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International Journal of Life Sciences
(ISSN: 2277-193x) (Scientific Journal Impact Factor: 6.106)

UGC Approved-A Peer Reviewed Quarterly Journal

Short Communication

Artificial Intelligence and Cardiovascular Medicine

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ARTICLE DETAILS

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Key words:
Artificial Intelligence, cardiovascular medicine, Angioedema, proactive management, healthcare, electronic health records. etc.

ABSTRACT

Artificial Intelligence (AI) and cardiovascular medicine represent distinct fields, yet their synergistic potential is vast, offering significant opportunities for advancement in healthcare. AI technologies, encompassing machine learning and data analytics, play a pivotal role in reshaping cardiovascular medicine, contributing to improved diagnostic precision, treatment strategies, and overall patient outcomes. Within diagnostics, AI algorithms analyze extensive datasets from medical imaging, such as MRIs or CT scans, identifying subtle patterns indicative of cardiovascular conditions. This accelerates the diagnostic process, ensuring prompt interventions. Machine learning models extend their capabilities to predicting the risk of cardiovascular events, drawing insights from a diverse range of patient data, including genetic information, lifestyle factors, and electronic health records. In treatment planning, AI facilitates the customization of therapeutic approaches. Considering individual patient characteristics and responses to treatments, AI systems recommend personalized interventions, optimizing effectiveness while minimizing side effects. Furthermore, AI contributes to the creation of predictive models, enabling healthcare professionals to anticipate potential complications and proactively manage patient care. This symbiotic relationship between AI and cardiovascular medicine holds immense promise, ushering in a new era of healthcare characterized by enhanced precision, personalized treatment, and proactive management of cardiovascular conditions.

1. Introduction

This article is an introduction to the well-known and intensively studied topic called cardiovascular medicine. The addition of Artificial intelligence to the field of cardiovascular medicine is similar to adding a cherry on top of a sweet or icing on the cake. The paper also aims to speak and explore more basic, rigorous, curiosity based open ended questions which have also been researched extensively but the current studies do not delve deeper into the subject to explore more basic, multi-disciplinary and interdisciplinary nature of the subject and this article also is an endeavor to explain some of the most ***important and thought provoking questions:***

1. Evaluate the bidirectional relationship between Angioedema and Brugada syndrome in terms of differentiated gene expression regulation.
2. Evaluate the relationship between gluconeogenesis and Brugada syndrome.

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Received: 25-December 2023; Sent for Review on: 26-December 2023; Draft sent to Author for corrections: 01 January 2024; Accepted on: 06- January 2024
Online Available from 10- January 2024
DOI: 10.13140/RG.2.2.24664.47364

