Utilize Circulating Genetic Variants as Biomarkers for Liver Damage brought on by Drugs

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Editorial

The adverse occurrence known as Drug-Induced Liver Injury (DILI) frequently results in the suspension of drug testing in clinical trials, drug use limitations, and drug withdrawals. Up to half of the preclinical candidate compounds that are abandoned owing to organ toxicity are thought to be the result of hepatotoxicity. Serum alanine aminotransferase and bilirubin are two examples of existing biomarkers of liver injury that offer decent signs of damage, but none of them are sufficiently specific or rise until after major damage has already taken place. For the evaluation of pharmaceuticals in both the clinical and preclinical stages, new trustworthy biomarkers of DILI are urgently needed. Authors that find new DILI biomarkers can increase the effect of their research articles with the use of open access [1].

Since they were first discovered in Caenorhabditis elegans, miRNAs have gained recognition as novel agents that exert post-transcriptional control over the majority of eukaryotic genomes. miRNAs are short (about 21 to 25 nucleotides in length), single-stranded, non-coding RNAs. MiRNAs are highly conserved across a wide range of animals, including humans and worms, indicating their extremely distant ancestry. According to reports, miRNAs play fundamental roles in cellular processes including development, cellular differentiation, proliferation, apoptosis, cell-cycle control, metabolism, and cancer [2]. They are expressed in all animal cells and have been linked to these processes. It is estimated that ~60% of human mRNAs are regulated by miRNAs, suggesting that miRNAs might very well form another layer of the regulatory circuitry that exists in the cell. Similar to mRNA, some miRNAs are produced in cell- or tissue specific manners. Recently, many reports concerning miRNAs related to disease have been published and suggest that miRNAs may serve as a new kind of biomarker for organ injury [3].

According to Wang et al. study's using a mouse model of DILI produced by acetaminophen, several plasma miRNA levels were found to be negatively linked with hepatic miRNA levels, indicating that hepatic damage was the cause of the release of these miRNAs into the bloodstream. Particularly, plasma levels of miRNA-122 and miRNA-192, which are mostly expressed in the liver, increased while liver levels decreased. Before the increase in ALT, the increases in both miRNAs were noticed, recently showed that urine miRNA profiles were altered in rats after administration of hepatotoxic dosages of acetaminophen or carbon tetrachloride, which was recently verified to cause an increase in serum miRNA-122 and miRNA-192 in patients with acetaminophen poisoning. In rats treated with acetaminophen and carbon tetrachloride, there was an increase in the concentration of the same 10 urinary miRNAs. Additionally, the patterns of reactions to the two hepatotoxicants could be distinguished from those to a nonhepatotoxicant (penicillin) and vehicle controls using miRNA expression profiles [4].

Although creating protein-based assays is theoretically more complex and vulnerable to more confounding factors than miRNA measurement, there are certain technological difficulties. Interlaboratory heterogeneity in miRNA quantification is a major issue, especially when multiple platforms are employed. The two most popular platforms for finding global miRNAs are RT-qPCR and microarrays; another recent technique based on direct sequencing is Next-Generation sequencing. Unfortunately, it appears that there is now little correlation between the various platforms. This is probably because varied primer designs and sequence heterogeneity have a significant impact. Comparisons between research are especially challenging due to the absence of standardised sample preparation techniques. Assessment of miRNA amount and quality is not as accurate as it is for total RNA. Finally, a range of alternative normalisation techniques have been applied, particularly for cell-free miRNA research, to account for technical and biological variability, although none of them is perfect [5].

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