Talk #1 (Basic): “Placental injury and adaptation: profiling an act of cell-defense”

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Intact placental function depends on villous trophoblast at the feto-maternal interface, which fulfills critical functions for embryonic development, including gas exchange, supply of nutrients and removal of waste products, endocrine regulation and immunological defense. Diverse physical, chemical and biological stressors adversely influence the homeostatic balance between trophoblast injury, adaptation and regeneration, resulting in placental dysfunction and substandard fetal growth. Our research focuses on understanding the molecular mechanisms that underlie trophoblast injury and adaptation to diverse insults. We postulate that placental injury, such as hypoperfusion and cellular hypoxia, impacts the expression of a unique set of trophoblast genes, resulting in regulation of proteins that either enhance trophoblast injury or attenuate the damage. We have applied novel analysis approaches to high-throughput oligonucleotide microarray screens to identify key players in trophoblast response to injury. Examples of such players include the lipid droplet protein adipophilin, NDRG-1 and PHLDA2. The functional analysis of these proteins may shed light on mechanisms of villous injury, and may provide new tools to identify and manage placental dysfunction and its consequences.