

Benjamin Franklin's Risk Factors for Gout and Stones: From Genes and Diet to Possible Lead Poisoning

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Benjamin Franklin's medical history shows that he suffered from repeated attacks of gout and a large bladder stone. These conditions caused him considerable pain, markedly decreased his mobility, and likely contributed in indirect ways to his decline and eventual death from a pulmonary disorder. This article examines Franklin's risk factors for gout and stones, both as Franklin understood them and as we know them today. Significantly, both of these disorders are associated with high blood levels of uric acid, a metabolic by-product. Franklin's risk factors included his gender, genetics, diet, drinking, advanced age, psoriasis, and exposure to lead. Although it is impossible to assign a weight to each of these factors, it can be shown that a number of factors, each capable of raising uric acid levels, converged and conspired against him.

FOUR DAYS after Benjamin Franklin died on 17 April 1790, his attending physician, Dr. John Jones, provided the *Pennsylvania Gazette*, the newspaper Franklin once owned, with a brief account of his last illness.³ Readers were informed that “[a]bout sixteen days before his death, he was seized with a feverish indisposition, without any

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³J. Jones, “On the Final Illness and Passing of Benjamin Franklin,” *Pennsylvania Gazette* (1790).

particular symptoms attending it till the third or fourth day, when he complained of a pain in his left breast, which increased till it became extremely acute, attended with a cough and laborious breathing." Franklin had "no doubt his present afflictions were kindly intended to wean him from a world, in which he was no longer fit to act the part assigned to him."

The summary of Franklin's final moments continued:

In this frame of body and mind he continued till five days before his death, when his pain and difficulty breathing entirely left him, and his family were flattering themselves with the hopes of a recovery, when an imposthumation [abscess], which had formed itself in his lungs, suddenly burst, and discharged a great quantity of matter, which he continued to throw up while he had sufficient strength to do it, but, as that failed, the organs of respiration became gradually oppressed—a calm lethargic state succeeded—and on the 17th instant, about 11 o'clock at night, he quietly expired, closing a long and useful life of eighty-four years and three months.

Jones then added a few sentences about Franklin's history of "pleurisy"—a term then used to describe virtually all the respiratory infections, febrile or not, that were accompanied by chest pain (e.g., pneumonia was sometimes called "malignant pleurisy"). Franklin had had serious lung infections in the past. One, which has been noted by almost all of his biographers, because he stated in his *Autobiography* that it nearly carried him off, occurred when he was twenty-one. That was after he returned to Philadelphia from an ill-fated trip to England, where he had hoped to buy printing equipment to start a shop of his own. Eight years later, he experienced a recurrence of his pleurisy with an abscess on his left lung that suddenly ruptured and almost suffocated him. His illness forced him to miss meetings of his Library Company from June through September 1735, although he showed a partial recovery in July. Jones cited this illness, writing, "It may not be amiss to add to the above account, that Dr. Franklin, in the year 1735, had a severe pleurisy, which terminated in an abscess of the left lobe of his lungs, and he was then almost suffocated with the quantity and suddenness of the discharge. A second attack of a similar nature happened some years after this, from which he soon recovered, and did not appear to suffer any inconvenience in his respiration from these diseases."

Hence, Franklin's immediate cause of death was asphyxiation due to the release of purulent matter into his respiratory passages. This condition has always been a grave one, because it can set off a cascade of inflammation, fluid exudation into the airspaces, and a respiratory distress syndrome that is both mechanical and hormonal.⁴ Clearly,

⁴B. Hirshberg et al., "Factors Predicting Mortality of Patients with Lung Abscess," *Chest*

Franklin had, by 1790, become too frail to overcome the reactions that followed.

But what had made Franklin's once strong body so weak? Although this question has no simple answer, some of the possible factors can be gleaned from Jones's article in the *Pennsylvania Gazette*. To quote: "The stone, with which he had been afflicted for several years, had for the last twelve months confined him chiefly to his bed; and during the extreme painful paroxysms, he was obliged to take large doses of laudanum to mitigate his tortures." Thus, the effects of a large stone rendered Franklin bedridden. Further, it is possible he could have been further weakened by his pain-reducing medication. Laudanum is an opiate drug that, depending on its dosage and patient conditions, could cause nausea, vomiting, constipation, depressed respiration, and weight loss.⁵

In 1789, Franklin wrote to his French friend Le Veillard from Philadelphia, where he lay confined because of a large bladder stone, "I have a long time been afflicted with almost constant and grievous Pain, to combat which I have been obliged to have recourse to Opium, which indeed has afforded me some Ease from time to time, but then it has taken away my Appetite and so impeded my digestion that I am become totally emaciated, and little remains of me but a Skeleton covered with Skin."⁶

Thus, Franklin blamed his loss of appetite and declining body mass solely on the opiate medication he was taking for a large stone that he could not pass. Little was known about uremia, which also could have accounted for or contributed to his loss of appetite.

