

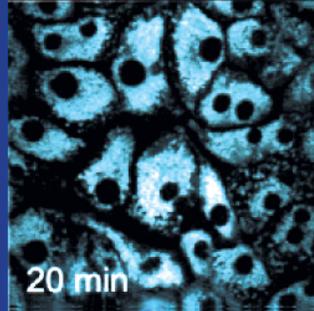
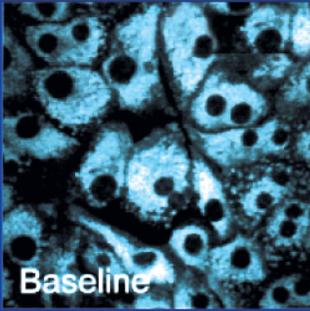
Microhydrin[®] Increases NADH Production and Mitochondrial Membrane Potential is enhanced in Intact Cells

Recent studies showed that Microhydrin[®] would reduce or add hydrogen to NAD⁺ converting it to NADH in vitro. It has also shown reducing activity by reducing cytochrome C (an electron transport molecule in the mitochondria) and other free radicals.

Intact biological liver cells were also shown to convert NAD⁺ into NADH within living cells when Microhydrin[®] was added to the live cell suspension. This study showed that Microhydrin[®] is capable of reducing or transferring H⁻ or electrons across the cell membrane and into the cell making it available for the NADH pool in the cell. In the vehicle control group, with no Microhydrin[®], NADH fluorescence decreased by 30% over 20 minutes. In the Microhydrin[®] treated cells, NADH fluorescence increased by 20%. These preliminary experiments suggest that Microhydrin[®] promotes electron transfer to NAD in intact living hepatocytes. Moreover, Microhydrin[®] prevented the spontaneous oxidation (or bleaching) of NADH that usually occurs during incubation in a simple balanced salt solution (200 ug/ml). Microhydrin[®] caused a continuous recharging of NADH within the mitochondria during the testing period rather than being utilized and depleted as in the control group. Micrographs, although not as sensitive to the naked eye as the instrumentation used to measure fluorescence changes, show individual dots of autofluorescence increasing after Microhydrin[®] treatment. These are the mitochondria with a greater concentration of NADH. By contrast, autofluorescence decreased in hepatocytes treated with vehicle control. NADH, but not NAD, is fluorescent, and oxidation of NADH back to NAD causes loss of fluorescence. Increased NADH production within the mitochondria is directly linked to the increased production of ATP. ATP is often referred to as the bioenergetic currency of the cell.

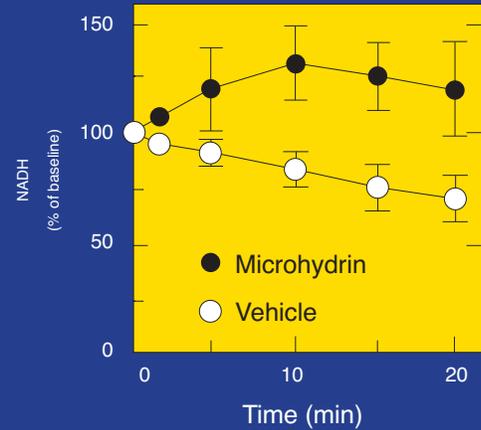
This research study also evaluated the effect of Microhydrin[®] on mitochondrial membrane potential, as measured with tetramethylrhodamine methyl ester (TMRM). In these experiments an increase of the mitochondrial fluorescence of TMRM represents an increase of mitochondrial depolarization (more negative membrane potential) further indication that NADH supply is being enhanced. In the control vehicle group fluorescence decreased by about 6% over 20 minutes. In the Microhydrin[®] group fluorescence increased about 25%. Actual photomicrographs show white bead-like membranes within the cytoplasm brighter after 20 min. of exposure to Microhydrin[®].

Microhydrin Increases Mitochondrial NADH in Intact Liver Cells Autofluorescence Confocal Microscopy



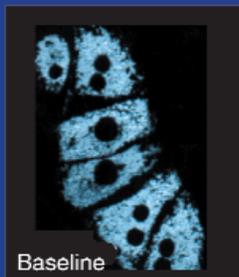
Microhydrin

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These results are indicating that the cell and membranes are resisting damage, as no signs of cellular stress or toxicity occurred during these tests when treated with Microhydrin®. Cells increased their energy production with the addition of Microhydrin®. It is also an indicator to researchers that the Microhydrin® silicate in a buffered solution is able to deliver the hydrogen reducing potential through the cellular membrane for incorporation into the mitochondrial electron transport chain. The combination of increased mitochondrial membrane potential and increased NADH suggest an enhancement of bioenergetic capacity of the mitochondria due to Microhydrin® in these preliminary results (unpublished results 1999).

Microhydrin Enhances Mitochondrial Membrane Potential in Intact Liver Cells Confocal Fluorescence Microscopy



200ug/ml Microhydrin added to cells

Microhydrin Enhances Mitochondrial Membrane Potential in Intact Liver Cells Confocal Fluorescence Microscopy