3. Evaluate the relationship between gluconeogenesis, acetol monooxygenase , and pulmonary edema.
4. Evaluate the relationship between Angioderma and Kaposi sarcoma and it's impacts on the Brugada syndrome.
5. Evaluate the relationship between Angioderma and Glioblastoma and it's impacts on the Brugada syndrome.
6. Evaluate the relationship between Angioderma and A53 diffuse large B cell lymphoma and it's impact on Brugada syndrome.
7. Evaluate the relationship between Angioderma and Aapoaii amyloidosis and it's impact on various cvds like hypersensitivity vasculitis, Brugada syndrome, congenital myopathy 5 with cardiomyopathy, Myopathy myofibrillar 1 etc.
8. Evaluate the relationship between Angioderma and Aapoaii amyloidosis and it's impact on various cvds like combined oxidative phosphorylation deficiency.
9. Evaluate the relationship between Angioderma and Myopathy myofibrillar 1, 2,3,6 .
10. Evaluate the relationship between the down regulated differentially expressed genes of Autism spectrum disorder and PCOD and its impact on heart regeneration and development.
11. Evaluate the relationship between Angioderma and Copy number variants of AMY2A and PTEN and it's impact on heart regeneration in humans, neonatal mice and zebrafish models.
12. Evaluate the relationship between Angioderma and Aapoaii amyloidosis and the impact of the Metagenomics, electronics, comparative genomics, Neurogeneomics, Cognitive genomics, Pangenomics, Panomics, Precision genomics in relationship with Acute lymphoblastic leukaemia, Acute myeloid leukaemia, Adrenocortical carcinoma, Kaposi sarcoma, lymphoma, primary CNS lymphoma, Anal cancer, Appendix cancer, Astrocytomas, Atypical teratoid , rhabdoid tumor, and it's effects on Myopathy myofibrillar 1, 2, 3, 6,7,10,11 .
13. Evaluate the relationship between cognitive genomics, Neurogeneomics, functional genomics, comparative genomics, pangenomics, culturomics, Panomics, Precision genomics, on Basal cell carcinoma, bileductcancer, bladder cancer, bone cancer, Glioblastoma, breast cancer, bronchial tumor, Burkitt lymphoma, Non hodgkin lymphoma, and their effects on various cvds like coronary artery disease autosomal dominant 1, Danon disease.
14. Evaluate the relationship between cognitive genomics, functional genomics, precision genomics, genomics, pangenomics, and Carcinoma of unknown origin, atypical teratoid , rhabdoid tumor medulloblastoma, CNS embryonal tumor, germcell tumor, primary CNS lymphoma, cervical cancer, cholangiosarcoma, chordoma, chronic lymphocytic leukaemia, cutaneous T cell lymphoma, and it's impact on the cardiotranscriptome , cardiometabolome, cardiocelluome, cardioelectrome, and the cardiomultiomics.
15. Evaluate the impact of the differential gene expression of the NGR3 Gene 10q23.1 chromosomal locus deletion and it's impact on various cvd like conjunctival disease and cardiogenic shock.
16. Evaluate the impact of differential gene expression of the oral mucosa in the cvds like Danon disease, cardiogenic shock, conjunctival disease.
17. Evaluate the impact of bidirectional relationship between AMPK signalling and oral mucosa and it's impact on finding new biomarkers for cvds.
18. Evaluate the relationship between AMPK signaling and Crumbs3 transcription factor and it's impact on cognitive genomics and differential gene expression in the cvds like Brugadas syndrome, long QT syndrome, and ventricular tachycardia.
19. The relationship between Crumbs 3 transcription factor regulation, AMPK signaling and the differential gene expression of oral mucosa due to hyperacidic environment and it's impacts on the cvds like cardiomyopathy familial hypertrophic 27, Gaucher disease type 1.
20. Evaluate the effect of excess AMPK signaling on Crumbs3 regulation, effect of culturomics, and cognitive genomics, cellulomics, and the relation between zeb2 transcriptional factor regulation and it's impact on cvds like Danon disease and rheumatoid arthritis.
21. The between Autotomy of cancerous cells and Mirna 154 regulation and it's impact on Gauchers disease type 2, Gauchers disease type 3 c.
22. The relation between SH2 domains and Mirna 154 and it's impact on cvds like glycogen storage disease 5 and 6.
23. The relation between the differential gene expression of cell cycle genes like cdc4 , cdc2, cdc42 in the cognitive genomics and it's impact on the cvds like Danon disease and glycogen storage disease type 3 a and achalasia.
24. The relation between E-3 ubiquitin ligase in heart regeneration and it's differential gene expression during cvds like achalasia and cardiofaciocutaneous syndrome and in homeostasis.

