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New myeloma-obesity research shows drugs can team with body's defenses

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Obesity increases the risk of myeloma, a cancer of plasma cells that accumulate inside the bones.

And with current obesity trends in the United States and especially in South Texas, that's ominous.

"I'm predicting an increase in multiple myeloma," said Edward Medina, M.D., Ph.D., "and with the obesity problems we see in the Hispanic population, there could be a serious health disparity on the horizon."

Dr. Medina, a hematopathologist and assistant professor in the Department of Pathology at The University of Texas Health Science Center at San Antonio, is looking at exactly how obesity causes an increased risk for myeloma.

What he and his colleagues have discovered is a potential way to not only boost the effectiveness of current chemotherapy treatments for myeloma, but at the same time a way to help the body help itself.

In a paper published this week in the journal *Leukemia*, Dr. Medina and his team look at an important little protein called adiponectin.

Myeloma is often called multiple myeloma because it occurs at many sites within the bone marrow. Healthy plasma cells produce antibodies that fight infection in the body, but myeloma cells produce high levels of abnormal antibodies that, when the cancer cells accumulate, they crowd

out production of other important blood cells, both red and white.

"They basically overtake the bone marrow," Dr. Medina said.

The disease can lead to bone pain and fragility, confusion, excessive thirst and kidney failure. While survival rates for patients with myeloma have increased in recent years, many people do not live more than five years beyond diagnosis.

Adiponectin is a protective protein that plays several roles in keeping the body healthy, including killing cancer cells. While adiponectin is produced by fat cells, Medina said, obese people have less of it. The reason for this paradox is that in cases of obesity, fat cells function abnormally, including producing less adiponectin. What they produce more of, however, are fatty acids, and it is likely that myeloma cells can feed on these fatty acids.

"Synthesizing fatty acids is important for myeloma cells to build vital structures, including cell membranes, that enable them to keep on growing," Medina said.

Focusing on adiponectin led Dr. Medina's lab to protein kinase A or "PKA"—a protein that, when activated by adiponectin, suppresses the fatty acids that myeloma cells need, leading to their demise.

The idea is to use the understanding of the pathways that adiponectin uses to kill myeloma cells to create a drug that would do the same thing.

"If we could pharmacologically suppress these fatty acid levels in obese myeloma patients, we could boost the effects of the chemotherapy that targets PKA or fatty acid synthesis, and potentially decrease the chemotherapeutic dose," Medina said. "Also, it would give your own body's protective measures more of a chance to work against the

cancer."

Provided by University of Texas Health Science Center at San Antonio