Daa By Udit Agarwal

??Holt,Rinchart and Winston 1983??????. -- ??: Modern digital and analog communication systems/B. P. Lathi
??????????
??????????????????????,????????????????????,????????????????
????????????????????
?????
?????????????C++?????????.????:?,?,??,?,???,????,??,??????,????,????,????,?
???,?????,????,???,k-d??????.
????????????????????????????????:??
???NP????????????????????????????????????
????????????????????? “???”????????????
??????27?,?????UML ????;????????????????????????;????????????????????????
?????:????
????????????????????????????
????:Edible mushrooms and their cultivation

The term ventricular remodeling refers to the series of events that lead to the orchestration of the biological processes that determine left ventricular size, shape, and function following acute myocardial infarction (AMI). The present study focuses on defining the mechanisms of two distinct phenomena that are known to regulate this process; namely, extracellular matrix (ECM) degradation via protease inactivation and cardiac myocyte death. AMI is followed by degradation of ECM of the heart leading to thinning and rupture of the left ventricular wall. A critical regulator of ECM degradation is plasmin, a protease. The generation of plasmin in turn is regulated by the oxidation-sensitive enzyme, Plasminogen Activator Inhibitor -1 (PAI-1). PAI-1 is an inhibitor of urokinase-like plasminogen activator (uPA), which converts plasminogen into plasmin. PAI-1 deficient (Pai-1-/-) mice die of ventricular rupture within 7 days following infarction. It has been previously reported that the activity of PAI-1 is sensitive to oxidation and hence inhibited by leukocyte-derived oxidant-generating systems, such as Myeloperoxidase (MPO) and inducible nitric oxide synthase (iNOS). Furthermore, the expression of ceruloplasmin (CP), another oxidant- generating enzyme, is increased in the blood post-AMI. Reduced nicotinamide adenine dinucleotide phosphate (NADPH) oxidase activity regulates the oxidant-generating capacity of MPO, iNOS, and CP. However, the relative contributions of each of these leukocyte-derived oxidant producing enzymes in PAI-1 oxidation is not known. We hypothesized that leukocyte-derived NADPH oxidase oxidizes PAI-1 and hence participates in ventricular rupture post-AMI. To address this issue, we transplanted Pai-1-/- mice with Pai-1-/-, wild type (WT), p47phox-/- (a subunit of NADPH oxidase complex, also known as neutrophil cytosolic factor-1), Mpo-/-, iNOS-/- and Cp-/- bone marrow, performed Left Anterior Descending (LAD) artery ligation in all the groups, and measured their survival for 21 days. Data demonstrate Pai-1-/- mice were dead by day 7 due to ventricular rupture and WT marrow partially rescued Pai-1-/- mice survival. Interestingly, p47phox-/- marrow-transplanted mice had a significantly better survival compared to WT marrow-transplanted Pai-1-/- mice suggesting that, NADPH oxidase is critical in inhibiting PAI-1-induced protease inactivation and ventricular rupture. PAI-1 activity was higher in p47phox-/- marrow-transplanted Pai-1-/- animals compared to WT marrow-transplanted Pai-1-/- animals, indicating that leukocyte-derived NADPH oxidase inhibit PAI-1 activity in this model of ventricular remodeling. Another determinant of ventricular remodeling is cardiac myocyte death. Several studies have identified the stromal cell-derived factor-1 [alpha]

(SDF-1[alpha])/CXC chemokine receptor 4 (CXCR4) axis to be important for the homing and survival of stem cells at the site of injury post-AMI. It has also been reported that Sdf-1-/- and Cxcr4-/- mice exhibit defects in hematopoesis, neurogenesis, vacsulogenesis, and ventricular septum formation and die within a few days of birth. Multiple reports claim that overexpression of SDF-1 at the infarcted region improves ventricular function by preservation of cardiac myocytes and increased vasculogenesis. Since the role of cardiac myocyte-derived CXCR4 was not well-defined in this context, we hypothesized that normal cardiac development is dependent on cardiac myocyte-derived CXCR4 expression. In the adult heart, SDF-1[alpha] and its receptor, CXCR4 do not get expressed at the same time. Post-AMI, there is a short and immediate period of SDF-1[alpha] expression, which is followed by CXCR4 expression in cardiac myocytes. Therefore, we postulated that cardiac myocyte-derived CXCR4 expression is not critical in adulthood post-AMI. To address these issues, we developed congenital and conditional cardiac myocyte-specific Cxcr4-/- mouse models. Our results demonstrate that congenital deletion of CXCR4 has no effect on cardiac function and septal defect. Furthermore, the ventricular function of conditionally deleted cardiac myocyte-specific CXCR4 was not significantly different from their WT littermates. Collectively, our observations suggest that leukocyte-generated NADPH oxidase-derived free radicals participate in ventricular rupture post-AMI by oxidizing PAI-1 and cardiac myocyte-derived CXCR4 has no major role in cardiogenesis during development and ventricular remodeling post-AMI due to mismatch in the timing of expression of SDF-1 and CXCR4 in the heart.
???????????????????????? ????????????????2008????????????????????????????????????
??? ????????????……
????????????????????????,?????18?????1947?????,?????????????
????????????????,?????????,????????????????????????????????,?????????.
Data Structures Using CAlgorithmsDesign and AnalysisData Mining and Data WarehousingLaxmi Publications, Ltd.Factors Affecting Ventricular Remodeling Post Myocardial Infarction
????????
????????????????????????:??,??????
??????????,?????????????
???Prentice Hall????????
Copyright: 010e01e1340709438b409781d89866ad