



Epic Signals: Unraveling the Communication Between Fat and Heart Tissue

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DESCRIPTION

For decades, adipose tissue was considered a passive energy reservoir, storing calories for later use. However, modern research has revealed that fat is a dynamic endocrine organ capable of active communication with multiple organ systems, including the heart. This inter-organ dialogue occurs via a complex network of hormones, cytokines, exosomes, and metabolites collectively known as adipokines. These signaling molecules influence cardiac structure, function, and disease susceptibility in ways that are only beginning to be understood. The concept of “epic signals” emphasizes that adipose tissue is not merely a bystander in cardiovascular health, but an active participant whose dysregulation can promote pathology. Recognizing and decoding this dialogue is rapidly emerging as a critical frontier in cardiovascular research.

Crosstalk between adipose and cardiac tissue

Among different fat depots, visceral fat exerts the most pronounced effects on the heart. Unlike subcutaneous fat, visceral fat releases a higher concentration of pro-inflammatory adipokines such as interleukin-6, tumor necrosis factor-alpha, and resistin. These molecules contribute to endothelial dysfunction, myocardial remodeling, and systemic inflammation, which in turn accelerate atherogenesis and impair cardiac contractility. Epicardial Adipose Tissue (EAT), located directly adjacent to the myocardium and coronary arteries, represents a specialized niche with powerful paracrine effects. Unlike distant fat depots, EAT communicates locally, releasing signaling molecules that modulate coronary artery tone, myocardial metabolism, and immune cell recruitment. Emerging evidence shows that changes in EAT volume or secretory profile correlate with arrhythmias, heart failure, and coronary artery disease, underscoring the clinical relevance of these epic signals.

Furthermore, the communication between fat and heart is bidirectional. The myocardium can secrete cardiokines and other molecular mediators that influence adipose tissue metabolism and inflammation. This feedback loop allows the heart to modulate its own microenvironment but also creates

vulnerability when signaling becomes dysregulated, as seen in obesity, diabetes, and metabolic syndrome. Animal models and human studies demonstrate that even subtle shifts in fat-derived signaling can precede overt cardiovascular disease, highlighting the potential of these molecules as early predictive markers. Understanding this crosstalk provides a more holistic view of cardiac physiology, linking metabolism, inflammation, and mechanical function in a unified framework.

Translating fat-heart signaling into clinical insight

The clinical implications of adipose-cardiac communication are profound, particularly for risk assessment, diagnostics, and therapy. Advances in imaging technology, including cardiac Magnetic Resonance Imaging (MRI) and Computed Tomography (CT), enable precise quantification of epicardial fat volume and density. These measurements serve as non-invasive biomarkers that reflect both metabolic status and localized cardiac risk. Complementing imaging, circulating adipokines such as leptin, adiponectin, and omentin provide molecular readouts of the fat-heart dialogue. When integrated with traditional cardiovascular risk factors, these biomarkers can improve patient stratification, identifying individuals at high risk who might otherwise appear healthy on standard evaluations.

Therapeutically, understanding epic signals opens the door to targeted interventions. Lifestyle modifications remain foundational: exercise, weight loss, and dietary changes can reduce visceral fat and shift adipokine profiles toward anti-inflammatory and cardioprotective patterns. Pharmacological strategies are increasingly informed by mechanistic insights into fat-heart crosstalk. Cutting-edge research explores exosome-mediated therapies, leveraging beneficial molecules secreted by adipose tissue to enhance myocardial repair and regeneration. These approaches exemplify a paradigm shift: rather than viewing fat solely as a cardiovascular threat, researchers and clinicians are beginning to harness its signaling potential as a therapeutic tool.

Despite these exciting developments, several challenges remain. Standardization of imaging protocols, validation of circulating

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biomarkers, and clarification of causal mechanisms are important for translating research findings into practice. Moreover, accessibility is a concern, particularly in low-resource settings where advanced imaging and molecular analyses may be limited. Interdisciplinary collaboration between cardiologists, endocrinologists, and metabolic researchers is essential to integrate this knowledge into effective prevention and treatment strategies. Additionally, public health initiatives aimed at obesity reduction gain renewed importance, as minimizing visceral fat may directly attenuate harmful cardiac signaling. The integration of epic signals into clinical decision-making promises more precise, individualized care, aligning interventions with each patient's unique cardiometabolic profile.

The discovery of epic signals highlights the intricate, active dialogue between adipose tissue and the heart. Far from being inert storage depots, fat cells act as communicators that influence cardiac function, disease progression, and therapeutic response. Decoding these signals is transforming our understanding of cardiovascular physiology and opening novel avenues for early diagnosis, risk prediction, and intervention. As standardization, technological accessibility, and clinical validation progress, incorporating adipose-cardiac communication into routine practice could become a cornerstone of precision cardiology. By embracing the complex interplay between fat and heart, researchers and clinicians have the opportunity to improve outcomes, personalize therapy.