It is noteworthy that his stone had confined him to his bed for at least a year, and to his home for a longer period. The increasingly debilitated condition of his urinary system, made worse by the huge lodged stone, would have put him at risk for a host of complications, including pneumonia, given his history of repeated bouts of pleurisy.

Franklin's medical history further reveals that he was incapacitated by repeated attacks of gout. That is another painful condition that affected his ability to get around and exercise (he loved swimming), and at times effectively immobilized him.

In this article, we shall look at Franklin's history of stones and gout, two conditions that clearly tortured him and, even if only indirectly,

115 (1999); N. G. Mansharamana and H. Kozel, "Chronic Lung Sepsis: Lung Abscess, Bronchiectasis, and Empyema," *Current Opinion in Pulmonary Medicine* 9 (2003).

⁵W. L. Way, H. L. Fields, and M. A. Schumacher, "Opioid Analgesics & Antagonists," in *Basic & Clinical Pharmacology*, ed. B. G. Katzung (New York: Lange Medical Books/McGraw-Hill, 2001).

⁶A. H. Smyth, *The Writings of Benjamin Franklin: Collected and Edited with a Life and Introduction* (New York: Macmillan, 1907), 10:35. Hereafter this will be referred to as *WBF*.

helped weaken his aging body, even before he turned to pain-reducing laudanum, which could have made matters worse. Our main objective is not to trace the onset and progression of his gout and stones, since this has already been done.⁷ Rather, it is to consider his risk factors for these painful conditions and what he understood about them relative to what we know today.

We hope to show in some detail that many factors lined up to make Franklin a likely candidate for gout and stones, beyond being an overweight male. Each is capable of increasing serum uric acid levels—a key component in cases of kidney stones and gout—either by increasing uric acid synthesis or by decreasing its elimination by the kidneys.⁸ We wish to make it clear that we are not proposing that these conditions directly caused Franklin’s death. Most likely, they led to physiological complications in a body already in decline—an aged body likely suffering from impaired renal functions and other problems that were already signaling an end to his days on earth prior to his final battle with pleurisy.

THE CO-OCCURRENCE OF STONES AND GOUT

Rufus of Ephesus and Paul of Aegina, in the second and seventh centuries, noticed that gout and kidney stones often afflicted the same individuals.⁹ Early in Franklin’s own century, physician George Cheyne added that chalkstones taken from the joints of gouty individuals seemed identical to the gravel stones found in the bladders of these patients. “They both have the same colour, taste and smell, and produce the same internal texture of parts, as far as can be known, and even the same outward shape,” wrote the highly regarded Cheyne in 1724.¹⁰

Many twentieth-century investigators have looked at the co-occurrence of gout with kidney, bladder, and urinary tract stones. In one study, only 1 percent of 108,947 general admissions to a medical clinic had urinary stones. In contrast, 14 percent of 324 patients admitted for gout had a history of urinary calculi.¹¹

⁷S. Finger, *Doctor Franklin’s Medicine* (Philadelphia: University of Pennsylvania Press, 2006).

⁸“Uric acid” and “urate” are, for the purposes of this article, equivalent terms that will be used interchangeably.

⁹W.S.C. Copeman, *A Short History of the Gout and the Rheumatic Diseases* (Berkeley: University of California Press, 1964).

¹⁰G. Cheyne, *An Essay of the True Nature and Due Method of Treating the Gout*, 6th ed. (London: Strahan, 1824), 72.

¹¹W. E. Kittredge and R. Downs, “The Role of Gout in the Formation of Urinary Calculi,” *Journal of Urology* 67 (1952).

The association between these conditions is starker when one considers just those stones containing uric acid. An adult living in the United States twenty-five years ago had a 0.01 percent lifetime chance of developing uric acid stones. For primary and secondary gout patients, however, the reported association jumped to 23 and 42 percent, respectively, with serum uric acid levels highly correlated with the risk of stones.¹² Approximately 80 percent of the stones taken from gouty patients are composed entirely of uric acid, with the remainder being calcium oxalate or calcium phosphate, although these stones frequently have a uric acid core. In contrast, uric acid stones account for only 5 to 10 percent of all urinary stones analyzed in the United States and Western Europe.¹³

Physicians who have studied Franklin records have argued that it is highly probable that his large bladder stone and gout stemmed from chronic hyperuricemia.¹⁴ We contend that this provides the most parsimonious explanation for the co-occurrence of these two diseases in this Founding Father's case. Unfortunately, there are no surviving stones or pathological specimens from Franklin's body to assay, rendering it impossible to test this working hypothesis directly.

