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Perspective

Drug Toxicity Development and Efficacy in Efficient to Low Blood Pressure

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DESCRIPTION

A number of cardiac (antiarrhythmic) and noncardiac medicines, particularly those that cause QT prolongation, have been linked to drug toxicity as a cause of SCA (e.g., psychotropic drugs and antibiotics, such as erythromycin and fluoroquinolones). Lethal ventricular tachyarrhythmias brought on by different medicines, however, are caused by a number of different electrophysiologic pathways. A potentially lethal arrhythmia may be started by concurrent anatomical and functional problems working together. An individual may be more susceptible to SCA if they use medicines and poisons together. Recent studies have shown a higher risk of SCA when cocaine and alcohol are used concurrently, presumably as a result of the production of the cardio toxic metabolite cocaethylene.

RHABDOMYOLYSIS

Drugs and Intoxications

Cocaine and heroin are the most common illicit drug-related causes of intoxications, with rhabdomyolysis complicating roughly 20% of cocaine overdoses. Rhabdomyolysis has been linked to other recreational substances like "bath salts" (of which methylenedioxypyrovalerone is the main component) and synthetic cannabinoids (often known as "spice"). Alcohol, amphetamines, phenylalkylamine derivatives, antipsychotic drugs, caffeine, and statins are additional substances that might cause rhabdomyolysis. Myalgias can occur in up to 10% of statin-taking patients, while rates of statin-induced rhabdomyolysis have been documented to range from 0 to 2.2 instances per 1000 person-years, with cerivastatin being linked to the highest rates. Statins can potentially cause an autoimmune anti-HMG-CoA antibody myopathy that is accompanied by severe increases in creatine kinase and proximal muscle weakening.

Exertional rhabdomyolysis has also been linked to dietary supplements, particularly those that contain mixtures of stimulants. When severe hyponatremia is promptly treated, rhabdomyolysis may also be observed.

Drug discovery and development depend heavily on the assessment of drug toxicity and safety. More than 70% of drug attrition and drug withdrawal are currently attributed to drug-induced toxicities in the liver, heart, kidney, and brain. To evaluate compound-induced cardio toxicity, skeletal muscle hypertrophy, hepatotoxicity, and neurotoxicity, label-free approaches like RWG and electric biosensors can be utilized.

One of the most frequent reasons why medications are discontinued is drug-induced cardiovascular toxicity, hence it is important to utilize better, more accurate *in vitro* models and high-throughput approaches to evaluate cardio toxicity early in the drug development process. Cardiomyocytes produced from stem cells are extremely reliable and physiologically appropriate models to precisely measure cardio toxicity caused by substances. By observing the impact of medications on the beating patterns of primary or stem cell-derived cardiomyocytes, the recently developed high frequency electric impedance biosensor device has been used to identify compound-induced cardio toxicity. It was discovered that this system could reproduce the effects of several well-known modulators of heart function. Recently, we have created a high frequency RWG imager to assess the cardio toxicity caused by the chemical. The results demonstrated that after 11 days of cultivation, cardiomyocytes produced from induced pluripotent stem cells form a monolayer on the surface of the biosensor and beat in synchrony, producing a regular beating pattern made up of a sequence of small valleys. When cardiomyocytes were exposed for one minute to the hazardous chemical isradipine, their heartbeat was entirely stopped.

Drug toxicity, which can have an impact on a variety of biological processes involving one or more target organs, continues to be a significant obstacle in the drug discovery process. The most frequently reported target organs and safety concerns that must be addressed before and during clinical development include the heart and liver, kidney, central nervous system, developmental and genetic toxicity, and. A close relationship exists between computational prediction and experimental validation in rational approaches to medication safety. The entire drug development value chain is covered by the applications for computational toxicology.

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Only under ideal circumstances can effectiveness be adequately measured. In a group of patients who are most likely to respond to a medicine, such as in a controlled clinical study, efficacy is therefore assessed under the guidance of an expert.

A medicine that is effective in clinical trials is frequently ineffective when used as directed. For instance, a medicine may be highly efficient at lowering blood pressure but be ineffective overall if patients stop taking it due to the myriad of side effects it produces. If doctors unintentionally give the wrong medication, effectiveness may also be lower than efficacy.

A less subjective calculation of a drug's anticipated benefits is called the Number Needed to Treat (NNT) (or any other intervention). The number of patients that must be treated before one patient can benefit is known as the NNT.