parthenogenetic activation can reduce ER stress, and thereby reduce apoptosis and promote in vitro development of porcine parthenogenetic embryos.

Dev Reprod. 2018 Sep;22(3):235-244.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6182227/

TUDCA treatment during IVC significantly increased blastocyst formation rates in a concentration dependent manner. Finally, embryo transfer after TUDCA treatment revealed a significant improvement in the rates of offspring production. These results show that treatment with 1000 μM of TUDCA significantly can improve poor embryonic development of cumulus-free IVM-IVF embryos.

PLoS One. 2018 Aug 27;13(8):e0202962.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6110502/

TUDCA was shown to downregulate the inflammatory cytoxines IL-1β-induced production of IL-6, IL-8, IL-1β and MCP-1 if females with gestational diabetes.

Mol Cell Endocrinol. 2016 Apr 15;425:11-25
https://pubmed.ncbi.nlm.nih.gov/26902174/

Hearing

TUDCA reduced medication induced hearing loss

Neurosci Lett. 2020 Mar 23;722:134838.
https://pubmed.ncbi.nlm.nih.gov/32061715/

TUDCA attenuated gentamicin-induced hair cell death by inhibiting protein nitration activation and ER stress, providing new insights into the new potential therapies for sensorineural deafness.

Toxicol Lett. 2018 Sep 15;294:20-26.
https://pubmed.ncbi.nlm.nih.gov/29751043/

Systemic treatment with TUDCA significantly alleviated hearing loss and suppressed hair cell death.

Neuroscience. 2016 Mar 1; 316: 311–320.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4724520/

Lungs

TUDCA reduced the extent of fibrotic area in the lungs and the expression levels of these proteins in sleep apnea induced chronic intermittent hypoxia.

BMC Pulm Med. 2020; 20: 92.
https://bmcpulmmed.biomedcentral.com/articles/10.1186/s12890-020-1123-0

TUDCA helped to modulate ER stress that causes fibrosis of the lung's idiopathic pulmonary fibrosis

Am J Respir Crit Care Med. 2020 Jan 15;201(2):198-211.
https://pubmed.ncbi.nlm.nih.gov/31738079/

TUDCA significantly decreased inflammatory, immune, and cytokine responses; mucus metaplasia; and airway hyperresponsiveness in people with asthma.

JCI Insight. 2019 May 2;4(9):e98101.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6538331/

TUDCA significantly decreased allergic airway disease induced inflammation, markers of ER stress, airway hyperresponsiveness, and fibrosis.

Am J Physiol Lung Cell Mol Physiol. 2016 Jun 1; 310(11): L1243–L1259.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4935467/

TUDCA alleviated ER stress/UPR activation, as evidenced by decreased expression of CHOP and GRP94 and reversal of ER stress–induced AEC apoptosis in cGrp78f/f lungs in Bronchopulmonary dysplasia chronic lung disease.

Am J Respir Cell Mol Biol. 2016 Jul; 55(1): 135–149.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4942208/

Cancer

The present study showed that TUDCA reduced the invasion of metastatic breast cancer cell line under normal oxygen concentration and hypoxic conditions. Thus, TUDCA is a candidate anti-metastatic agent to target the ER stress pathway.

Oncol Lett. 2016 Sep;12(3):2227-2231
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4998512/

Eyes

TUDCA treatment in photoreceptor and RGC degeneration models not only inhibited apoptosis but also promoted cell survival and function.

Mol Vis. 2019 Oct 14;25:610-624
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6817734/

TUDCA therapy can ameliorate the photoreceptor degeneration and rectify the abnormities in visual signal transmission in retinopathies with progressive photoreceptor degeneration.

Biomed Pharmacother. 2019 Sep;117:109021.
https://pubmed.ncbi.nlm.nih.gov/31387173/

TUDCA treatment inhibited microglial activation and inflammation, resulting in the preservation of retinal structure.

J Cell Physiol. 2019 Aug;234(10):18801-18812
https://pubmed.ncbi.nlm.nih.gov/30924157/

In diabetic mice this study showed that with the use of UDCA it reduced the reduced retinal expression of tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), interleukin-6 (IL-6), intercellular cell adhesion molecule-1 (ICAM-1), inducible nitric oxide synthase (iNOS) and vascular endothelial growth factor (VEGF) which helps to prevent the breakdown of the of blood-retinal barrier that happen in diabetic retinopathy.

