Developing Anti-cancer Drugs by Blockade of IP6-HSP90 Binding

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Technology description

Value Proposition:
A cellular enzyme known as inositol pyrophosphate 6 kinase 2 (IP6K2) has an important role in programmed cell death (apoptosis). JHU scientists discovered an interaction between IP6K2 and a protein known as Heat Shock Protein-90 (HSP90). Binding of IP6K2 with HSP90 inhibits the activity of IP6K2 and prevents cell death. JHU scientists discovered that known chemotherapy drugs can block binding of IP6K2 and HSP90 to allow apoptosis.
Technical Details:
The goals of cancer treatment are to reduce tumor cell growth and to kill existing cancerous cells. Some chemotherapy agents work with normal cellular metabolism to induce cell death. While effective, many chemotherapy drugs have serious and cytotoxic side effects which are significantly debilitating to the cancer patient. These side effects are often caused by collateral damage to DNA or inhibition of key protein activity in non-cancerous cells. There is a large unmet need to identify effective chemotherapy compounds with fewer side effects. JHU scientists have identified a previously unknown cellular protein-protein interaction that regulates cell death (apoptosis). JHU scientists have shown for the first time that known chemotherapy agents can block protein binding to induce apoptosis. Other agents may effectively promote cell death of tumor cells in this manner without side effects of known chemotherapeutics.

Advantages

• Newly discovered apoptosis mechanism allows development of a screen for effective anti-cancer drugs that selectively block IP6K2- HSP90 binding, but not mechanisms that are associated with major cytotoxicity, to improve quality of life for cancer patients during chemotherapy treatment.
• Screening method based on standard methods of measuring protein- protein interactions allows easy adaptation to a high-throughput screen using automated technology for faster time to discovery and development of highly selective anti-cancer drugs.

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