Effects of acrylic-Induced Oxidative Damage on endoplasmic Reaction

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Abstract: order. study. effects. acrylonitrile (ACN)-induced oxidative damage. Endoplasmic Reticulum Stress (ERS) signaling pathways. Rat Liver 50 healthy adult male SD rats. randomly divided. 5 groups

10 rats. Group. According. body weight. Rats ' groups. treated. 0.012.52550.0 mg in kg⁻¹ACN via gavage. NAC group. treated by intragastric administration. 300.0 mg in kg⁻¹NAC 30 min reperfusion 50.0 mg in kg⁻¹ACN1 time/day 6 days/week. 13 weeks.. Levels. GSH, MDA. Activity SOD GSH-Px. Cat. liver tissue. measured by spectrophotometry method. mRNA, protein expressing levels. ERS-related GRP78CHOP, caspase-12. detected by RT-PCR, Western Blot.:

Showed. levels. GSH low, middle ACN group. significantly decreased compared. control group.. Activity. GSH-Px, SOD. Levels MDA. low dose group. significantly increased compared. control group.CAT activity. middle. dose groups. significantly decreased. comparison. control group.Compared. high ACN Group GSH levels. significantly increased. NAC intervention group. Also MDA levels. SOD activity. significantly decreased.GRP78CHOP, caspase-12. high ACN group showed significant higher mRNA levels. Group.. Expression CHOP, caspase-12 mRNA. NAC group. significantly lower.. high ACN Group.Western Blot showed. expression levels. GRP78CHOP, caspase-12 protein. high ACN group. significantly higher.. Group.. Expression levels. GRP78CHOP, Caspase-12 protein. NAC group. lower.. high ACN Group.Our study indicated. exposure. ACN could induce oxidative damage. rats ' Liv-er. Then activate ERS Signaling Pathway. NAC could reduce. degree. oxidative damage. antagonist. ERS Signaling Pathway.Further study. needed. find. mechanism. this Oxidative Damage inducted by ACN. ERS Signaling Pathway.

Keywords: Acrylonitrile; liver; oxidative damage; Endoplasmic Reticulum Stress
(Thapsigargin TG) And Bray feeder bacteria A (blefeldans BFA) of Poseidon yuann cell apoptosis and cell in ROS

Of tired about and apigenin (Apigenin AP), N-B Cysteine (N-acetylcytisteine NAC), GSH. Such as antioxidant may be expressed the suppression ROS of students and reduce TG and BFA. Cause ERSTips TG And BFA. Poseidon yuann Cell ERS 1, ROS. Tired Oxidation Induced caused AP Can antioxidant of role Suppression ERS. Cause of cell apoptosis and ERS Related [10].

Protein of expression Oxidation Induced can ERS of students. Dang sheng ERS Cell can be phase of signal ERS In order to protect cell of normal function. But when-induced anti-body can't complete repair start phase of apoptosis pathway cell apoptosis of students. NAC Is L-Cysteine of B compounds can dry Free Radical of generation and clear Generated Free Radical to antioxidant role increase body oxidation-induced of ability.

In recent years of research More liver disease of Disease Mechanism [11] And ERS cell apoptosis related And on ACN. Cause of liver whether and? ERS. About at present has not been Road. So this paper ACN SDRats chronic exposure with set N-B semi-Cysteine (NAC) Dry of ACN. Chronic exposure caused by rats liver Oxidation ERS Signal Pathway of influence. ACN Liver toxicity mechanism in-depth study provide science basis.

1. Material and Methods (Materials, methods)

1.1 Of and points

SPF Adult Health male SDRats 50 Only weight 250~300 G By Gan Traditional Chinese Medicine University Medical of center provide (Of qualified: Scxk (Gan) 2011-0001) Suitable 1 Weeks after body weight were randomly divided 5 Every 10 Only 12.5, 25.0, 50.0 mg in kg^{-1} ACN [12]. Irrigation stomach exposure, NAC First 300.0 mg in kg^{-1} NAC Irrigation stomach 30 min After reperfusion 50.0 mg in · Anti-Conditions 95 Of 2 min 95 Of 10 s 55 Annealing 15 s 40 A on. Finally, Pfaffi Method Analysis [14] MRNA Of phase expression.

