

Note: Treatment colors indicate the disease mechanism that the treatment affects:

Protein Aggregation

Inflammation

Abnormalities in Energy Metabolism

Free Radical Damage

Glutamate Toxicity

Changes in Gene Transcription

Treatment	Type (Drug/ Supplement/ Etc.)	Mechanism of Action	Source	Most Recent Studies
<u><b>Carnitine</b></u>	Supplement meant to slow progression	Energy Buffer: <ul style="list-style-type: none"> <li>Facilitates entry of <b>fatty acids</b> into the <b>mitochondria</b> to increase energy production</li> </ul>	<ul style="list-style-type: none"> <li>Produced by the body</li> <li>Found in red meat</li> </ul>	<b>A) Alzheimer's disease clinical study:</b> <ul style="list-style-type: none"> <li>Published by Thal, et al. (2000)</li> <li>Failed to show significant difference in a 1-year, multicenter, double-blind, placebo-controlled, randomized trial of 229 Alzheimer's Disease patients of ages 45 - 65 treated with acetyl-L-carnitine (ALC) or placebo.</li> </ul>
<b>Possible Side Effects:</b> High doses may cause diarrhea.				
<u><b>Coenzyme Q10</b></u>	Supplement meant to slow progression	Antioxidant and Energy Buffer: <ul style="list-style-type: none"> <li>Acts as an electron and proton carrier during <b>cellular respiration</b> to increase energy production</li> </ul>	<ul style="list-style-type: none"> <li>Produced by all mammalian tissues</li> <li>Can be chemically synthesized</li> </ul>	<b>A) Animal study:</b> <ul style="list-style-type: none"> <li>Ferrante, et al. (2002)</li> <li>Survival increased and development of motor deficits, brain atrophy, neuronal inclusions, and motor deficits decreased in mouse model of HD.</li> <li>Greater benefits when mice were treated with both coenzyme Q10 and remacemide.</li> </ul> <b>B) Clinical study:</b> <ul style="list-style-type: none"> <li>Huntington Study Group (2001)</li> <li>Study of 347 patients showed that Coenzyme Q10 and remacemide failed to significantly slow the progression of HD.</li> <li>Disease seemed to progress more slowly in patients treated with Coenzyme Q10, but results are inconclusive.</li> <li>See also <i>Remacemide</i></li> </ul>
<b>Possible Side Effects:</b> Some users experienced mild insomnia, rashes, nausea, abdominal pain, and elevated liver enzymes.				
<u><b>Creatine</b></u>	Supplement meant to improve symptoms and slow progression	Energy Buffer and Membrane Stabilizer: <ul style="list-style-type: none"> <li>Converted in muscle cells to phosphocreatine (PCr) which acts as a reservoir for the energy rich molecule <b>ATP</b></li> <li>Helps prevent inactivation of the membrane protein mtCk (mitochondrial creatine kinase) to stabilize cell <b>membranes</b></li> </ul>	<ul style="list-style-type: none"> <li>Found in foods such as meat and fish</li> <li>Can be chemically synthesized</li> </ul>	<b>A) Clinical study:</b> <ul style="list-style-type: none"> <li>Verbessem, et al. (2003)</li> <li>One year study of 41 patients showed that 5g/day creatine supplementation did not improve cognitive or motor functioning over the placebo control.</li> </ul> <b>B) Animal study:</b> <ul style="list-style-type: none"> <li>Alpaslan et al. (2003)</li> <li>Dietary supplementation with creatine improved survival, motor performance, maintained brain and body weight, and reduced protein aggregates and overall nerve cell death in a mouse model of HD.</li> </ul>
<b>Possible Side Effects:</b> Creatine use has been linked to dehydration and decreased blood volume. Fluid intake must be increased when taking creatine.				