Eur J Pharmacol. 2018 Dec 5;840:20-27.
https://pubmed.ncbi.nlm.nih.gov/30268667/

In diabetic retinopathy TUDCA was shown to partly rescue the damage caused by constant endothelial activation induced by TNF-α further exacerbated by hyperglycemia results in activation of ER stress and chronic proinflammation in a feed forward loop ultimately resulting in endothelial junction protein alterations leading to visual deficits in the retina.

J Cell Biochem. 2018 Nov; 119(10): 8460–8471.
https://pubmed.ncbi.nlm.nih.gov/30054947/

Review study that looks at the neuroprotective strategies of TUDCA for retinal disease.

Prog Retin Eye Res. 2018 Jul; 65: 50–76.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6563519/

TUDCA has neuroprotective effects that could constitute a suitable therapy to prevent neurodegeneration and visual loss in retinitis pigmentosa.

PLoS One. 2017 May 25;12(5):e0177998.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5444790/

TUDCA interacts specifically with rhodopsin, which may contribute to its wide-ranging effects on retina physiology and as a potential therapeutic compound for retina degenerative diseases.

Exp Eye Res. 2018 May;170:51-57.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5983371/

TUDCA could ameliorate Diabetic Retinopathy by decreasing NO content and down-regulating the harmful protein expression that causes damage. Thus, the experiment results suggested that TUDCA might be a potential for the prevention and treatment of Diabetic Retinopathy

J Ethnopharmacol. 2016 Jun 5;185:162-70.
https://pubmed.ncbi.nlm.nih.gov/26988565/

TUDCA helped retinal ganglion cells by activating the antioxidant pathway, partially restoring the functionality of retinal neurocircuitry and significantly improving the visual response properties of retinal ganglion cells.

Invest Ophthalmol Vis Sci. 2015 Oct;56(11):6638-45.
https://pubmed.ncbi.nlm.nih.gov/26469749/

NMDA-mediated neurotoxicity model combined with a functional and morphological evaluation of the retina to demonstrate a neuroprotective effect of TUDCA on Retinal ganglion cell in vivo. They showed that systemic administration of TUDCA not only attenuated the functional changes associated with NMDA-induced retinal damage, but also delayed Retinal ganglion cell loss.

PLoS One. 2015; 10(9): e0137826.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4574963/

Studies with TUDCA show that it may act as a potent protector of eye tissue. One study on animals with a genetic eye disease that leads to blindness found that TUDCA may prevent its progression. From 3 days old till 30 days old, these animals received TUDCA. The TUDCA-treated animals adjusted to dark and light two times better than the animals who didn't receive TUDCA. Plus, the TUDCA-treated animals had five times more light receptors than the other animals at the 30-day mark.

Invest Ophthalmol Vis Sci. 2008 May;49(5):2148-55.
https://pubmed.ncbi.nlm.nih.gov/18436848/

Bones

TUDCA may contribute to the regeneration of bone tissue by improving angiogenesis.

Bone. 2020 Jan;130:115073
https://pubmed.ncbi.nlm.nih.gov/31626993/

TUDCA as a specific inhibitor of endoplasmic reticulum stress attenuated bone cancer pain and reduced the expression of certain cancer signaling proteins.

Mol Pain. 2019 Jan-Dec;15:1744806919876150
https://pubmed.ncbi.nlm.nih.gov/31452457/

TUDCA could protect NP cells from compression-induced death in intervertebral disc degeneration.

Evid Based Complement Alternat Med. 2018 Mar 12;2018:6719460
https://pubmed.ncbi.nlm.nih.gov/29721028/

It was found that after a spinal fusion surgery TUDCA use produced a higher amount of bone fusion formation, higher quality bone growth without adipose tissue and higher trabecular thickness. Results show TUDCA stimulated better results in spinal bone regeneration compared to rhBMP-2 which has been the main medication used in the past.

Tissue Eng Part A. 2018 Mar;24(5-6):407-417.
https://pubmed.ncbi.nlm.nih.gov/28826347/

TUDCA plays a critical role in enhancing osteogenesis by significantly decreased apoptosis and the inflammatory response in vivo and in vitro, which is important to enhance bone tissue regeneration.

