

Endocrine and Autoimmune Aspects of the Health History of John F. Kennedy

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At the age of 43 years, John F. Kennedy was the youngest man ever elected president. Throughout both his campaign and his presidency, he was portrayed as the epitome of youth and vigor. In fact, he had the most complex medical history of anyone to occupy the White House. The recent opening of his White House medical records has provided researchers greater insight into the multiple medical conditions that afflicted Kennedy. A recent review of these

records, coupled with other available sources, allows new understanding of his health history that can now be explained in the context of a unifying autoimmune endocrine disorder.

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At 43 years of age, John Fitzgerald Kennedy was the youngest man ever elected to the presidency of the United States. To all appearances, he was the picture of health and vitality. In actuality, he had the most complex health history of anyone to occupy the White House. In an era of less media scrutiny, he was able to conceal the true nature of his complex medical problems throughout his presidency. After years of requests by researchers, Kennedy's White House medical records were first made available by the John F. Kennedy Presidential Library & Museum to historian Robert Dallek and his medical consultant, Dr. Jeffrey Kelman, in 2002. Since then, other teams of researchers have examined Kennedy's medical records. I was allowed to review President Kennedy's medical records on 3 occasions in May and July of 2008, as well as the correspondence between various specialists at the Mayo Clinic and Lahey Clinic and Kennedy's father, Ambassador Joseph P. Kennedy, concerning the young John F. Kennedy. The library's medical records consist almost entirely of the medical files of White House physicians Janet Travell and George Burkley, as well as correspondence from various specialists, hospital discharge summaries, and radiography results. None of the medical files of any of the medical specialists involved in Kennedy's health care are at the John F. Kennedy Presidential Library & Museum. Review of the White House medical files revealed that Kennedy's major medical diagnoses can be explained on an endocrine autoimmune basis—namely, the autoimmune polyendocrine syndrome type 2 (APS 2).

The concept of a polyendocrine syndrome was first suggested in 1849 by Thomas Addison, when he described a patient with adrenocortical failure and probable pernicious anemia (1). In the early 20th century, Parkinson described the association of diabetes and pernicious anemia

(2), while Schmidt described the occurrence of lymphocytic infiltrates of the thyroid and adrenal glands in 2 patients dying of Addisonian crisis (3). Roitt and colleagues identified autoantibodies to thyroglobulin in patients with Hashimoto thyroiditis in 1956 (4), and the identification of autoantibodies to islet cells, adrenal cortex, gastric parietal cells and steroid-producing gonadal cells followed (5). In 1980, Neufeld and colleagues characterized the autoimmune polyglandular syndromes into 2 distinct syndromes, APS types 1 and 2 (6, 7).

Autoimmune polyendocrine syndrome type 2 is defined by the coexistence of autoimmune adrenocortical insufficiency and evidence of adrenalitis with either autoimmune thyroid disease or type 1 diabetes mellitus. Adrenal failure or autoantibodies plus autoimmune thyroiditis is also termed *the Schmidt syndrome* (8).

In their 1976 book, *The Search for JFK*, authors Joan and Clay Blair disclosed for the first time what had long been suspected—that Kennedy unequivocally had Addison disease, defined as adrenocortical insufficiency (9). In September 1947, Kennedy, then a Congressman from Massachusetts, collapsed during a visit to England. Sir Daniel Davis, the admitting physician, diagnosed adrenal crisis and told Kennedy's friend, Pamela Churchill, "That young American friend of yours, he hasn't got a year to live" (10, 11). After he was treated, Kennedy sailed back to New York accompanied by a nurse and was then admitted to the Lahey Clinic in Boston. The cover story provided to his constituents stated that he had a severe recurrence of the malaria he had contracted in the Pacific during World War II.

Endocrinologist Elmer C. Bartels cared for Kennedy at the Lahey Clinic and remained his endocrinologist for several years after. He told the Blairs in 1973 that after returning from England, Kennedy was treated with desoxycorticosterone acetate (DOCA)—an early, weak synthetic adrenal hormone—that was implanted in pellet form under the skin of his thigh every 3 months. By 1950, cortisone was available in oral form, and Kennedy was taking 25 mg daily in addition to his DOCA implants.

See also:

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Although Kennedy was not formally diagnosed as having Addison disease until September 1947, earlier manifestations of adrenal deterioration may have occurred. When he announced his candidacy for Congress in 1945, Kennedy was a very frail-looking 150 lb (he was 6 feet tall). As the campaign progressed, he seemed tired, hollow-eyed, and anemic-looking to his staff workers (12). The day before the primary, Kennedy marched in the annual Bunker Hill parade on 17 June 1946. At the end of the 5-mile parade through Charlestown, Massachusetts, on that hot and sunny day, Kennedy collapsed. Aides claimed that he turned “yellow and blue . . . He appeared to me as a man who probably had a heart attack” (12).