SOME MAJOR RISK FACTORS FOR FRANKLIN'S GOUT AND STONES

Genetics

Above and beyond the greater propensity for males to be affected by gout, a significant percentage of the close relatives of gout patients have "hereditary hyperuricemia," with estimates ranging from 18 to 40 percent.¹⁵ Geneticists have now begun to identify some of the genes that can cause hyperuricemia and gout, especially in early-onset cases.¹⁶

¹²T.-F. Yü, "Urolithiasis in Hyperuricemia and Gout," *Journal of Urology* 126 (1981).

¹³M. A. Becker and M. Jolly, "Clinical Gout and the Pathogenesis of Hyperuricemia," in *Arthritis and Allied Conditions*, ed. W. J. Koopman and L.W. Moreland (Philadelphia: Lippincott Williams & Wilkins, 2005), 2321.

¹⁴"Hyperuricemia" refers to the presence of excessive uric acid in the bloodstream. For an early but thorough discussion of this hypothesis, see G. W. Corner and W. E. Goodwin, "Benjamin Franklin's Bladder Stone," *Journal of the History of Medicine and Allied Sciences* 8 (1953).

¹⁵M. A. Becker, "Gout: Pathogenesis of Hyperuricemia," in *Primer on the Rheumatic Diseases*, ed. H. R. Schumacher Jr. (Atlanta: Arthritis Foundation, 1988), 195; Kittredge and Downs, "The Role of Gout in the Formation of Urinary Calculi"; Becker and Jolly, "Clinical Gout and the Pathogenesis of Hyperuricemia"; C. M. Wise, "Crystal-Associated Arthritis in the Elderly," *Clinics in Geriatric Medicine* 21 (2005).

¹⁶L. S.-C. Cheng et al., "Genomewide Scan for Gout in Taiwanese Aborigines Reveals Linkage to Chromosome 4q25," *American Journal of Human Genetics* 75 (2004); T. C. Hart et al., "Mutations of the UMOD Gene are Responsible for Medullary Cystic Kidney Disease 2 and Familial Juvenile Hyperuricaemic Nephropathy," *Journal of Medical Genetics* 39 (2002).

Franklin's papers do not suggest a family history of gout, but they do show that at least one of his relatives suffered from kidney stones. In 1744, Franklin composed a letter about bladder stones, which he sent to his parents in Boston, commenting on various treatments, including surgery, solvent injections, and heavy syrup ingestion.¹⁷ At the end of his letter, he asked his parents to share the information with "Br. John." Since he would go on to send John a flexible urinary catheter (below), it seems likely that the letter was intended for his brother, or perhaps for both his father and his brother, although this is uncertain.

We do know that eight years after this letter, Benjamin informed his brother John that he would do everything he could to help him deal with his painful stones.¹⁸ This was when he designed his flexible urinary catheter, informing his sibling that he "went immediately to the Silversmith's and gave Directions for making one (sitting by 'till it was finish'd), that it might be ready for this Post." Although flexible catheters had been made on a small scale in Europe, this might have been the first flexible urinary catheter made in the American colonies, and Franklin included specific instructions on how to insert the device in a way that he hoped would not be too painful.

Diet

Franklin enjoyed red meats and rich foods, and often indulged in them, particularly while in Europe, where he served his colony and later his new country. One gout historian has written, perhaps over-dramatically, that "the gluttonous and bibulous English gentry of the 18th and 19th centuries . . . had no idea of temperance. . . . A full service had 32 dishes, usually 16 of different meats and as many side dishes."¹⁹

This way of life was not foreign to Franklin. His alter ego, Poor Richard, had repeatedly preached moderation in his *Almanack*, and for the most part Franklin seemed to follow Poor Richard's advice—at least into middle age.²⁰

But his diet later in life, in Europe, would not be considered moderate by today's standards. In France, for example, a good meal for him might have included hors d'oeuvres, beef, mutton, veal, fish, cheeses, butter, pastries, bonbons, and other sweets. Furthermore, heavily salted meats were basic to his diet when he traveled, most notably on his sea voyages.