Bone. 2016 Feb;83:73-81
https://pubmed.ncbi.nlm.nih.gov/26499839/

High expression of ER stress markers, reduced cell proliferation, increased apoptosis and decreased synthesis of type II collagen. These effects were alleviated by treatment with TUDCA. These findings provide new insights on the mechanisms underlying ER stress in cartilage degeneration and osteoarthritis development.

Int J Mol Med. 2015 Oct;36(4):1081-7.
https://pubmed.ncbi.nlm.nih.gov/26238983/

Blood Cells

When TUDCA was used in addition to cell cultures for artificial RBC production it was shown to have produced higher numbers of and have higher viability.

Biochem Biophys Res Commun. 2016 Sep 30;478(4):1682-7.
https://pubmed.ncbi.nlm.nih.gov/27596970/

Joints

The TUDCA-treated cells also had less cholesterol in their membranes, making them more "squishy." That effect may help joints to slide versus grind. Plus, TUDCA seemed to activate the genes related to making better cartilage. The TUDCA-treated cells had more protein and chondroitin sulfate — two components of healthy cartilage.

Biomater Sci 2019 Aug 1;7(8):3178-3189.
https://pubmed.ncbi.nlm.nih.gov/31143889/

Stem Cells

TUDCA treated mesenchymal stem cells (MSCs) transplantation augmented the blood perfusion ratio, vessel formation, and transplanted cell survival rate.

Sci Rep. 2016; 6: 39838.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5177936/

TUDCA significantly induced neural stem cells proliferation, self-renewal, and neural differentiation in the SVZ, without affecting dentate gyrus derived neural stem cells. More importantly, expression levels of mitochondrial biogenesis-related proteins and mitochondrial antioxidant responses were significantly increased by TUDCA.

Mol Neurobiol. 2018 May;55(5):3725-3738.
https://pubmed.ncbi.nlm.nih.gov/28534273/

In people with a high BMI greater than 30, bone marrow mesenchymal stem cells were found to displayed severely impaired osteogenic and diminished adipogenic differentiation, decreased proliferation rates, increased senescence, and elevated expression of ER stress-related genes. After treatment with TUDCA it was shown to greatly improve stem cell production.

J Cell Physiol. 2018 Nov;233(11):8429-8436.
https://pubmed.ncbi.nlm.nih.gov/29797574/

This study demonstrates that TUDCA protects MSCs against inflammation and apoptosis in ER stress caused by prion protein expression.

Int J Mol Sci. 2018 Jan 25;19(2):352.
https://pubmed.ncbi.nlm.nih.gov/29370069/

TUDCA significantly induced neural stem cells (NSCs) proliferation, self-renewal, and neural differentiation in the subventricular zone (SVZ), without affecting dentate gyrus -derived NSCs. More importantly, expression levels of mitochondrial biogenesis-related proteins and mitochondrial antioxidant responses were significantly increased by TUDCA in SVZ-derived NSCs.

Mol Neurobiol. 2018 May;55(5):3725-3738.
https://pubmed.ncbi.nlm.nih.gov/28534273/

IMMUNE SYSTEM

TUDCA was found to be a potent inhibitor for viral infections by hepatitis B virus and hepatitis D virus. TUDCA helps to block the virus from entering your cells. It stands in front of your cell receptors like a shield.

Journal of Virology 2014 Mar; 88(6): 3273–3284.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3957944/

TUDCA blocked Influenza A from entering your cells and stopped viral replication. TUDCA helped the immune cells to kill the virus faster.

The Journal of Biological Chemistry 2012 Feb 10; 287(7): 4679–4689
https://pubmed.ncbi.nlm.nih.gov/22194594/

TUDCA inhibits RSV (Respiratory Syncytial Virus) infection from spreading (replicating) in your body.

Am J Respir Crit Care Med 191;2015:A4050
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4351573/

HSV-1 infection showed that both replication and multiplication of the virus were inhibited by TUDCA

J Med Virol. 2020 May 1. doi: 10.1002/jmv.25963.
https://pubmed.ncbi.nlm.nih.gov/32356915/

TUDCA restored suppression of TNF-α and IL-1β expression and attenuated the enhanced viability of macrophages.

Cytokine. 2020 Mar;127:154959.
https://pubmed.ncbi.nlm.nih.gov/31877413/

TUDCA reduced the ability of Pseudorabies virus ability to reproduce by reducing endoplasmic reticulum stress and inhibiting the unfolded protein response typically triggered in viral infections.

