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Mini Review

CARDIOMYOCYTES DERIVED FROM INDUCED PLURIPOTENT STEM CELLS FOR DRUG DEVELOPMENT

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ABSTRACT

By providing platforms for studying the mechanisms of disease pathogenesis that could lead to new therapies or reveal drug sensitivities, induced pluripotent stem cell (iPSC) technology is creating exciting new opportunities for cardiovascular research. The potential applications of iPSC-derived cardiomyocytes in drug development and drug toxicity testing are examined in this review, with an emphasis on the accomplishments that have already been achieved in these areas. In addition, the essential actions that must be taken before this technology can be utilized extensively in drug discovery and toxicology evaluations are emphasized. The rapid adaptation of the technology to human cells and the discovery that somatic cells can be reprogrammed into pluripotent stem cells, which are capable of differentiating from all adult cell types, have led to high expectations regarding the potential applications.

Keywords: Cardiomyocyte; Cardiovascular; Drug sensitivities

INTRODUCTION

The field of cardiovascular medicine, which deals with cell types that are difficult to obtain from human patients or probands, finds the technology particularly appealing. The initial reports on the generation of human iPSC have already highlighted the potential usefulness of iPSC technology in drug development and drug toxicity testing among the cardiovascular field's potential applications. The purpose of this review is to define the potential role that iPSC-derived cardiomyocytes could play in this situation, to highlight the progress that has already been made in this area, and to talk about the important steps that need to be done before this technology can be used in drug development and toxicity testing in a large scale. The ID and portrayal of potential medication focuses on, the evaluating of compound libraries for drugs with an ideal impact, as well as the assessment of medication possibility for conceivable unfavorable impacts all require solid test frameworks. These test systems can be created using animal models, immortalized cell lines, or engineered from primary cellsHowever, the current cardiomyocyte-based test systems have a number of drawbacks for cardiovascular pharmacology [1,2].

DISCUSSION

It is difficult to obtain primary human cardiomyocytes and they cannot be expanded in vitro or maintained in culture for extended periods of time. There are no immortalized human cardiomyocyte cell lines that accurately replicate crucial cardiac physiology features like action potentials. Alternately, overexpression systems of the potential drug target molecule can be produced using human cell cultures derived from embryonic sources, such as HEK lines. This permits concentrating on the impacts of a medication on a particular quality or sub-atomic instrument, yet neglects to give data on the compound's generally cell (cardiomyocyte) result. As a result, animal models are used in a lot of this field's research at the moment. For instance, hereditarily changed mice are as often as possible used to concentrate on the physiology that underlies human coronary illness. However, if cardiomyocytes from laboratory animals are used to simulate aspects of human cardiovascular disorders, species differences pose a significant challenge. Murine hearts, for instance, beat about six to ten times faster than human hearts and have significantly shorter action potentials that are shaped by various ion channels. The long-QT syndrome type1 disease characterized by a prolonged QT interval in the electrocardiogram and a susceptibility to potentially fatal arrhythmias may be caused in humans by mutations of the KCNQ1 gene, which encodes the ion channel that is responsible for the IKs current. However, KCNQ1 genetic ablation in mice did not always result in a cardiac phenotype comparable to that of patients with long-QT syndrome. The fact that the repolarization of the cardiac action potential is controlled by different ionic currents in mice and humans (for example, the delayed rectifier currents IKr and IKs are the main repolarizing currents in humans, but other currents, like the transient outward potassium current Ito, play important roles in the repolarization phase of mouse ventricular myocardium) is most likely the root cause of these differences. Not only are these species differences important for modeling a rare disorder like congenital long-QT syndrome, but they also raise questions about the reliability of rodent models for predicting drug-induced QT interval prolongation, which is a major problem in drug development because it could have negative effects. Better human heart disease model systems are needed individually and have not yet been developed. By providing an unlimited supply of both healthy and diseased human cardiomyocytes, induced pluripotent stem cells (iPSC) have the potential to close this gap. In particular, there are at least three areas in which pharmacology and toxicology could benefit greatly from cardiomyocytes derived from iPSCs. The discovery of novel drug targets is one area. Cardiomyocytes that are patient-specific can be obtained from patient-specific iPSC lines that have been generated from genetically inherited diseases. In vitro examinations with these cardiomyocytes could prompt the ID of atoms engaged with the pathophysiology of the illnesses that address conceivable novel medication targets. Utilizing iPSC-derived cardiomyocytes in phenotypic assays to search compound libraries for cardiovascular-beneficial effects is another way in which they have the potential to advance pharmacology. Safety pharmacology is the third topic. Candidate drugs must be checked for cardiotoxicity, including but not limited to their ability to prolong the QT interval and cause torsades de pointes. Phenotypic assays based on iPSC-derived cardiomyocytes have the potential to either supplement or replace assays based on primary cardiomyocytes from laboratory animals or cell lines overexpressing ion channels,

which are currently in use. It is widely acknowledged that cardiomyocytes derived from iPSCs have a significant potential to advance drug discovery and toxicity testing. However, a number of significant issues must be resolved in order to realize this potential. If these cells are to be included in standardized assays, it will be necessary to standardize the methods for iPSC generation, cardiac differentiation, and quality control [3-6].

CONCLUSION

The commercial availability of well-characterized cardiomyocytes derived from iPSCs may be a significant step toward this objective. Another crucial step is the improvement of differentiation protocols to produce cells that are more like adult cardiomyocytes. The immature phenotype of cardiomyocytes generated using current protocols may or may not be a problem, depending on the intended use. The degree to which the phenotype observed in iPSC-derived cardiomyocytes treated with a particular drug correlates with the clinical findings in patients treated with the same drug is the most crucial question that needs to be answered for each proposed assay. Before the assay's usefulness can be reliably predicted, this will need to be systematically investigated by testing as many drugs as possible.

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