<p><u>Cystamine</u></p>	<p>Drug meant to improve symptoms and slow progression</p>	<p>Decrease Huntingtin Aggregation and Prevent Apoptosis</p> <ul style="list-style-type: none"> <li>Inhibits the activity of <b>transglutaminase (TGase)</b>, an <b>enzyme</b> that plays a role in the formation of huntingtin aggregates</li> <li>May prevent nerve cell <b>apoptosis</b> by inhibiting <b>caspases</b></li> <li>May have antioxidant properties</li> </ul>	<ul style="list-style-type: none"> <li>Chemically synthesized</li> <li>So far only available as the prescription drug Cystagon (cysteamine bitartrate), which is converted to cystamine in the body</li> <li>Currently used to treat cystinosis, a genetic disorder in which the <b>amino acid</b> cystine builds up in the cell's <b>lysosomes</b></li> </ul>	<p><b>A) Lab study:</b></p> <ul style="list-style-type: none"> <li>Lesort, et al. (2003)</li> <li>Found that cystamine inhibits the activity of a certain <b>caspase</b>, a molecule involved in early cell death. Cystamine's interaction with this molecule is not connected to its interaction with TGase.</li> </ul> <p><b>B) Animal study:</b></p> <ul style="list-style-type: none"> <li>Karpuj, et al. (2002)</li> <li>HD mice treated with cystamine showed improved motor symptoms, prolonged survival, and decreased weight loss.</li> <li>Showed that cystamine decreased TGase activity but found that this was not linked to a decrease in <b>neuronal inclusions (NIs)</b>.</li> <li>Instead, cystamine increased the expression of certain <b>genes</b> that have protective effects in the cell.</li> </ul>
<p><b>Possible Side Effects:</b> Most common side effects are nausea, diarrhea, and upset stomach. Should be taken along with one that reduces acid secretion in the stomach.</p>				
<p><u>Dichloroacetate</u></p>	<p>Supplement/ small molecule meant to improve symptoms and slow progression</p>	<p>Increase Energy Metabolism:</p> <ul style="list-style-type: none"> <li>Stimulates the enzyme complex pyruvate dehydrogenase in <b>mitochondria</b> to increase energy metabolism</li> <li>Thought to slow nerve cell death by lowering cellular concentrations of <b>lactic acid</b></li> </ul>	<ul style="list-style-type: none"> <li>A derivative of acetic acid</li> <li>Chemically synthesized</li> </ul>	<p><b>A) Animal study:</b></p> <ul style="list-style-type: none"> <li>Andreassen, et al. (2001)</li> <li>Study in <b>transgenic</b> mice (mice whose genes have been artificially altered to mimic HD symptoms) found that dichloroacetate treatment significantly improved motor function and survival, and decreased weight loss compared to control transgenic mice.</li> </ul>
<p><b>Possible Side Effects:</b> May be carcinogenic.</p>				
<p><u>Geldanamycin (GA)</u></p>	<p>Antibiotic drug meant to slow progression</p>	<p>Huntingtin Aggregation Inhibition:</p> <ul style="list-style-type: none"> <li>Binds to heat shock protein 90 (<b>Hsp 90</b>) and prevents it from binding to heat shock factor-1 (<b>HSF-1</b>)</li> <li>HSF-1 activates production of <b>Hsp 40 and Hsp 70</b>, proteins that bind to and prevent huntingtin aggregation</li> </ul>	<ul style="list-style-type: none"> <li>Produced by the fungus <i>Streptomyces hygroscopicus</i></li> </ul>	<p><b>A) Lab study:</b></p> <ul style="list-style-type: none"> <li>Hay, et al. (2004)</li> <li>Found that geldanamycin induced expression of Hsp 40 and Hsp 70 and delayed protein aggregation by only a couple of weeks in nerve cells from a mouse model of HD.</li> </ul> <p><b>B) Lab study:</b></p> <ul style="list-style-type: none"> <li>Sittler, et al. (2001)</li> <li>Showed that GA could reduce huntingtin aggregation in mouse HD mouse model nerve cells by up to 80% and increase amounts of Hsp 40, Hsp 70, and Hsp 90.</li> </ul>
<p><b>Possible Side Effects:</b> GA is toxic to many other types of cells other than <b>nerve cells</b>.</p>				

