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ORIGINAL ARTICLE

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 Liver50 healthy adult male SD rats. randomly divided. 5 groups

10 rats. GroupAccording. body weight.. Rats 'groups. treated. 012.52550.0 mg in kg^{-1}ACN via gavage..NAC group. treated by intragastric administration. 300.0 mg in kg^{-1}NAC 30 min reperfusion 50.0 mg in kg^{-1}ACN1 time/day6 days/week. 13 weeks.. Levels. GSH, MDA. Activity SODGSH-PxCat. 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 GroupGSH 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, antagonize. 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

Cell leaching and fat dropGuangSuch.RatsACNIrrigation stomach exposure after,Rats LiverMDAContent increased,CatActivity drop[4] Low,GSHContent reduce;Regina mice of study also get similar of fruit tipsACNCan. Body of oxidation balance,Of liver. CometACNExposure of and Liver CellDNATrailing rate there-Anti-off

Oxidation Induced is refers to for antioxidant mechanism by in the free and reactive oxygen species and protein, fat and nucleic acid and in Polymer Anti-[6] Make after of the structure function the harm. Network-induced(Endoplasmic Reticulum StressERS)Is refers to when the outside world poison or stimulation caused by network(Endoplasmic ReticulumER)Of in Environment students changeERIn not folding protein or folding proteinERThe which environment of change and students of-induced. The most in recent years of related research most[7-8] Cause of liver and Oxidation Induced about and oxidation-inducedROSCan oxidation in retinal influence network Membrane onCA^2Ion of function caused by cell inCA^2Balance disorder,[9]

ERSAnd cell apoptosisSo Oxidation Induced isERSThe sensitive factor. In Vitro StudyERSPoison Hu Bu

(ThapsigarginTG)And Bray feeder bacteriaA (blefeldansBFA)Of Poseidon yuan cell apoptosis and cell inROS

Of Tired about and apigenin(ApigeninAP),N-B Cysteine(N-acetylcysteineNAC),GSHSuch as antioxidant may be expressed the SuppressionROSOf students and reduceTGAndBFACauseERSTipsTGAndBFAPoseidon yuan CellERSIsROSOf Tired Oxidation Induced causedAPCan antioxidant of role SuppressionERSCause of cell apoptosis andERSRelated[10]

Protein of expressionOxidation Induced canERSOf students. Dang shengERSCell can be phase of signalERSIn 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.NACIsL-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] AndERSOf cell apoptosis relatedAnd onACNCause of liver whether and?ERSAbout at present has not been Road. So this paperACN SDRats chronic exposure with setN-B semi-Cystine(NAC)DryOfACNChronic exposure caused by rats liver OxidationERSSignal Pathway of influence,ACNLiver toxicity mechanism in-depth study provide science basis.

1. Material and Methods(Materials, methods)

1.1 Of and points

SPF Adult Health maleSDRats50Only weight250~300 GBy Gan Traditional Chinese Medicine University Medical of center provide (Of qualified: Scxk (Gan) 2011-0001)Suitable 1Weeks after body weight were randomly divided5Every10Only 12.5,25.0,50.0 mg in kg^{-1}ACN[12]Irrigation stomach exposure,NACFirst300.0 mg in kg^{-1}NACIrrigation stomach30 minAfter reperfusion50.0 mg in · Anti-Conditions95 Of2 min95Of10 s55

Annealing15 s40A on. Finally,PfafflMethod Analysis [14] MRNAOf phase expression .

1.2 Western BlotStep

Extraction liver protein quantitative and of system protein products lineSDS-PAGEGel swimming beam after Will objective protein movedPVDFMembrane on,4,200 MaMembrane75 minAfter 220 MaMembrane75 min. InTBSTPreparation5%Skim milk powder sealed3 H4LevelBed night incubation a-(GRP78, CHOP,Caspase-12,GAPDH,β-TubulinProtein A anti-5%Skim milk powderTBSTDilute dilute proportion points1: Anti-rabbit cloning two anti-dilute proportion1: 2000Goat Anti-Mouse OfRoom Temperature(22 ± 1)Humidity50%Night alternating

1.3 Oxidation original related of Set

Exposure beam after,Die rats. Every random take6Only rats said take100~200 mgLiver by weight(G):Uniform mediated(ML) = 1: 9Of proportion fully Grinding 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-PCRStep

Before the start of the Will required equipment high bacteriaTr-izolMethod extraction liverRNA0.1% DEPCWater dissolvedRNAAfter SetRNADegree and quantitative500 ng in ·MuL^{-1}With. AccordingNatural 20MuL RNAAnti-anti-System(Total RNA 10MuLRNaseFree DH_2O 6MuL5 × primescript RT Master Mix/-sector in Real Time 4MuL)Conditions,42Anti--60 min 70And5 minThe anti-complete liverRNAAnti-CDNA. The light quantitativePCRLiver GRP78,PERK,CHOP,Caspase-12,β-Actin mRNATable Up to Level(Primers sequence table1)PCRAnti-SystemNatural 20MuL 2. Fruit)

2.1 ACNExposure andNACDry rats liver antioxidant ability

And fat oxidation of influence Factors variance analysis fruit showed low, inACNRats LiverGSHContent andAs

than were reduce(P<0.05);LowACNRats LiverGSH-PxVitality andAs than increased P<0.05). Low, highACNRats LiverSODVitality and MDAContent andAs than were increased(P<0.05). In, highACNRats LiverCatVitality andAs than were reduce (P<0.05).NACDryAfter reversibleACNOf Rats LiverGSHContent,MDAContent andSODVitality 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 andNACDry Rats LiverERSRelated Gene Expression level of influence

RT-PCRFruit showed highACNRats LiverGRP78,CHOP,Caspase-12 mRNAExpression Level andAs than were L High(P<0.05);Low, inACNRats LiverGRP78,CHOP mRNAExpression Level andAs than were increased(P<0.05).NACDryAfter Rats LiverCHOP,Caspase-12 mRNAExpression Level and ACNThan were reduce(P<0.05). Fruit table3.

1 ACNExposure andNACDry rats liver network induced related protein expression level of influence of immune printedFig.1 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[Natural 20]. Normal-under in network of molecular Glucose Protein78 (glucose-regulated proteinGRP78)And network membrane on the doubleRNANetwork-induced Double-stranded RNA like endoplasmic reticulum Ki-nasePERK), Activation factor6 (activating transcrip-tion factor-6ATF6)And inositol need1 (inositol-requi-ring enzyme-1IRE1) 3A cross-membrane protein each other together no activity whenERSStudents a large number of not folding protein or folding protein will, including network cavity aggregation and. Network of normal Function thisGRP78Will and3A cross-membrane protein dissociation and to of not folding or folding protein play a help protein folding of function reduceERSSoGRP78Has been widely

IsERSStudentsRecordsOfPointsSub. WithAndGRP78Solution from3Kind of feel protein was exposure activationPERK,ATF6,IRE1 3Of way startERS.ERSFor 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 whenERSOf can

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