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Perspective

Applications of Molecular Therapies and Cardiac Physiology

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DESCRIPTION

Molecular therapeutics for neuromuscular diseases are developing quickly and show promise for life-threatening conditions with limited available treatments. The muscular dystrophies that are the focus includes Duchenne, Becker, and limb girdle muscular dystrophies. Exon skipping with antisense oligonucleotides or mutation suppression needing tiny compounds that allow dose-dependent stop codon readthrough are two methods for repairing damaged genes. These goods have undergone or are undergoing clinical trials. Muscular dystrophies are being targeted by gene replacement techniques, and the potential for delivery is growing by vascular route administration throughout the extremities. A promising advancement in gene therapy uses a strategy for gene expression through recombination techniques, which increases the potential for delivering genes that are often too large to fit inside an adeno-associated virus. The therapeutic genes that do not replace but rather speed up functional recovery through alternative pathways, including myostatin inhibition, are also covered . It is simple to overlook the fact that only a small percentage of cancer patients will ever be eligible for these medicines when faced with the vast number of targets for molecular therapy. Advanced, malignant solid tumor patients are typically treated with non-targeted chemotherapy. It is common knowledge that two people with the same tumor may not respond to chemotherapy in the same way. It's likely that these disparate reactions are caused by molecular variations between malignancies. In fact, it makes sense that specific markers predicting chemotherapy response could be found given that the mechanisms of action of the majority of chemotherapy agents are related either to enzymes involved in DNA repair, such as ERCC1 and platinum chemotherapy, or to the transport of molecules across the cell membrane.

There is currently no known treatment for lymphedema, despite the fact that numerous therapeutic approaches efficiently reduce extra volume, reduce complications, and improve function. Due to these factors, attention has been placed on the potential use of efficient molecular treatments. Therapeutic lymphangiogenesis, which is based on understandings of the developmental biology of the lymphatics, is now the most fascinating of these. Even while it is obvious that exercise conditioning is helpful in cardiac disease, it is yet unclear whether modulation of these molecular targets, such as PI3K/Akt, C/EBP, or paracrine agents like PGF or IL-6, has the capacity to affect heart physiology in a clinical setting.

CONCLUSION

A molecular therapy that aims to locate and target these important molecular targets may also come with hazards. First off, it is abundantly obvious that physiologic hypertrophy is a coordinated response of the myocardium to a physiological causes, not just the cardiocyte level but also the mitochondrial function, extracellular matrix content, and vascularity.

It could be harmful to focus only on one aspect of this answer while ignoring the others. For instance, one may design a cardiac phenotype that has enhanced contractile ability but is substrate- and/or energy-limited (although the fact that transgenic animals overexpressing PI3K activity appear to do well and transgenic animals overexpressing PGF are protected against failure secondary to pressure overload is encouraging). Additionally, the connection to a cell proliferation signal (through CITED4) raises concerns about the complexity of ventricular remodeling in the setting of the adult heart, although being potentially very interesting. Exercise is also a dose-dependent and intermittent stimulus, making it difficult to develop a molecular therapeutic that replicates this distinct physiology.

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