<p><u>Ginkgo Biloba</u></p>	<p>Supplement meant to improve symptoms and slow progression</p>	<p>Antioxidant and Anti-Inflammatory:  <ul style="list-style-type: none"> <li>Reduces <b>free radical</b> formation</li> </ul> </p>	<ul style="list-style-type: none"> <li>Extract prepared from leaves of Ginkgo biloba tree</li> <li>Available in health and nutrition stores</li> </ul>	<p>Effect on HD specifically not well studied.  <b>A) Alzheimer's disease animal study:</b>  <ul style="list-style-type: none"> <li>Stackman, et al. (2003)</li> <li>Found that treatment with ginkgo biloba blocked deterioration of spatial learning and memory in a mouse model of AD.</li> </ul> <b>B) Lab and animal study:</b>  <ul style="list-style-type: none"> <li>Smith, J.V., and Luo, Y. (2003)</li> <li>Found that ginkgo biloba reduced the levels of free radicals in both AD-associated cell cultures and in a <i>Caenorhabditis elegans</i> (worm) model of AD.</li> </ul> </p>	
<p><b>Possible Side Effects:</b>  May cause upset stomach, rash, or headache. Should not be taken by people who have blood clotting disorders or are regularly taking aspirin, or by anyone taking monoamine oxidase inhibitors (MAOIs).</p>		<p>Drug meant to improve symptoms</p>	<p>Anti-Inflammatory:  <ul style="list-style-type: none"> <li><b>Steroid hormones</b> that can inhibit the production of proteins involved in the inflammatory response</li> </ul> </p>	<ul style="list-style-type: none"> <li>Hormones naturally produced in the body</li> <li>Drugs are chemically synthesized and mimic natural hormones</li> <li>Examples: prednisone, dexamethasone, hydrocortisone</li> </ul>	<p>Effect on HD specifically not well studied.  <b>A) Alzheimer's disease clinical study:</b>  <ul style="list-style-type: none"> <li>Dziedzic, et al. (2003)</li> <li>Study of 18 AD patients and 12 non-AD subjects found that treatment with dexamethasone inhibited the production of <b>cytokines</b> that cause inflammation and did so more effectively in AD patients than in controls.</li> </ul> </p>
<p><u>Histone deacetylase (HDAC) inhibitors</u></p>		<p>Drug meant to improve symptoms</p>	<p>Gene transcription regulation:  <ul style="list-style-type: none"> <li>Prevent <b>histone deacetylases</b> from condensing DNA in order to extend <b>transcription</b> of genes</li> </ul> </p>	<ul style="list-style-type: none"> <li>Can be chemically synthesized</li> <li>Examples: sodium butyrate, suberoylanilide hydroxamic acid (SAHA)</li> </ul>	<p><b>A) Animal study:</b>  <ul style="list-style-type: none"> <li>Hockly, et al. (2003)</li> <li>Found that treatment with SAHA improved motor impairment in a mouse model of HD but that higher doses of SAHA had toxic effects.</li> </ul> </p>
<p><u>Lamotrigine</u></p>		<p>Drug meant to improve symptoms</p>	<p>Anti-glutamate:  <ul style="list-style-type: none"> <li>Is used as an anti-epileptic drug</li> <li>Prevents release of <b>glutamate</b> from overactive nerve cells</li> <li>Counteracts the increased sensitivity that nerve cells in HD have to glutamate</li> </ul> </p>	<ul style="list-style-type: none"> <li>Chemically synthesized</li> </ul>	<p><b>A) Clinical study:</b>  <ul style="list-style-type: none"> <li>Higgins, et al. (2002)</li> <li>Open label study of 20 HD patients tested increasing doses of lamotrigine for safety and efficacy over seven weeks.</li> <li>Patients treated with lamotrigine showed improvements on cognitive tests but not in motor, functional, or behavioral aspects.</li> <li>Study could not reproduce results of 1999 study (Kremer, et al.) that showed improvements in <b>chorea</b> with treatment.</li> </ul> </p>
<p><b>Possible Side Effects:</b>  Most severe side effects reported were nausea, skin rashes, insomnia, or depression.</p>					