Even earlier, in August 1940, Dr. Paul O’Leary of the Mayo Clinic described his evaluation of Kennedy’s health to Kennedy’s father in a letter (13):

Jack’s examination revealed a rather unique finding, which he perhaps discussed with you by this time. His blood pressure was exceedingly low, being in the neighborhood of 85, and for a boy of this age it should be approximately 120. This so-called static hypotension is a comparatively recently recognized entity and it might well be that some of Jack’s gastrointestinal difficulties are attributable to this finding.

In 1954, despite his adrenal disease, Kennedy had back surgery in New York. A 1955 article in the *Archives of Surgery* titled “Management of Adrenocortical Insufficiency During Surgery” described his case, although Kennedy was identified only as “Case 3” (14). From that point on, the Chief of Endocrinology at New York Hospital, Dr. Ephraim Shorr, managed Kennedy’s condition. After Shorr’s death in 1956, his associate, Dr. Eugene J. Cohen, directed Kennedy’s endocrinologic management.

During the 1960 campaign for the presidency, Kennedy’s political enemies charged that the senator had Addison disease, and the coverup of this diagnosis, orchestrated by Dr. Janet Travell, has been well documented (15–20). The crux of the cover-up rested on the cleverly worded statement claiming that Kennedy “does not now nor has he ever had an ailment described classically as Addison’s disease, which is a tuberculous [sic] destruction of the adrenal gland” (17). In fact, Addison disease has an autoimmune cause in nearly 80% of cases and tuberculosis accounts for only 10% (21). This narrow definition of Addison disease was successful in deflecting further probes.

In the oral history given by Travell in 1966, she described that Kennedy was diagnosed as having hypothyroidism when he was hospitalized at New York Hospital in May 1955 (22). With the consent of Shorr as the consulting endocrinologist, Kennedy began treatment with liothyronine. The discharge summary from New York Hospital for Kennedy’s hospitalization from 26 May 1955 to 2 June 1955 contains record of basal metabolic testing, namely a basal metabolic rate of -15 , consistent with mild hypothy-

roidism; of note, liothyronine is not listed as one of the discharge medications (23). No further thyroid diagnostic testing results are contained in the medical records kept by either Dr. Travell or by Dr. Burkley, Travell’s successor as White House physician (24). The White House medical records indicate that Kennedy was taking liothyronine, 25 μg twice daily, throughout his presidency.

Thyroid gland function is mentioned in 2 pieces of correspondence in Joseph P. Kennedy’s files. In a letter dated 16 December 1935, Dr. William P. Murphy wrote to Joseph P. Kennedy after John F. Kennedy was hospitalized at the Peter Bent Brigham Hospital, that, “The basal metabolic rate was minus 11 per cent which is just below the lower border of normal.” In his conclusions, Murphy wrote, “Of these various studies, then, the relatively low basal metabolic rate may be of importance . . .” (25). In a contrasting interpretation, Dr. Walter C. Alvarez of the Mayo Clinic wrote to Joseph Kennedy on 9 February 1939 after John F. Kennedy had had an evaluation, “Because a common cause of somnolence is a low rate of activity in the thyroid gland, we checked his basal metabolic rate. It was -11 , which is well within normal limits” (26).

The fact that Kennedy, who unequivocally had Addison disease, also had hypothyroidism leads to the plausible conclusion that there was an autoimmune basis for his medical problems, and APS 2 explains these conditions. In 50% of patients with this syndrome, adrenocortical failure is the initial endocrine abnormality (27). Autoimmune thyroid disease is found to coexist with Addison disease in two thirds of cases (27, 28). Typically, APS 2 occurs in early adulthood, with a peak onset at 30 years of age (29), the exact age that John F. Kennedy was when his Addison disease was diagnosed.

Adrenal cell autoantibodies (ACAs) were first reported in 1957; their detection involved an assay that used complement fixation techniques (30). In 1963, the assay for ACAs was improved using an indirect immunofluorescence test. Immunofluorescence testing found ACAs in 40% to 90% of patients with idiopathic Addison disease (30). In 1992, testing for antibodies to the enzyme steroid 21-hydroxylase by using radiobinding assays became the gold standard for detecting autoimmune Addison disease. Antibodies to 21-hydroxylase were found in 80% to 90% of patients with idiopathic Addison disease in almost all participants who had had disease for less than 15 years and in 60% of participants who had had disease for more than 15 years (30). Whether Kennedy was tested for ACAs is not known because the office records of Dr. Eugene Cohen, Kennedy’s endocrinologist and most important physician, are not available to researchers.

