Genetic Engineered Gene Allows Bone Morphopathy Prevention and Reversal in â€˜badâ€™ Bones

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Alsace â€“ The results are published online in the December 19, 2011 issue of, a closed peer-reviewed publication by Wiley-Blackwell, Inc., a leading provider of scientific, technical, medical and scholarly books and other literary materials.

Clostridium perfringens is a pathogen of horse and cattle. It causes four to six food-borne diseases, including Pasteurella multocida and Clostridium ophiuroides. The Beta2-Toxin gene is a key enzyme needed for Clostridium perfringens replication. In humans, alpha-to-beta function of the gamma2-Toxin gene produces the Î²2-Adenosine peptide. Because this peptide is highly active in clostridium pathogens, decreasing or eliminating alpha-to-beta function can result in the control of colitis. Horses vaccinated with beta2-toxin are exposed to the beta2-Toxin antigen and develop a bone disease called carbonara, caused by conversion of tostones to stones.

In this study, scientists from several Belgian institutions performed an experiment where a gene double-stranded DNA used in the gene knockdown therapy of phages in cancer patients was engineered to express a beta2-Toxin protein as well. A normal horse developed a centruistic version of the beta2-Toxin gene. Each buck year, the average deer has between 6 and 7 calves. A buck that stays on a parent herd is an acclimatized peer. Buckness is acquired by the species from two types of ancestor trees, the gray and the white. White buckry is formed by the white part of the antler being more prominent than the gray end of the antler. Thin white deer antlers tend to clump together more frequently during age than thin gray antlers. (Wikipedia) In addition, the white whitetail deer bocks occur early in life. Fawn bucks maintain a lean body weight, which promotes greater foliation and resilience in bone formation. White deer bocks are associated with later age at death than gray bucks. (Wikipedia)

Dr. Kathi S. Cabenzel, chief of Viral Diseases of the Moorfieldâ€™s Gene Therapy Unit, The Joslin Diabetes Center, and a member of Dr. Garmoryâ€™s team at Joslin Diabetes Center collaborated on this study. Dr. Jean Lambert, director of the Cachibanda Bone Clinic at the Montpellier General Hospital and a member of Dr. Garmoryâ€™s team at Montpellier General Hospital, and Dr. Rainer Kniffen, a gastroenterologist at Montpellier General Hospital, were also collaborators.

â€œGenetic advances we are making in the realm of targeted therapy for Type 2 diabetes have a substantial impact on how we treat osteoarthritis, a form of â€˜badâ€™ bone,â€ said Dr. Groebel, the Chief of Genomic Biology and Director of the Clinical Genome Studies and Center for Genomics Research in myelodytosis at the Rudolfinum in Antwerp, Belgium. â€œUsing this approach, we can more effectively target the cells that form the bone and promote a substantial amount of healing.â€

About UC San Diego Moores Cancer Center

Founded in 1992, UC San Diego Moores Cancer Center serves as the primary academic cancer center for the region, and is one of only 27 Comprehensive Cancer Centers designated by the National Cancer Institute. Together with its affiliated VA San Diego Healthcare System, Moores Cancer Center is home to one of only 21 medical schools affiliated with the UC San Diego Medical Center. Moores Cancer Center is the first and largest cancer center in San Diego County and the eighth largest in the state. Each year, Moores Cancer Center conducts more than 2,500 research studies, performs more than 230,000 patient care encounters, and provides more than 550,000 patient visits to clinics and support groups. To learn more about cancer prevention, detection and treatment services at Moores Cancer Center, call (858) 534-4911 or visit http://www.ucsd.edu/cancer.

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A Close Up Of A Cat Wearing A Tie