<u>Lipoic acid</u>	Supplement meant to improve symptoms and slow progression	Antioxidant: • Acts as a <b>coenzyme</b> in the <b>mitochondria</b> to aid <b>enzymes</b> in breaking down sugar • Scavenges <b>free radicals</b> and prevents them from causing damage to cells	• Produced by the body • Found in certain vegetables and in red meat	<b>A) Animal study:</b> • Andreassen, et al. (2001) • Tested the effects of lipoic acid treatment on two different mouse models of HD. • Showed that one group had decreased weight loss and that both groups survived at least a week longer than untreated mice.
	<b>Possible Side Effects:</b> High doses may cause upset stomach and nausea, as well as low blood sugar levels and fatigue or insomnia.			
<u>Minocycline</u>	Drug meant to slow progression	Antioxidant and Anti-Inflammatory: • Able to cross the <b>blood-brain barrier</b> and inhibit inflammatory response • Decreases free radical formation • Inhibits production of <b>caspases</b> , enzymes involved in HD progression	• Antibiotic in the tetracycline family • Commonly used to treat acne and some forms of arthritis	<b>A) Clinical study:</b> • Bonelli, et al. (2003) • Study of 14 HD patients showed that minocycline was well-tolerated, produced only minor side effects, and after 6 months improved motor function and performance on the Mini-Mental State Examination when compared to untreated HD patients.
	<b>Possible Side Effects:</b> May cause nausea, vomiting, diarrhea, dizziness, and increased sensitivity to sunlight. Should not be used along with penicillin antibiotics, blood thinning medication, or mineral supplements.			
<u>Nicotinamide</u>	Supplement meant to improve symptoms	Increase Energy Metabolism: • Precursor to <b>NAD</b> , a molecule used in the <b>electron transport chain</b> step of <b>cellular respiration</b> • Inhibits an enzyme that depletes <b>ATP</b> and other molecules involved in energy production • May protect against toxicity caused by <b>malonate</b>	• A form of vitamin B3 and related to niacin • Synthesized by the body • Found in meats, peanuts, and sunflower seeds	<b>A) Animal study:</b> • Beal, et al. (1994) • Showed that a combination treatment of nicotinamide and coenzyme Q10 significantly reduced energy deficits in brains of male rats treated with the toxin malonate.
	<b>Possible Side Effects:</b> High doses may cause liver damage, ulcers, and rashes.			
<b><u>Non-steroidal Anti-inflammatory Drugs (NSAIDs):</u></b>				
<u>Aspirin</u>	Drug meant to slow progression	Non-steroidal Anti-inflammatory Drugs (NSAIDs): • Inhibit enzymes called <b>cyclooxygenases</b> to suppress the inflammatory response • May slow nerve cell death	• Chemically synthesized	<b>A) Lab study:</b> • Casper, et al. (2000) • Found that treatment with any of three NSAIDs (aspirin, acetaminophen, or ibuprofen) decreased cell death in a mixture of nerve cells and <b>glial cells</b> that were exposed to glutamate, which has been shown to cause toxicity and nerve cell death in HD.
	<b>Possible Side Effects:</b> Most common side effect is stomach irritation and more rarely, the development of ulcers. Should not be taken with other blood thinning medication.			