Vet Microbiol. 2019 Dec;239:108485.
https://pubmed.ncbi.nlm.nih.gov/31767094/

TUDCA showed anti-inflammatory properties decreasing aberrant MUC1 accumulation in Sjogren Syndrome.

Rheumatology (Oxford). 2020 Apr 1;59(4):742-753.
https://pubmed.ncbi.nlm.nih.gov/31377809/

TUDCA abolished the proton conductivity of viral M2 by disrupting its oligomeric states, which induces inefficient viral infection. The identification and application of TUDCA as an inhibitor of M2 proton channel will expand our understanding of IAV biology and complement current anti-influenza A virus's arsenals.

Sci Bull (Beijing). 2019 Feb 15;64(3):180-188.
https://pubmed.ncbi.nlm.nih.gov/32288967/

In infections of herpes simplex virus 1, it was shown that TUDCA helped to decrease levels of IL-17, have improved lung functions, milder inflammation, and higher survival rate because of the ability to decrease endoplasmic reticulum stress.

J Cell Mol Med. 2019 Feb; 23(2): 908–919.
https://pubmed.ncbi.nlm.nih.gov/30378252/

Inhibition of the Unfolded Protein Response with TUDCA significantly diminished Brucella replication.

PLOS Pathogens December 5, 2013
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3855547/

When patients were given TUDCA, it alleviated ER stress induced lung injury in a septic host infected with Pseudomonas aeruginosa Consequently, in the lungs of the septic host infected with P. aeruginosa, symptoms of pneumonia improved and the infecting bacteria were cleared. Thus, for septic patients, determination of immune status may guide the selection of appropriate immunomodulation, and ER stress can be a novel therapeutic strategy restoring the immune response in patients with endotoxin tolerance.

J Leukoc Biol. 2018 Nov;104(5):1003-1012
https://pubmed.ncbi.nlm.nih.gov/29924419/

TUDCA partially reversed those HSV-1-mediated adverse effects in mice with bleomycin-induced lung injury.

Front Immunol. 2017 Dec 11;8:1756.
https://pubmed.ncbi.nlm.nih.gov/29312299/

TUDCA decreased mortality in aged hosts that was associated with increased NLRP3 inflammasome activation, improved pathogen clearance, and decreased pneumonitis during infection.

Am J Physiol Lung Cell Mol Physiol. 2018 Mar 1;314(3):L372-L387
https://pubmed.ncbi.nlm.nih.gov/29097427/

TUDCA helped to maintain lymphocyte homeostasis by significantly reducing lymphocyte apoptosis and this correlated with four-fold improvement in survival for treating sepsis-induced lymphopenia in humans.

Sci Rep. 2016 Oct 3;6:34702.
https://pubmed.ncbi.nlm.nih.gov/27694827/

TUDCA helped to decrease ER stress in viral-induced myocarditis but it also suggests that manipulating ER stress might be a novel therapeutic strategy against virus-induced inflammatory diseases.

Can J Cardiol. 2015 Aug;31(8):1032-40.
https://pubmed.ncbi.nlm.nih.gov/26111668/

In a lab, researchers examined what would happen if they pretreated human cells with TUDCA and then exposed them to the influenza A virus. They found that TUDCA prevented the cells from reaching excess ER stress due to the virus. TUDCA also seemed to prevent the influenza A virus from replicating.

J Biol Chem. 2012 Feb 10;287(7):4679-89.
https://pubmed.ncbi.nlm.nih.gov/22194594/

Another lab study with human liver cells reviewed the effect of TUDCA on the hepatitis B and D viruses. These viruses infect your cells by attaching themselves to receptors on the outside of the cellular membrane. Then they bust their way in, almost like picking a lock. Pretreating the cells with TUDCA essentially "covered the lock" so the hepatitis virus couldn't gain access into the cell.

J Virol. 2014 Mar;88(6):3273-84.
https://pubmed.ncbi.nlm.nih.gov/24390325/

Mitochondria

TUDCA acts as a mitochondrial stabilizer.

