My project is a representation of corticosteroid induced osteoporosis. The overall object of this project is to go over how the stages of bones development and repair can be disrupted using corticosteroids. My diagram shows the bone markings of two different types of bones. One is a healthy bone before corticosteroid use, this bone shows the periosteum (Red Fruit by the Foot), which covers the outer surface of the bone and contains blood vessels, nerves, and lymphatic vessels that nourish compact bone. Articular cartilage (Blue Fruit by the Foot), which covers where the epiphyses meets other bones to form joints; helping to reduce friction and shock. Compact bone (White Chocolate) found under the periosteum, is known for being the dense to provide protection and strength to bones. Spongy bone (Rice Krispies), also known as cancellous bone, containing osteocytes housed in a network of matrix spikes called trabeculae. And an Epiphyseal line (White Chocolate), (former growth plate), an osseous tissue that replaced cartilage once the bone stops growing. The second diagram depicts a bone with osteoporosis. In this diagram you can see the difference as the compact bone has become much thinner and less trabeculae in cancellous bone making the appearance of larger holes.

Corticosteroids are a type of anti-inflammatory drug that can being taken orally or intramuscular. This steroid is used to treat autoimmune disorders, allergies, asthma, adrenal insufficiency, and many other conditions. One dangerous side effect of prolonged use is osteoporosis. Osteoporosis is a disease characterized by a decrease in bone mass that occurs when the rate of bone resorption exceeds the rate of bone formation. While osteoporosis can involve any bone, it most commonly affects the proximal ends of the femur, vertebrae, and wrist. With the loss of bone density, the osseous tissue may not provide enough support for everyday functions causing increased risk for fractures.

Corticosteroids can decrease calcium and vitamin D reabsorption from renal tubules, which increases the renal excretion of calcium. The release of excess calcium, hypercalciuria, is a related maker of bone metabolism during treatment with corticosteroids. Calcium is a critical component of bone health, especially in the form of calcium phosphate and calcium carbonate. Vitamin D, is another critical component, needed to accompany calcium to be absorbed by the small intestine. Without enough calcium circulating in the blood, Calcitonin cannot be activated to inhibit osteoclast activity and stimulate calcium uptake by the bones. Use of these steroids are also known to decrease the release of sex hormones such as estrogen and testosterone that are known for promoting osteoblastic activity and production of bone matrix.

In bone remodeling there are four phases, beginning with the resting stage where preosteoclasts become attracted to a remodeling site and fuse to form osteoclasts. In the resorption phase, osteoclasts resorb bone matrix and calcium is released into the blood for use. After the osteoclasts are removed there is a transition phase when mesenchymal stem cells appear along the resorption pit, where they can increase in numbers and mature into osteoblasts. The final phase of bone formation, osteoblasts release osteoid at the site to form a new soft nonmineralized matrix. The new matrix is later mineralized with calcium and phosphorus and is continuously packed forming new bone, replacing the older, less mineralized bone.

Glucocorticoid, a type of corticosteroid, can induce osteoporosis and is the most common form of secondary osteoporosis. These steroids are known to inhibit bone remodeling and increase risk of fractures. Glucocorticoid changes the balance between osteoclast and osteoblast activity in bone remodeling. This steroid stimulates osteoclast to resorb bone and reduces osteoblast mediated bone formation, resulting in increased overall bone resorption. The two main effects of Glucocorticoid on bone metabolism is, inducing apoptosis in osteoblasts and osteocytes thereby decreasing bone formation. As well as, prolonging the lifespan of osteoclasts and increasing bone resorption, later leading to osteoporosis.

Citations:

Duzen, O., Erkoc, R., Begenik, H., Soyoral, Y. U., & Aldemir, M. N. (2012). The Course of Hypercalciuria and Related Markers of Bone Metabolism Parameters Associated with Corticosteroid Treatment. *Renal Failure,* *34*(3), 338-342. doi:10.3109/0886022x.2011.648596

Adami, G., Rahn, E. J., & Saag, K. G. (2019). Glucocorticoid-induced osteoporosis: From clinical trials to clinical practice. *Therapeutic Advances in Musculoskeletal Disease,* *11*. doi:10.1177/1759720x19876468

Briot, K., & Roux, C. (2015). Glucocorticoid-induced osteoporosis. *RMD Open,* *1*(1). doi:10.1136/rmdopen-2014-000014

Betts, J. G. (2013). *Anatomy & physiology*. Houston, TX: OpenStax College, Rice University.