<p><b>Acetaminophen</b></p>	<p>Drug meant to slow progression</p>	<ul style="list-style-type: none"> <li>• See above (Aspirin)</li> </ul>	<ul style="list-style-type: none"> <li>• Chemically synthesized</li> <li>• Examples: Tylenol, Tempra</li> </ul>	<ul style="list-style-type: none"> <li>• See above (Aspirin)</li> </ul>
<p><b>Possible Side Effects:</b> May cause lightheadedness. High doses may cause liver damage.</p>				
<p><b>Ibuprofen</b></p>	<p>Drug meant to slow progression</p>	<ul style="list-style-type: none"> <li>• See above (Aspirin)</li> </ul>	<ul style="list-style-type: none"> <li>• Chemically synthesized</li> <li>• Examples: Advil, Motrin, Aleve</li> </ul>	<p>Effect on HD specifically not well studied.</p> <p><b>A) Alzheimer's disease animal study:</b></p> <ul style="list-style-type: none"> <li>• Lim, et al. (2001)</li> <li>• Found that ibuprofen treatment in a mouse model of AD reduced expression of <b>cytokines</b> that are usually elevated in AD patients and reduced <b>microglial</b> activation, which could delay some AD symptoms.</li> <li>• Also see above (Aspirin)</li> </ul>
<p><b>Possible Side Effects:</b> Most common side effect is stomach irritation and more rarely, the development of ulcers. Should not be taken with other blood thinning medication.</p>				
<p><b>Omega-3 Fatty Acids:</b></p>				
<p><b>EPA (LAX-101)</b></p>	<p>Supplement meant to slow progression</p>	<p>Omega-3 Fatty Acids:</p> <ul style="list-style-type: none"> <li>• Have anti-inflammatory properties</li> <li>• May improve energy metabolism</li> <li>• May prevent nerve cell damage by decreasing neuron excitability</li> </ul>	<ul style="list-style-type: none"> <li>• Found in fish and fish oil</li> </ul>	<p><b>A) Clinical study:</b></p> <ul style="list-style-type: none"> <li>• Puri, et al. (2002)</li> <li>• Six month study of 7 advanced HD patients showed that treatment with ethyl-EPA may improve motor function and psychological state as well as prevent brain atrophy.</li> </ul>
<p><b>Possible Side Effects:</b> May cause upset stomach and diarrhea.</p>				
<p><b>Docosahexa-enoic acid (DHA)</b></p>	<p>Supplement meant to improve symptoms</p>	<p>Omega-3 Fatty Acids:</p> <ul style="list-style-type: none"> <li>• Have anti-inflammatory properties</li> <li>• May reduce risk of heart attacks</li> </ul>	<ul style="list-style-type: none"> <li>• Found in fish and fish oil</li> </ul>	<p>Effect on HD specifically not well studied.</p> <p><b>A) Alzheimer's disease animal study:</b></p> <ul style="list-style-type: none"> <li>• Hashimoto, et al. (2002)</li> <li>• Found that treatment with DHA delayed a decline in learning ability and showed antioxidant properties in a rat model of AD. It also appeared to decrease nerve cell death.</li> </ul>
<p><b>Possible Side Effects:</b> May act as a blood thinner.</p>				
<p><b>Rapamycin</b></p>	<p>Drug meant to slow progression</p>	<p>Decrease Huntingtin Aggregation:</p> <ul style="list-style-type: none"> <li>• Promotes <b>autophagy</b> by inhibiting the protein <b>mTOR</b></li> <li>• Helps cells break down <b>huntingtin</b> and prevent aggregations</li> </ul>	<ul style="list-style-type: none"> <li>• Antibiotic produced by <i>Streptomyces hygroscopicus</i></li> <li>• Can be chemically synthesized in a better acting form called <b>CCI-779</b></li> </ul>	<p><b>A) Animal study:</b></p> <ul style="list-style-type: none"> <li>• Ravikumar, et al. (2004)</li> <li>• Found that treatment with rapamycin decreased nerve cell death in a fly model of HD.</li> <li>• Found that treatment with rapamycin of HD mice reduced protein aggregates, decreased nerve cell death and improved motor symptoms, but only when treatment began before the appearance of HD symptoms.</li> </ul>
<p><b>Possible Side Effects:</b> When tested in cancer patients, the most common side effect was acne-like lesions and rashes.</p>				