J Lipid Res. 2009 Sep; 50(9): 1721–1734.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2724780/

TUDCA suppressed cytotoxicity by reducing protein misfolding and increasing functional mitochondrial

Cell Reports (2018).
https://www.cell.com/cell-reports/fulltext/S2211-1247(17)31875-2

We also found that TUDCA partially restored the production of ATP that was inhibited by particulate matter, and its recovery ability was significantly higher than that of ginsenoside Rb1, indicating that the process of ER stress leading to cell damage may also occur via the mitochondrial pathway.

Antioxidants 2019, 8(9), 383
https://www.mdpi.com/2076-3921/8/9/383

TUDCA was shown to act as a modulator of mitochondrial activity and turnover, with implications in neurodegenerative diseases like Parkinson's disease.

Biochim Biophys Acta. 2017 Sep ;1863(9):2171-2181.
https://pubmed.ncbi.nlm.nih.gov/28583715/

Tests on animals show that TUDCA helps relieve stressed mitochondria through producing more antioxidants to mitigate ROS. Rather than triggering cell death, they did the exact opposite. TUDCA sparked them to make more mitochondria.

Mol Neurobiol. 2018 May;55(5):3725-3738.
https://pubmed.ncbi.nlm.nih.gov/28534273/

Your cellular energy factories mitochondria want to work as efficient energy producers. But toxins, heavy metals, medications, viruses, etc. can sometimes stand in the way. These factors can change the mitochondrial membrane, making it more difficult for your mitochondria to release the energy molecules, or ATP. And your cells can't use the energy without access.

Cell Cycle. 2014;13(22):3576-89
https://pubmed.ncbi.nlm.nih.gov/25483094/

In the presence of TUDCA, both types of cells had a significantly higher amount of ATP release than without. This means energy will no longer stay trapped in the mitochondria where your cells can't access it. The ATP will instead exist in the cytoplasm, or watery part of your cells.

Hepatology. 2018 Jul;68(1):187-199.
https://pubmed.ncbi.nlm.nih.gov/29360145/

TUDCA and NAC

TUCDA and NAC were found to decrease ROS and ER stress, increased production of VEGF-A may induce compensatory angiogenesis after acute myocardial ischemia through initiating ROS-ER stress-autophagy axis in the vascular endothelial cells.

J Cell Physiol. 2019 Aug;234(10):17690-17703
https://pubmed.ncbi.nlm.nih.gov/30793306/

N-acetyl-L-cysteine, and the endoplasmic reticulum stress inhibitor TUDCA, reversed the inhibitory effects of advanced glycation end products that effected testosterone production in diabetic mice.

Int J Mol Med. 2016 Jun 16.
https://www.spandidos-publications.com/10.3892/ijmm.2016.2645

Taken together NAC and TUDCA were shown to reduce cytoxicity effects of bavachin in the liver by there combined properties to reduce ROS, attenuate apoptosis, reduce ER stress,

Biol Pharm Bull. 2018 Feb 1;41(2):198-207.
https://pubmed.ncbi.nlm.nih.gov/29187671/

TUDCA and NAC taken together showed inhibition of AGE formation rescues against experimental diabetes-induced cardiac remodeling and contractile dysfunction possible through regulation of autophagy and ER stress.

Toxicol Lett. 2018 Mar 1;284:10-20.
https://pubmed.ncbi.nlm.nih.gov/29174818/

Combination strategy of NAC and TUDCA surpasses the standard of care in acetaminophen-induced liver injury and might represent an attractive therapeutic opportunity for acetaminophen-intoxicated patients.

Liver Int. 2017 May;37(5):748-756.
https://pubmed.ncbi.nlm.nih.gov/27706903/

It was found that he combination of NAC and TUDCA were able to inhibit the advanced glycation end products induced decrease of testonsterone through decreasing oxidative stress and ER stress in Leydig Cells.

Int J Mol Med. 2016 Aug;38(2):659-65.
https://pubmed.ncbi.nlm.nih.gov/27315604/

The combination of TUDCA and NAC may improve their ability to squelch free radicals and lower inflammation, according to animal research.

Liver Int. 2017 May;37(5):748-756.
https://pubmed.ncbi.nlm.nih.gov/27706903/

TUDCA and Melatonin

TUDCA and melatonin treatment could restore the expression levels of ER-stress through inhibition of meiotic maturation of oocyte

Dev Reprod. 2017 Dec;21(4):407-415.
https://pubmed.ncbi.nlm.nih.gov/29354786/