Opinion

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Empagliflozin's Protection against Lipid Peroxidation and Cardiomyocyte Dysfunction

Minzhi Yunshan*

Department of Medical Sciences, Translational and Clinical Research Institute, Newcastle University, UK

***Corresponding author**: Minzhi Yunshan, Department of Medical Sciences, Translational and Clinical Research Institute, Newcastle University, UK, E-mail: yunshanminzhi@gmail.com

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Description

Cardio toxicity is the most common reason that a drug is pulled from the market because it is one of the major safety concerns associated with its use. At the moment, it has been reported that several drugs that have been shown to be clinically effective have a high risk of cardio toxicity when used in clinical settings. Doxorubicin, arsenic trioxide, isoproterenol, cyclophosphamide, and others are the most evocative examples. The adverse effects limit the use of the aforementioned drugs in clinical settings and have a significant negative impact on human health. Numerous strategies have been implemented to prevent drug-induced cardio toxicity over the past few years, including early detection of cardio toxicity using biomarkers, limiting doses, altering drug delivery methods, and combining with cardio protective agents. Among them, combining with a cardio protective agent has attracted more attention and been regarded as a promising strategy for on-going treatment.

Doxorubicin-Induced Cardio Toxicity Could Be Reduced *In Vivo* and *In Vitro* by Peril Aldehyde

As a result, finding an efficient cardio protective agent to prevent drug-induced cardio toxicity has become a significant challenge for many researchers. Interestingly, it has been demonstrated that some natural plant phenolic acid compounds have a significant protective effect against drug-induced cardio toxicity. Phenolic acids' role in drug-induced cardio toxicity and their cardio protective potentials were examined in this study to serve as a point of reference for future applications of phenolic acids in the fight against drug-induced cardio toxicity. For drug development, preclinical investigation of drug-induced cardio toxicity is crucial. *In vitro* high-throughput inter-digitated electrode-based recording of cardio myocytes mechanical beating is frequently used to evaluate such cardio toxicity. Artificial neural network analysis is typically used to automatically analyse the features from the beating signals for the drug-induced cardio toxicity assessment. Signals are segmented into cycles, and feature points are located in the cycles. However, the creation of particular algorithms is required for signal segmentation and the positioning of feature points for various signal shapes. As a result, research efficiency may suffer, and such algorithms can only be used with signals of varying morphologies. Nonlinear dynamic analysis-assisted neural network is used in this bio sensing system to directly extract features from beating signal time series instead of requiring signal segmentation. This NDANN-based bio sensing system can accurately identify drug-induced cardio toxicity with accuracy greater than 0.99 by processing beating time series with a fixed duration to avoid signal segmentation. The singular medications were ordered with high exactness's over 0.94 and drug-prompted cardio toxicity levels were precisely anticipated. Our NDANN-based bio sensing system's ability to screen newly developed drugs is demonstrated by this result, which is important for practical applications. This bio sensing system based on NDANN can make bio signal processing more efficient and serve as a new platform for screening for drug-induced cardio toxicity. Anthracyclines, which are used to treat cancer, are linked to significant cardio toxicity, which can cause death. Chemotherapeutic drug Doxorubicin is used to treat solid tumors and hematologic malignancies. However, its clinical application is hindered by life-threatening cardio toxicity, such as heart failure and dilation. Maintaining mitochondrial homeostasis in the cardiovascular system is aided by processes of mitochondrial quality control, such as mitophagy, mitochondrial proteostasis, and mitochondrial dynamics and biogenesis. Defective mitochondrial quality control appears to play a significant role in the etiology of DOX cardiomyopathy, according to recent research. Doxorubicin-induced cardio toxicity could be reduced *in vivo* and *in vitro* by peril aldehyde. Remarkably, peril aldehyde fundamentally hindered DOX-initiated cardiovascular inadequacy, myocardial injury, oxidative pressure, myocardial fibrosis, provocative reaction, and cardio myocyte apoptosis. In a novel approach, the inhibition of NHE1 phosphorylation and activation of PI3K/AKT phosphorylation are connected to the curative effect of peril aldehyde on the cardio toxicity caused by doxorubicin.

Epoxy Eicosatrienoic Acids Play a Significant Role in the Human Heart's Function

The severe cardio toxic effects of the broad-spectrum anti-tumour drug Doxorubicin restrict its clinical application. The primary component of the volatile oil that is extracted from the stems and leaves of the herbaceous plant-perilla is called peril aldehyde. This oil has antioxidant, anti-inflammatory, hypolipidemic, and other health benefits. The purpose of this study was to confirm a possible mechanism for the protective effect of peril aldehyde on DOX-induced cardio toxicity in rats. In rats subjected to DOX-induced cardio toxicity, the findings demonstrated that PAE could significantly ameliorate myocardial fibrosis, reduce oxidative stress, and suppress inflammatory responses. PAE could mechanistically cause DOX-induced cardio toxicity. As a result, the results indicate that peril aldehyde may be a promising cardio protective agent for the treatment and prevention of DOX-induced cardio toxicity. Despite its cardio toxicity, Trastuzumab is rarely used in clinical settings for target therapy in breast cancer patients with high HER2 levels. Cardio toxicity from anti-tumor medications is linked to DNA damage and Ferro ptosis. According to our findings, TZM challenge led to overtly elevated levels of troponin I and LDH in the blood, as well as detrimental myocardial re-modeling increased heart weight, chamber size, cardio myocyte area, and interstitial fibrosis, contractile dysfunction and intracellular Ca2+ mishandling, oxidative stress, lipid peroxidation, mitochondrial ultra-structural damage, DNA damage, apoptosis, and Ferro ptosis, all of which were. According to an *in vitro* study, Empagliflozin's protection against lipid peroxidation and cardiomyocyte dysfunction but not DNA damage was mitigated by the Ferro ptosis inducer erastin, while DNA damage mimicked TZM-induced lipid peroxidation and cardiomyocyte contractile dysfunction. Similarly, Ferro ptosis inhibition *in vivo* and *in vitro* replicated Empagliflozin's cardio protection against TZM exposure. Endogenous substances called epoxy eicosatrienoic acids play a significant role in the human heart's function. EETs have been shown to have physiological effects on vascular function regulation, angiogenesis, myocardial fibrosis, myocardial hypertrophy, and cardiovascular inflammation in animal models of CYP and sEH-related cardiovascular diseases. At the same time, clinical studies have shown that most CYP2J2 substrates and inhibitors alter the amount of EETs in drugs, which can cause cardio toxicity. Triptolide (TP), a key component of Tripterygium wilfordii, a well-known traditional Chinese medicine used to treat a wide range of autoimmune and inflammatory conditions, is one of its main components.