Kennedy’s Addison disease was clearly not due to tuberculous destruction; rather, it was originally described as “idiopathic” Addison disease, or adrenal atrophy. Although no comments were made on his adrenal glands in the official autopsy, in 1992, one of the pathology residents present at the autopsy confirmed that the autopsy team

found almost no adrenal tissue, consistent with adrenal atrophy (31). No scarring, inflammation, or granuloma formation was detected. As pathologist George Lundberg stated, this observation is “diagnostic of severe Addison’s disease, probably idiopathic, almost certainly *not* of tuberculous origin” (32).

Although the presence of 2 diseases that are commonly of autoimmune origin make it probable that Kennedy had APS 2, the presence of confirmatory antibodies is essential to fulfill the diagnostic criteria (28, 30). The inheritance pattern in APS 2 is polygenic. In a recent study of a large cohort of patients with Addison disease in Norway, the frequency of HLA class 2 haplotypes did not significantly differ in patients with isolated Addison disease or those with APS 2. As noted in previous studies, the researchers found an increase of the HLA DR3-DQ2 and HLA DR4-DQ8 haplotypes in Addison disease and APS 2 compared with healthy control persons (33). Approximately one half of patients with APS 2 have relatives with autoimmune diseases (28). It is public knowledge that Kennedy’s younger sister, Eunice, has Addison disease (34) and that his son, John F. Kennedy, Jr., had Graves disease.

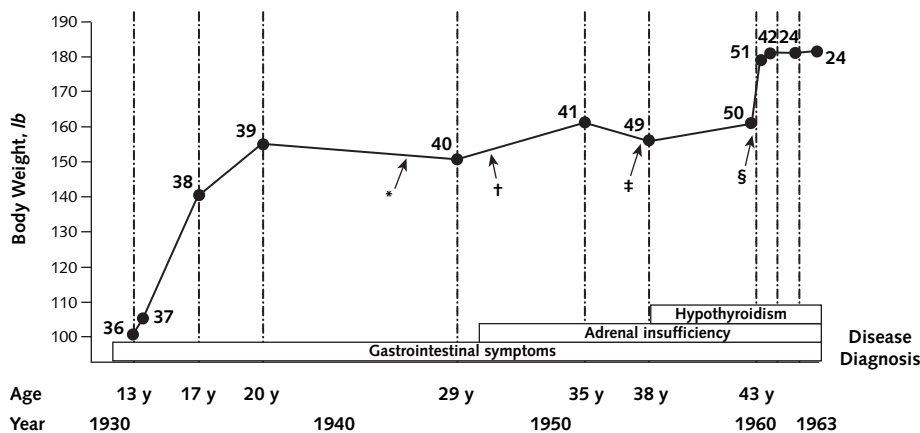
Other autoimmune conditions are associated with APS 2. These include chronic atrophic gastritis (with or without pernicious anemia) in 4.5% to 11% of cases, and hypergonadotropic hypogonadism in 4% to 9% of cases (28). In addition, the incidence of celiac disease is much higher in patients with autoimmune adrenal and thyroid disease (35), an interesting fact given that Kennedy had gastrointestinal symptoms (cramping, diarrhea, inability to gain weight) for most of his life (Figure).

Janet Travell’s listing of Kennedy’s medications for 12 October 1961 represented a typical day for the president. He was taking ascorbic acid, 500 mg twice daily; hydrocortisone, 10 mg daily; prednisone, 2.5 mg twice daily; methyltestosterone, 10 mg/d; liothyronine sodium, 25 µg twice daily; fludrocortisone, 0.1 mg/d; and diphenoxylate hydrochloride and atropine sulfate, 2 tablets as needed (43). Records at the John F. Kennedy Presidential Library & Museum do not contain the results of any of Kennedy’s endocrine testing.

Kennedy received testosterone daily during his entire presidency. His daily dose of oral methyltestosterone started out as 10 mg, and on several occasions it was increased to 25 mg/d. The summary medication sheet for May 1962 indicates that his physicians began trying different testosterone preparations at that time (44). From 1 to 7 May, he received between 50 mg and 75 mg of testosterone aqueous suspension for 4 days, and from 10 to 28 May, he received either 5 mg or 10 mg of oral fluoxymesterone.

As noted earlier, APS 2 is associated with hypogonadism as a result of the development of steroid-cell autoantibodies that react with Leydig cells of the testes, as well as thecal cells of the ovary (30). Kennedy’s requirement for testosterone can be ascribed to autoimmune hypergonadotropic hypogonadism as a result of APS 2. However, whereas the incidence of ovarian failure in women with APS 2 is 10%, progression to gonadal failure is very rare among men with Addison disease, even in the presence of high-risk steroidal cell autoantibodies (8). Kennedy fathered 4 children from 1956 to 1960 and hence was apparently not hypogonadal. Another diagnostic possibility

Figure. John F. Kennedy’s medical profile.



