Evolution and the workaround

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Living things are resourceful, which is a comforting thought unless the living thing in question is a pathogen or a cancer cell. Noxious cells excel at developing drug resistance, outwitting immune systems, and evading cellular controls. They even show an unhealthy talent for surviving internal perturbations such as mutations that affect the function of vital genes, and they do this by evolving new mechanisms to perform old tasks. Somehow the bad guys find a workaround.

That observation led Norman Pavelka, Giulia Rancati, and Rong Li, researchers at the Stowers Institute for Medical Research in Kansas City, MO, to step back and consider the basic process by which cells adapt to the loss of seemingly irreplaceable genes. The researchers reasoned that understanding how cells adapt to internal perturbations could offer insight into how pathogens and cancer cells mutate to evade the body's defenses and resist treatment with drugs.

The Stowers researchers used the benign budding yeast Saccharomyces cerevisiae as their model organism and deleted a key cell division gene called MYO1. Surely, eliminating this important gene would shut down cell division. This seemed to be the case in the beginning, and yet as the MYO1 defective cells were cultured and subjected to consecutive rounds of selection for best growers, the yeast came up with new strategies to carry out division. When the researchers analyzed the genetic content of these evolved strains, they found that those who were best at cell division had accumulated multiple copies of many of their chromosomes.

Intriguingly, cancer cells also accumulate extra chromosomes as they

become more aggressive. The theory is that these extra chromosomes provide “backup” copies of important genes, allowing the original copies to mutate in ways that help the cells survive stresses (such as drugs) that are meant to kill them.

The observation that both yeast and cancer cells evolve chromosome duplications to work around lethal stresses suggests that drugs aimed at defeating this process might be particularly effective against pathogens and cancers adept at rapid drug resistance, the researchers say.

To stay alive, you have to be both sturdy and flexible. The Stowers researchers look to these evolved yeast strains for future explanations of how the duplication of genetic information contributes to the robustness and adaptability of all living things.

Source: American Society for Cell Biology