Research Interest

Dr. Baker's research program addresses three areas:

- Basic research on cardiovascular development and causes of cardiovascular diseases
- Translational Research to improve clinical outcomes following cardiac surgery
- Protection of the child's cardiovascular system against injury from radiation

Information gained from these research studies is providing needed insight to conduct translational research—the development of innovative approaches to the diagnosis, treatment and prevention of cardiovascular disease.

Dr. Baker's research program serves as a nexus to translate Basic Science discoveries into clinical applications at MCW and Children's Hospital of Wisconsin. Dr. Baker has developed and maintained strong collaborations between basic scientists and clinicians to translate laboratory findings made in his MCW-based space into clinical applications. He has achieved this through extensive interactions and collaborations with basic scientists and clinicians.

Protecting children's hearts during surgical repair of cyanotic congenital birth defects

Dr. Baker's research program continues to actively investigate the problem of protecting children's hearts during surgical repair of cyanotic congenital birth defects. Results of these studies will contribute to our understanding of how to improve protection of the child's heart during cardiac surgery and will improve the clinical management of children with cyanotic heart disease. Dr. Baker's research program is funded by an R01 grant from the National Institutes of Health.
Dr. Baker has also shown that erythropoietin, a drug used to treat anemia, protects the heart against injury from ischemia/reperfusion. In collaboration with his clinical colleagues Dr. Baker is conducting a clinical trial at Children’s Hospital of Wisconsin to demonstrate the ability of erythropoietin to protect the child’s heart during surgical repair of congenital heart defects. This project reflects Dr. Baker’s commitment to translational research at MCW.

### Protecting adult hearts against injury during a heart attack

Dr. Baker has shown that thrombopoietin, a naturally occurring protein being developed as a pharmaceutical to increase platelet count in cancer patients during chemotherapy, can also protect the heart against injury during a heart attack. Currently there are no therapies available to directly protect the heart against the damaging effects of a heart attack. Dr. Baker's team has shown that administering a single dose of thrombopoietin to rats during a heart attack decreased the extent of permanent muscle damage to the heart and increased the ability of the heart to function afterwards, when compared with no drug treatment. Additionally, Dr. Baker and his colleagues found that a single cardioprotective treatment with thrombopoietin did not increase platelet count. This novel finding suggests the cardioprotective actions of thrombopoietin are separate from its ability to increase platelet count. Dr. Baker has submitted a US and worldwide patent application on the tissue protective properties of thrombopoietin. Dr. Baker’s discovery is licensed to Cardiopoietis, a Wisconsin LLC, formed to develop drugs for the treatment of heart attacks.

### Radiation injury to the cardiovascular system

Dr. Baker's research program is actively investigating the effect of radiation on the cardiovascular system. There is an urgent need to understand the risk of injury to vital organs, such as the heart, following a radiological terrorist attack or nuclear accident, define the mechanisms underlying the injury, and devise treatment strategies. The extent to which exposure to 10 Gy total body irradiation (TBI), a potentially survivable dose in a radiation accident or radiological terrorism event results in injury to the cardiovascular system is unknown. Dr. Baker’s studies have shown that a single TBI exposure with a dose of 10 Gy results in a time dependent increase in serum total cholesterol, LDL cholesterol, and triglycerides, all of which are biomarkers for the increased risk for cardiovascular disease. Hypercholesterolemia is associated with morphological injury to the vascular endothelium resulting in stenosis, decreased density of the smaller diameter coronary vessels, and a decrease in ventricular function at 120 days following TBI, manifest functionally as a decline in global radial and circumferential strain. TBI decreased expression of eNOS and iNOS and increased expression of protease activated receptor-1 and fibrinogen in the first 120 days after TBI. Dr. Baker is supported by a R01 grant from the National Institute of Allergy and Infectious diseases to determine the impact of radiation on the heart.
Recent publications