<p><u>Remacemide</u></p>	<p>Drug meant to improve symptoms</p>	<p>Anti-glutamate:  <ul style="list-style-type: none"> <li>Typically used as an antiepileptic drug</li> <li>Blocks the <b>NMDA glutamate receptor</b> in nerve cells to reduce toxicity and cell death</li> </ul> </p>	<ul style="list-style-type: none"> <li>Chemically synthesized by Astra Zeneca</li> </ul>	<p><b>A) Animal study:</b></p> <ul style="list-style-type: none"> <li>Ferrante, et al. (2002)</li> <li>Survival increased and development of motor deficits, brain atrophy, neuronal inclusions, and motor deficits decreased in mouse model of HD.</li> <li>Greater benefits when mice were treated with both coenzyme Q10 and remacemide.</li> </ul> <p><b>B) Clinical study:</b></p> <ul style="list-style-type: none"> <li>Huntington Study Group (2001)</li> <li>Study of 347 patients showed that Coenzyme Q10 and remacemide failed to significantly slow the progression of HD.</li> <li>Disease seemed to progress more slowly in patients treated with Coenzyme Q10, but results are inconclusive.</li> <li>See also <i>Coenzyme Q10</i></li> </ul>
<p><b>Possible Side Effects:</b> Possible dizziness, nausea, or vomiting.</p>				
<p><u>Riboflavin</u></p>	<p>Supplement meant to improve symptoms and slow progression</p>	<p>Increase Energy Metabolism:  <ul style="list-style-type: none"> <li>Component of two <b>coenzymes</b> necessary for the production of <b>ATP</b></li> <li>May have antioxidant properties</li> </ul> </p>	<ul style="list-style-type: none"> <li>Vitamin B2</li> <li>Found in certain meats and plants</li> </ul>	<p>Effect on HD specifically not well studied.</p> <p><b>A) Complex I deficiency clinical study</b></p> <ul style="list-style-type: none"> <li>Ogle, et al. (1997)</li> <li>Study of one patient with a <b>complex I</b> deficiency, which causes abnormalities in energy production, showed that riboflavin supplementation caused an improvement in symptoms in the patient.</li> </ul>
<p><b>Possible Side Effects:</b> No known serious side effects.</p>				
<p><u>Riluzole</u></p>	<p>Drug meant to improve symptoms and slow progression</p>	<p>Anti-glutamate and Energy buffer:  <ul style="list-style-type: none"> <li>Interferes with proteins in messenger cascade set off by the <b>neurotransmitter</b> glutamate</li> <li>Helps prevent glutamate toxicity, delaying nerve cell death</li> <li>May have positive effects on cells with defective metabolism</li> </ul> </p>	<ul style="list-style-type: none"> <li>Chemically synthesized</li> <li>Example: Rilutek, by Aventis</li> </ul>	<p><b>A) Clinical study:</b></p> <ul style="list-style-type: none"> <li>Huntington Study Group (2003)</li> <li>8 week study of 63 HD patients showed that riluzole may reduce <b>chorea</b>, with higher doses causing more improvement.</li> <li>While a 200 mg/day dose improved chorea and motor function, it also caused fatigue and elevated a certain liver enzyme, which may result in abdominal pain and more serious consequences. The 100 mg/day dose did not cause significant improvement.</li> </ul>
<p><b>Possible Side Effects:</b> Possible diarrhea, nausea, or vomiting.</p>				
<p><u>Selenium</u></p>	<p>Supplement meant to slow progression</p>	<p>Antioxidant:  <ul style="list-style-type: none"> <li>Protects cells from <b>oxidative damage</b> caused by <b>free radicals</b> and <b>peroxides</b></li> </ul> </p>	<ul style="list-style-type: none"> <li>Found in certain foods such as whole grains, yeast, brazil nuts, and seafood</li> </ul>	<p><b>A) Animal study:</b></p> <ul style="list-style-type: none"> <li>Santamaria, et al. (2003)</li> <li>Used rats treated with <b>quinolinic acid (QUIN)</b>, which produces oxidative damage to cells similar to that caused by HD.</li> <li>Showed that QUIN rats treated with selenium sustained significantly less oxidative damage and had 70% less nerve cell degeneration than did untreated QUIN rats.</li> </ul>
<p><b>Possible Side Effects:</b> Excess intake may cause hair, nail, and tooth loss. May also cause nausea, fatigue, and skin inflammation.</p>				

<p><b><u>Trehalose</u></b></p>	<p>Drug meant to improve symptoms and slow progression</p>	<p>Huntingtin Aggregation Inhibition:  <ul style="list-style-type: none"> <li>• May bind to and stabilize <b>huntingtin</b> protein to prevent aggregation</li> </ul> </p>	<ul style="list-style-type: none"> <li>• Disaccharide</li> <li>• Produced by the body</li> <li>• Found in food such as lobster, honey, and yeast</li> </ul>	<p><b>A) Animal study:</b></p> <ul style="list-style-type: none"> <li>• Tanaka, et al. (2004)</li> <li>• Found that treatment with trehalose decreased the formation of protein aggregates, improved motor function, decreased brain atrophy and increased lifespan in a mouse model of HD.</li> </ul>
<p><b>Possible Side Effects:</b>          May cause bloating and diahrrea.</p>				
<p><b><u>Tauroursode-oxycholic acid (TUDCA)</u></b></p>	<p>Drug meant to improve symptoms and slow progression</p>	<p>Prevents <b>Apoptosis</b> and Huntingtin Aggregation:  <ul style="list-style-type: none"> <li>• Protects the membrane of the <b>mitochondria</b> to prevent the cascade of events that leads to cell death</li> </ul> </p>	<ul style="list-style-type: none"> <li>• A digestive fluid found in human bile</li> <li>• Found in large quantities in the bile of black bears</li> </ul>	<p><b>A) Animal study:</b></p> <ul style="list-style-type: none"> <li>• Keene, et al. (2002)</li> <li>• Showed that nerve cells of HD mice treated with TUDCA formed fewer <b>neuronal inclusions (NIs)</b>.</li> <li>• HD mice treated with TUDCA showed less deterioration of motor abilities than untreated mice.</li> </ul>
<p><b>Possible Side Effects:</b>          No known serious side effects.</p>				