25. The relation between AHA1 gene and it's functional analysis on cognitive genomics, Neurogeneomics, functional genomics, precision genomics.
26. The relation between the AHA 1 gene and STAT3 protein in zebrafish heart regeneration and the relation between the cognitive genomics, pangenomics, phenome wide studies, in mouse model and zebrafish models.
27. Evaluate the impact of the cognitive genomics, Neurogeneomics, comparative genomics, Panomics, cellulomics, precision genomics on CBPL5/ BRL5 strain of mouse and in AMPK mutants of opossum models and also mRen2 mutants in murine model.
28. Evaluate the impact of nutriomics of ketogenic and glucogenic diet and it's relation with the BALKB/CT mutant strain of mouse.
29. Evaluate the relationship between the effects of cellulomics , Panomics , pangenomics , comparative genomics, precision genomics, and their effect on differential gene expression in C57BL/6 Boy strain and SJL mice .
30. Evaluate the impact of culturomics, of Mozart, comparative genomics, with the skeletal muscle transcriptome, of C57BL6/Boy strain with an inbred strain of NOD. CB17 / Prkdscidl/J strain.
31. Evaluate the impact of long non coding rnas in the pathophysiology of Acromial dimples , and it's bidirectional relationship between Brugadas syndrome, tetralogy of fallot, Atherosclerosis, ventricular tachycardia, and bradycardia, hypertension, rheumatic heart disease, dilated cardiomyopathy, familial hypercholesterolemia , aortic aneurysm, thrombolic cytopenia, peripheral artery disease.
32. Evaluate the impact of the GWAS studies, Transcriptome wide studies, Proteomic association studies , electrome widein studies , metabolome wide studies , of AHA1 , Crumbs3 , PTEN, Oct2, Nanog, Sox, Klf on the various cvds like Danon disease and mitochondrial phosphate carrier deficiency.
33. Evaluate the impact of Genome wide studies (GWAS), Transcriptome wide studies (TWAS), Metabolome wide studies (MWAS), Electrome wide studies (EWAS), studies of A2M, ABL1, ADCY5, AGPAT2 , AGTR1, AIFM1, AKT1, APEX1, APOC3, APOE, APP, APTX in various cvds like mitochondrial DNA depletion syndrome 1, 3, 6b, .
34. Evaluate the impact of the GWAS, TWAS, PWAS,MWAS, EWAS, studies of AR, ARGHAP1, ARNTL, AIF2, ATM, ATp50, ATR, BAK1, BAX, BCL2, BDNF, BLM, BM1, BRCA1, BRCA2, BSCL2, BUB1B, BUB3, C1QA, CACNA1A, CAT, CCNA2 , CDC42, CDK1, CDK7, CDKN1A, CDKN2A, CDKN2B, CEBPA, CEBPB, CE1P, CHECK 2, CISD2, CLU, CLOCK , CNR1, COQ7, CREB1, CSNK1E, CTF1, CTGF, CTNNB1, DBN1, DNAJC1, DDIT3, DGAT1, DLL3, E2F1, EEF2, EFEMP1, EGF, EGFR, ELN, EMD, EP300, EPOR, EPS8, ERBB2 in various cvds like mitochondrial DNA deletion syndrome 5,9,12 and microphthalmia in zebrafish and opossum models.
35. Evaluate the impact of SCN lesions on heart regeneration in zebrafish and opossum models and also in mice models.
36. Evaluate the impact of X inactivation in zebrafish and opossum heart regeneration model.
37. Evaluate the impact of vaginal microbiome in human heart regeneration.
38. Evaluate the impact of glycomical studies on various cvds like mitochondrial DNA depletion syndrome 6, microphthalmia syndromic 6.
35. Evaluate the impact of foodomical studies on various cvds like mitochondrial DNA deletion syndrome 12, vascular Myelopathy, corneal neovascularisation.
36. The impact of metabolomical studies on various cvds like primary triglycerides deposit cardiomyovasculopathy.
37. The impact of culturomical studies on various cvd like atrial standstill and doxorubicin induced cardiomyopathy.
38. The impact of culturomical studies on the prognosis of atherosclerosis.
39. The impact of biomes on cvds.
40. The impact of ethomics and videomics on cvds .
41. The impact of cognitive genomics, precision genomics, pangenomics, Panomics, comparative genomics, Neurogeneomics, on cvds like Danon disease, Brugada syndrome, tetralogy of fallot, alcohol septal ablation, amyloid heart disease, aneurysm of abdominal, aortic, thoracic, and peripheral artery disease.
42. The impact of culturomical studies on the pharmacogenetical approaches in gene knockout using CRISPR screens.
43. The impact of large molecular protein aggregates, solid and liquid condensates formed during the exocytosis of the lysosomes.

2. Conclusion

In a nutshell, the collaborative interplay between AI and cardiovascular medicine holds the promise of ushering in an era marked by heightened precision, personalized care, and proactive healthcare management. This symbiotic relationship has the potential to revolutionize how cardiovascular conditions are diagnosed, treated, and managed, ultimately improving the overall landscape of healthcare outcomes in this critical domain.

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