The time of diagnosis of gastrointestinal symptoms, adrenal insufficiency, and hypothyroidism are plotted against Kennedy’s body weight at various ages. Arabic numbers at each solid circle are the references from which body weight information was obtained.

* Sinking of PT-109, August 1943.

† Addison disease diagnosed, September 1947.

‡ Unsuccessful back surgery, October 1954.

§ Suspected initiation of testosterone therapy, July to August 1960.

to consider is autoimmune (or lymphocytic) hypophysitis, an autoimmune syndrome that results in a picture of pan-hypopituitarism. Since 1962, 460 cases have been described in the literature (45). This all leads to conjecture as to when Kennedy started testosterone therapy.

Given Kennedy's history, his long-term steroid therapy could have affected his testosterone levels. This is very likely the case, as the prevalence of low testosterone levels in glucocorticoid-treated men is high because of glucocorticoid-induced suppression of all components of the hypothalamic–pituitary–gonadal axis. Typically, daily administration of more than 5 mg to 7.5 mg of prednisone or its equivalent increases the risk for gonadotropin and testosterone suppression and for alterations in muscle and bone mass (46). The medical records contain several notes indicating that Kennedy requested and received additional testosterone, such as on 12 July 1961, when Dr. Travell recorded, “methyl T 10 mg (by request)” (47).

In addition, Robert Dallek and his associate Dr. Jeffrey Kelman have stated that Kennedy was taking testosterone to keep his weight up (48). This, too, is highly likely. On the basis of available records, it cannot be determined exactly when Kennedy began testosterone therapy. Janet Travell recalls that Kennedy weighed 155 lb when she met him in May 1955 (49). Kennedy's White House medical records indicate that his daily weight was between 170 and 180 lb (24). There are possible clues as to when his testosterone therapy was initiated. One clue was provided by his lifelong friend Lem Billings, who stated that Kennedy's weight had surged during the 1960 presidential campaign: “If you look at pictures of him in July 1960 and then look at pictures of him taken in January of 1961, you'll find he was much heavier.” The total weight gain, Billings noted, was about 15 lb (50). In her autobiography, Travell included a photograph that she took of president-elect Kennedy on 15 November 1960. He is bare-chested and is leaping to catch a football. He appears to be trim, muscular, and well proportioned. This is confirmed by the observation of Rose Kennedy, who stated in a diary entry on 3 November 1960 that “Jack looks unusually well. His cheeks have filled out amazingly since I saw him in June. He has lost that lean Lincolnesque look which I secretly like better” (51).

In her autobiography, Travell described Kennedy the first time she met him: “In spite of his Florida suntan, I thought he appeared pale and anemic, and that indeed proved to be the case” (49). In her 1966 oral history, she embellished on this when asked by the interviewer if then-senator Kennedy had any other medical problems. “Well, of course when I first saw him he was extremely anemic. He was really anemic. He had impaired vibration sense which is indicative of peripheral neuritis . . . characteristic of a vitamin B₁ deficiency.” She also stated, “Senator Kennedy was put on a course of vitamin B₁₂, vitamin B₁ and B-complex injections. His blood count—his hemoglobin and red cells—did respond” (52).

Although the White House medical records contain no mention of peripheral neuropathy, they indicate that Kennedy had received regular vitamin injections, including vitamin B₁₂, throughout his presidency (24). It is unlikely that Kennedy was thiamine deficient, and anemia is not associated with thiamine deficiency. Travell's description is more consistent with vitamin B₁₂ deficiency. The coexistence of pernicious anemia is consistent with a diagnosis of APS 2: Pernicious anemia is found in 2% to 25% of patients with APS 2 (27).

As tempting as it is to link this finding to APS 2, Travell's description of Kennedy's anemia is problematic. She described her first meeting with him as having taken place on 26 May 1955. Later that day, she had admitted him to New York Hospital, where he remained until 2 June. His discharge summary for that hospitalization does not list a diagnosis of anemia, and it records a hemoglobin level of 15.5 g/dL and a hematocrit of 41% (23). He was clearly not anemic at that time. None of the complete blood counts that are in the medical files at the John F. Kennedy Presidential Library & Museum, covering 1961 to 1963, reveal anemia of any type.

In summary, John F. Kennedy had many medical conditions during his lifetime. Addison disease was diagnosed when Kennedy was 30 years of age, and he was found to have hypothyroidism when he was a senator. The coexistence of autoimmune adrenal disease and hypothyroidism is consistent with APS 2. Possibly because of autoimmune disease but probably as a result of long-term steroid replacement therapy, Kennedy's endocrinologist prescribed treatment with testosterone, and this may have been initiated during the 1960 presidential campaign. Despite his many medical conditions as well as his recurrent back problems, John F. Kennedy managed to convey an image of health and vigor that masked the true state of his health to the U.S. public.

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