proliferation, and cisplatin-resistance) were sustained afterremoval of TGF-β1.

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Determination of clanobutin residues from pork, milk, eggs using liquid chromatography tandem mass spectrometry

Dan Zhang[1], Jin-A Park[1], Seong-Kwan Kim[1], Sang-Hyun Cho[1], Weijia Zheng[1], Ho-Chul Shin[*1]

[1]Department of Veterinary Pharmacology and Toxicology, College of Veterinary Medicine, Konkuk University, Neungdong-ro, Seoul, 05029, Korea

Introduction: Clanobutin is widely used in cholagogue or choleretic drug in livestock.It is needed to develop a quanlitative and quantitative method to residue analysisin livestock products for food safety. The purpose in this study was to developa method for determining of clanobutin residues in pork, milk, eggs byLC-MS/MS.

Materials and Methods: The method for the determination was used a triple quadrupole massspectrometer operating in MRM mode under positive scan mode acquiring twodiagnostic product ions for clanobutin. The ion transition (parent ion > production) was 347.7 m/z >260.1, 245.1 m/z. The mobile phase was used mixture of 0.1% formic acid and 10 mM ammonium formate in water and acetonitrile. Theextraction solution was 0.1% formic acid in acetonitrile. The refined extractwas evaporated to dryness and diluted with mobile phase and the solution wasused for analysis.

Results: The limit of quantification was 1 μg/kg. The meanrecoveries of clanobutin in pork, milk, eggs was 79.24 ± 10.20, 100.45 ± 8.43, and 89.95 ± 5.22 (Mean ± RSD, %) respectively.

Conclusions: In this study, LC-MS/MS method has been applied successfully for determinationof clanobutin residue in pork, milk, eggs. The procedure is simple and allowsfor the high sensitivity determination of the residues. The method also evidencedacceptable recovery and precision. It will contribute to the animal productfoods safety.

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Determination of iodo quinolinesulfonicacid residues from pork, milk, beef using liquid chromatography tandem massspectrometry

Jin-A Park[1], Dan Zhang[1], Seong-Kwan Kim[1], Sang-Hyun Cho[1], Weijia Zheng[1], Ho-Chul Shin[*1]

[1]Department of Veterinary Pharmacology and Toxicology, College of Veterinary Medicine, Konkuk University, Neungdong-ro, Seoul, 05029, Korea

Introduction: Iodo quinolinesulfonic acid is widely used inveterinary medicine as anti-inflammatory drug. It is needed to develop aquanlitative and quantitative method to residue analysis in livestock productsfor food safety. The purpose in this study was to develop a method for determiningof iodo quinolinesulfonic acid residues in pork, milk, beef by LC-MS/MS.

Materials and Methods: The method for the determination was used a triplequadrupole mass spectrometer operating in MRM mode under positive scan modeacquiring two diagnostic product ions for iodo quinolinesulfonic acid. The iontransition (parent ion > product ion) was 349.74 m/z >223.0, 159.1 m/z.The mobile phase was used 0.1% acetic acid in water and methanol. Theextraction solution was 0.1% formic acid in acetonitrile. The refined extractwas evaporated to dryness and diluted with mobile phase and the solution wasused for analysis.

Results: The limit of quantification was 2 μg/kg. The meanrecoveries of iodo quinolinesulfonic acid in pork, milk, beef was 80.60 ± 5.58, 91.94 ± 6.80, and 98.20 ± 9.71 (Mean ± RSD, %) respectively.

Conclusions: In this study, LC-MS/MS method has been appliedsuccessfully for determination of iodo quinolinesulfonic acid residue in pork,milk, beef. The procedure is simple and allows for the high sensitivitydetermination of the residues. The method also evidenced acceptable recoveryand precision. It will contribute to the animal product foods safety.

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PPARγ activation of anti-diabetic agent, troglitazone enhances TRAIL-induced apoptosis via autophagy flux.

Nazim Uddin MD[1], Rasheduzzaman MD[1], Ji-Hong Moon[1], Ju-Hee Lee[1], You-Jin Lee[1], Jae-Won Seol[1], Sang-Youel Park[*1]

[1]Biosafety Research Institute, College of Veterinary Medicine, Chonbuk National University, Iksan, Jeonbuk 54596, South Korea

Introduction: Members of the tumor necrosis factor (TNF) cytokine family, such as TNFα and Fas ligand(FasL), play important roles in inflammation and immunity. TNF- related apoptosis-inducing ligand (TRAIL) is a member of this family that selectively induces cancer cell death but does not cause cytotoxicity to normal cells. Troglitazone and rosiglitazone are used in the treatment of type II diabetes to lower blood glucose levels and promote the sensitivity of a number of tissues to insulin. In this study, we demonstrated that troglitazone could induce TRAIL-mediated apoptotic cell death in TRAIL-resistant lung adenocarcinoma cells.

Materials and Methods: The human lung cancer cell line A549 was cultured in RPMI1640 and maintained at 37°C and 5% CO_2. Cells were treated for with troglitazone and then exposed to 200 ng/ml TRAIL,with or without the autophagy inhibitor. Western blotting were performed to detect gene products and proteins involved in PPARγ, ATG5, LC3 and p62 in cancer cells. Knockdown of ATG5 was also performed to investigate whether autophagy is associated with the PPARγ in cancer cells.

Results: Pretreatment of troglitazone activated PPARγ in a dose-dependent manner and conversion of LC3-I to LC3-II and PPARγ was inhibited in the presence of GW9662, which