1.2 Western Blot Step

Extraction liver protein quantitative and of system protein products line SDS-PAGE Gel swimming beam after Will objective protein moved PVDF Membrane on, 4, 200 Ma Membrane 75 min After 220 Ma Membrane 75 min. In TBST Preparation 5% Skim milk powder sealed 3 H 4, Level Bed night incubation a-(GRP78, CHOP, Caspase-12, GAPDH, β-Tubulin Protein A anti-5% Skim milk powder TBST Dilute dilute proportion points1: Anti-rabbit cloning two anti-dilute proportion 1: 2000 Goat Anti-Mouse Of Room Temperature (22 ± 1) Humidity 50% Night alternating.

1.3 Oxidation original related of Set

Exposure beam after, Die rats. Every random take 6 Only rats said take 100~200 mg Liver by weight (G): Uniform mediated (ML) = 1: 9 Of proportion fully Gridding low temperature centrifugal (42 R in min^{-1} 10 min) After sub-supernatant according to box operation tomorrow of step completes the oxidation original refers to.

1.4 RT-PCR Step

Before the start of the Will required equipment high bacteria Tr-izol Method extraction liver RNA 0.1% DEPC Water dissolved RNA After Set RNA Degree and quantitative 500 ng in · MuL^{-1} With. According Natural 20 MuL RNA Anti-anti-System (Total RNA 10 MuL RNase Free D H2O 6 MuL 5 × primerscript RT Master Mix/-sector in Real Time 4 MuL) Conditions, 42 Anti--60 min 70 And 5 min The anti-complete liver RNA Anti-CDNA. The light quantitative PCR Liver GRP78, PERK, CHOP, Caspase-12, β-Actin mRNA Table Up to Level (Primers sequence table I) PCR Anti-System Natural 20 MuL 2. Fruit

2.1 ACN Exposure and NAC Dry rats liver antioxidant ability

And fat oxidation of influence Factors variance analysis fruit showed low, in ACN Rats Liver GSH Content and As
than were reduce(P<0.05); LowACNRats LiverGSH-PxVitality and As than increased P<0.05). Low, highACNRats LiverSODVitality and MDACContent and As than were increased(P<0.05). In, highACNRats LiverCatVitality and As than were reduce (P<0.05). NACDryAfter reversibleACNOf Rats LiverGSHContent, MDACContent and SODVitality of change.

Because in objective protein molecular weight and reference protein points Sub-of close, A reference protein can't distinguish between so GAPDH, β-Tubulin 2A reference protein.

2.2 ACNExposure and NACDry Rats Liver ERS Related Gene Expression level of influence

RT-PCR Fruit showed highACNRats Liver GRP78, CHOP, Caspase-12 mRNA expression Level and As than were low High(P<0.05); low, inACNRats Liver GRP78, CHOP mRNA expression Level and As than were increased(P<0.05). NACDryAfter Rats Liver CHOP, Caspase-12 mRNA expression Level and ACN Than were reduce(P<0.05). Fruit table 3.

1 ACN Exposure and NAC Dry rats liver network induced related protein expression level of influence of immune printed Fig. I Western blot analysis. effect. ACN. EXPRESSION. ERS related proteins for, intervention. effects. NAC. Rats Liver Of rats liver is by Oxidation Induced caused.

Network is the body in protein synthesis, folding and

Oxygen, A of lack of, virus, oxidation-induced, toxicity of and the physical and chemical factors of situation Under CAN network of function ERS[^20]. Normal-under in network of molecular Glucose Protein 78 (glucose-regulated protein GRP78) And network membrane on the doubleRNA Network-induced Double-stranded RNA like endoplasmic reticulum Ki-nase PERK), Activation factor 6 (activating transcription factor-6 ATF6) And inositol need 1 (inositol requi-ring enzyme-1 IRE1) 3A cross-membrane protein each other together no activity when ERS Students a large number of not folding protein or folding protein will, including network cavity aggregation and. Network of normal Function this GRP78 Will and 3A cross-membrane protein dissociation and to of not folding or folding protein play a help protein folding of function reduce ERS So GRP78 Has been widely

Is ERS Students Records Of Points Sub. With And GRP 78 Solution from 3 Kind of feel protein was exposure activation PERK, ATF 6, IRE 1 3 Of way start ERS. ERS For cell self-preserving mechanism normal situation under involved in body external stimulation of multiple signal and gene network control has guaranteed cell holding its from the role but when ERS Of can

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