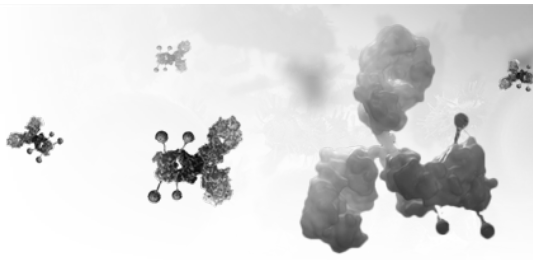

Goat anti He-dcyd polyclonal antibody (CABT-L526)

This product is for research use only and is not intended for diagnostic use.

PRODUCT INFORMATION

Specificity	Heptanone-etheno-2'-deoxycytidine
Target	He-dcyd
Immunogen	Dinitrophenol-modified protein
Source/Host	Goat
Species Reactivity	N/A
Conjugate	Unconjugated
Applications	ELISA, IHC, WB
Format	Liquid
Size	1 ml
Preservative	0.1% Sodium Azide
Storage	Short term: Refrigerate at 4˚C; Long term: Freeze at-20˚C

BACKGROUND

Introduction	The reaction of the lipid peroxidation product 4-oxo-2-nonenal (ONE) with 2`-deoxycystindine results in the formation of the DNA adduct heptanone-etheno-2`-deoxycystindine (HεdC). Analysis of DNA adducts in various human tissues demonstrate that HεdC is ubiquitous to many, if not all, human tissues. In several cases extremely high levels of these DNA adducts were found suggesting that they can represent significant DNA modifications with the potential

45-1 Ramsey Road, Shirley, NY 11967, USA
Email: info@creative-diagnostics.com
Tel: 1-631-624-4882 Fax: 1-631-938-8221

for deleterious effects on human health. HεdC has been shown to be highly mutagenic in both bacterial and human cells. When an HεdC adduct was placed in a plasmid that was replicated in both bacterial and human cells, it was able to completely block DNA replication in the bacteria and only small fractions of progeny were detected in the human cells suggesting that H εdC is a very strong block to DNA synthesis. Additionally, HεdC also strongly miscodes when bypassed in bacterial and human cell systems resulting in a miscoding frequency of about 45% and 90% respectively. HεdC has been found to be the most abundant DNA adduct in a colorectal cancer model where COX-2 is up-regulated. While COX-2 levels are low to undetectable in normal intestinal cells, toxic insults can lead to increased oxidative stress which produces an increase in reactive oxygen species leading to an increase in COX-2 levels. The decomposition of polyunsaturated fatty acids by COX-2 can lead to increased production of ONE and the subsequent modification of 2`-deoxycystindine to form HεdC. Using a mouse colorectal cancer model where the min mice produce excess COX-2 and are more likely to have spontaneous colorectal cancer than the wild type mice, it has been shown that increased levels of COX-2 lead to increased levels of HεdC. Additionally, min mice have a statistically higher level of intestinal HεdC than the wild type mice. HεdC has also been found to be the most abundant DNA adduct in breast epithelial cells that overexpress COX-2.

Keywords hedcyd; dcyd;Heptanone-etheno-2`-Deoxycytidine; εdC; He-dcyd;HE-DCYD;

45-1 Ramsey Road, Shirley, NY 11967, USA
Email: info@creative-diagnostics.com
Tel: 1-631-624-4882 Fax: 1-631-938-8221