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The same gene may produce both the trouble in development and increased susceptibility to cardiotropic viruses in ARVD extended to cardiomyopathies

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ARVD is the most frequent presentation of ARVCs which includes Brugada syndrome, RVOT VT etc. A recent report has demonstrated for the first time that two mutations (one is new) the other is desmoplakine (a desmosomal protein) seems responsible for both the induction of the trouble in development but also biologic signs of inflammation probably due to an increased susceptibility to cardiotropic viruses (not yet identified). This concept is in agreement with a decrease in LVEF observed sometimes abruptly during the time course of ARVD leading in the most severe cases to irreversible CHF. It seems possible to extrapolate this phenomenon to other forms of cardiomyopathies known to show the spectrum of multiple aspect of myocarditis going from the fulminant form leading to hyper acute heart failure to complete healing without sequel including chronic, chronic active and completely healed myocarditis. The problem is therefore to identify the inflammatory agent which can be performed nowadays by the recent technic of time of flight viral RNA identification by mass spectroscopy. This approach needs endomyocardial biopsy which is a safe procedure if properly performed by a trained personnel following a strict protocol. In particular IDCM and HCM are well known to exhibit the same clinical patterns which can explain the wide spectrum of prognosis observed in these conditions. The abolition of the phenotype by small molecule in ARVD Zebra fish and mouse also open a completely new formidable area of resarch for the future.

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Identifying active molecules from Chinese her medicine with *in silico* and *in vitro* approaches

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With the super computing support from TH-2, we have developed *in silico* tool kits to virtually screen Chinese herbs against specific diseases, and identify the active molecules and their targets. The *in silico* study results then guide us to design our in vitro experiments for bioassays and elucidating mechanisms of actions. Employing this protocol, we discovered *Sauruaceae* as the Epstein–Barr virus (EBV) inhibitor, and identified active molecules from *Buzhong yiqi tang* (Chinese compound herbal medicine) for myasthenia gravis. Phytochemistry and chemoinformatics studies revealed the active compounds were lignans and their target was topoisomerase 2 (Topo II) for the EBV inhibitors. With virtual and biological screening experiments, we revealed that the active compounds of *Buzhong yiqi tang* were 2-phenyl-benzo heteropentacyclic mimics, which led to the discovery of new acetylcholinesterase inhibitors. These methods pave new road for phenotypic and target based drug innovations.

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