In 1960, the election of John F. Kennedy at the age of 43 made him the youngest president to serve in American history. While Kennedy was a renowned public figure, famous for his political career both in the Oval Office and in the United States Congress, many details of his medical history were kept hidden from the public.

John Fitzgerald Kennedy was born in 1917 to a prominent political family and grew up in Massachusetts the second oldest of nine children. His family placed great value on the athletic, academic, and professional achievements of their children, with Kennedy's brothers Robert and Ted also going on to have careers as politicians. Throughout his political career, John F. Kennedy maintained a carefully curated reputation of charisma, success, and fitness in the public eye. His persona as a strong, youthful candidate with a history of heroism during World War II and a love of football and other sports helped bolster this image during his political campaigns. He even used his supposed excellent health as a talking point in the 1960 Democratic primaries against Lyndon B. Johnson, who had suffered a near-fatal heart attack five years previously. In reality, Kennedy suffered from health complications that often left him weak or in pain. (Altman, 1992)

Around the age of 30, during his first term in Congress, Kennedy was diagnosed with a rare endocrine disorder called Addison's disease. In patients with Addison's disease, the adrenal glands do not produce enough steroid hormones, such as the glucocorticoid cortisol. This results in symptoms including muscle weakness, fatigue, weight loss, hypoglycemia and hypotension. Many patients also report gastrointestinal symptoms, such as nausea, abdominal pain and constipation. Hyperpigmentation, especially darkening of the lining of the mouth and gums, as well as darkening of the creases on the palms of the hands and nail beds is also common. (Hanley & Holt, 2011)

Throughout his political career, Kennedy's family and medical team carefully dismissed or outrightly denied allegations concerning his health. Approximately 20% of Addison's disease cases result from tuberculosis infection (Mandel, 2009). Kennedy's Addison's was not tuberculosis-induced, however, and this fact allowed him to skirt an admission of his diagnosis to the public. During his 1960 presidential campaign, Kennedy's opponents tried to use the rumors of his disease as political ammunition contesting his fitness for the role of president. His doctors responded by “[releasing] a cleverly worded statement saying that he did not have Addison's disease caused by tuberculosis,” and the matter was dropped (Mandel, 2009). His brother Robert Kennedy also stated that his brother “Does not now [have], nor has he ever had, an ailment described classically as Addison's disease.” Robert Kennedy's statement rested on a technicality. When the disease was first described in 1855 by its namesake Thomas Addison, it was “classically described” in its tuberculosis-induced form. John F. Kennedy's Addison's disease was attributed to unknown autoimmune causes, which did not fit this “classical” description (Mandel, 2009).

Kennedy suffered from chronic back pain that was multifactorial in origin. Publicly his recurrent back problems were attributed to being born with an unstable lower back joint, to a football injury and to his time in the navy (Pait & Dowdy, 2017). In addition, the corticosteroid medications that Kennedy took as treatment for his Addison's disease may have left his bones fragile and more prone to injury.
Kennedy underwent a well-documented back surgery in 1954 that put him on leave for eight months during his time as a Senator for Massachusetts. Kennedy’s back surgery was a risky procedure, as patients with Addison’s disease are at a heightened risk for complications during surgery and require additional hormone replacement during the process. This is due to cortisol’s critical role in the body’s stress response. Without the ability to increase the production and secretion of cortisol in response to stress, a severe injury, surgery, or infection can trigger a life-threatening condition known as an adrenal crisis. In an adrenal crisis, a critical lack of glucocorticoid hormones in the body results in dangerously low blood pressure and a loss of consciousness, requiring immediate emergency medical treatment (Hanley & Holt, 2011). While Kennedy did not suffer an adrenal crisis during his surgery, he faced post-operative complications caused by multiple instances of infection. During this time, on two occasions he was administered the Catholic Last Rites, reserved for individuals considered to be close to death (Pait & Dowdy, 2017; Altman, 1992). The infections were likely the result of immunosuppressant effects of the many corticosteroids he used to manage his condition.

On November 22, 1963, Kennedy was assassinated by gunshot while riding in an open-air vehicle in Dallas, Texas. The subsequent autopsy found that his adrenal glands had been destroyed by his autoimmune condition. Members of the family did not wish for his abdominal cavity to be examined during the autopsy, as it would have revealed the extent of his illness (Altman, 1992). Without corticosteroid treatment, Kennedy may have died many years previously. His Addison’s disease has been posthumously attributed to an autoimmune disease known as autoimmune polyendocrine syndrome type II (APS2), which also affected his thyroid (Mandel, 2009). It is unknown how his condition may have affected him, had he lived longer. At the time of his death, Kennedy was only 46 years old.

References

Questions
1. The word “glucocorticoid” is a portmanteau, or a word that combines the meanings of two or more words. What do we learn about cortisol by knowing that it is a glucocorticoid?

2. Diagram negative feedback regulation of cortisol in normal healthy person.

3. Define the terms “primary adrenal insufficiency” and “secondary adrenal insufficiency.”
4. Did John F. Kennedy have primary or secondary adrenal insufficiency? Use evidence from the text to support your answer.

5. Which hormones would you expect to have a low plasma concentration in a patient with Addison’s disease?


7. Why does Addison’s disease cause hyponatremia and hyperkalemia?

8. Why would a patient with Addison’s disease have low blood pressure?

9. Why would a patient with Addison’s disease have low blood sugar?

10. Diagram negative feedback regulation of the hypothalamic pituitary adrenal axis in a patient with primary adrenal insufficiency.

11. Explain how Addison’s disease (i.e., low plasma concentrations of cortisol) results in hyperpigmentation.

12. Why is it essential for a patient with Addison’s disease to receive treatment?

13. Toward the end of his life, Kennedy suffered from fragile bones and immune suppression. Why are these symptoms more consistent with the catabolic effects of excess cortisol?