¹⁷L. W. Labaree, ed., *The Papers of Benjamin Franklin* (New Haven: Yale University Press, 1959–), 2 (1960): 413–14. Hereafter this will be referred to as *PBF*.

¹⁸*PBF*, 4 (1961): 385–87.

¹⁹E. Skovenborg, "Lead in Wine through the Ages," *Journal of Wine Research* 6 (1995): 59.

²⁰Finger, *Doctor Franklin's Medicine*, 27–36.

Like others in his century and even back into antiquity, Franklin knew that eating certain “rich” foods would increase his weight and invite attacks of gout. Specifically, he knew that physicians had long associated excessive amounts of red meat, offal foods, and shellfish with gout, long known as a rich man’s disease.²¹ By keeping careful notes, Franklin confirmed that certain foods worsened the pain he felt in his big toe and joints, and he also began to suspect that eating certain foods in large quantities might contribute to his stones. Consequently, he cut back on his rich foods in his later years, when he had more frequent gout attacks and when his stones caused him greater pain.

Gout sufferers are still advised to cut back on meat and fish, because purines from those sources are converted into uric acid.²² But unlike people today, Franklin apparently never wanted his gout completely cured. He reasoned, as did many physicians in the eighteenth century, that gout might somehow provide “protection” against more serious, potentially fatal illnesses.²³ Hence, his goal was only to “manage” his condition, and one way to do this without taking stomach-upsetting drugs, or worthless and possibly dangerous quack remedies, was to modify his diet.

Franklin would not have known about the role played by uric acid in these disorders. The association of gout with high levels of uric acid was not made until three years after his death. In 1793, Murray Forbes reasoned that, if urine contains uric acid, so must blood, and he further postulated that the precipitation of too much uric acid could produce the deposits capable of causing so much pain.²⁴ William Hyde Wollaston confirmed Forbes’s hypothesis in 1797.²⁵

Alcohol

Alcohol intake can be another significant risk factor. In fact, a low-purine diet may lead to only a modest reduction in serum uric acid levels, whereas eliminating alcohol can have more striking effects, at least for some individuals. Still, for many people now, as was true in Franklin’s eighteenth century, these two risk factors tend to occur together, with greater effects when combined than for a purine-rich diet or alcohol alone.

²¹R. Porter and G. Rousseau, *Gout: The Patrician Malady* (New Haven: Yale University Press, 1998).

²²Becker and Jolly, “Clinical Gout and the Pathogenesis of Hyperuricemia.” Purines are a result of the breakdown of DNA and are therefore present in all foods, but are more densely supplied by foods of animal origin.

²³Finger, *Doctor Franklin’s Medicine*, 276–93.

²⁴M. Forbes, *A Treatise upon Gravel and upon Gout* (London: Cadell, 1793).

²⁵W. H. Wollaston, “On Gouty and Urinary Concretions,” *Philosophical Transactions of the Royal Society* 87 (1797).

Alcohol can act in two distinct ways to raise serum urate levels. First, it inhibits renal urate secretion by competing for the same sites of elimination. Specifically, it triggers the production of lactic acid and beta-hydroxybutyrate, which in turn slow uric acid excretion by the kidney's proximal convoluted tubules. Second, even modest amounts of alcohol increase urate formation by stimulating the liver to break down adenosine triphosphate, a purine derivative, into free purines.²⁶

Franklin loved wine and in good humor wrote some drinking song lyrics to the then-popular melody of "Derry Down," probably in 1745.²⁷ That Noah's antediluvian predecessors died because they had only water to drink was one of his themes. Another was that these people became wicked because, in their sobriety, they could no longer find truth—*in vino veritas*. The Abbé Morellet, who had known Franklin since 1772 and was one of his drinking companions in France, reasoned in a song of his own that the visiting American's real motive for coming to France was to drink the wines.²⁸ The song was sung in Franklin's honor in 1779, and each stanza ended with the words "Glasses in hand, let us sing to Benjamin."

Franklin knew full well that overindulging in wine could be a risk factor for gout. In the 1734 edition of his *Poor Richard's Almanack*, he warned,

Be temperate in wine, in eating, girls, and sloth;
Or the Gout will seize you and plague you both.²⁹

He also knew that, once permitted into the body, the troublesome gout would almost assuredly return.

Nevertheless, Franklin continued to enjoy his wines and, as was true for his meats, he did not completely abstain when hobbled by the gout. Particularly telling are the notes of the Duc de Croÿ, who visited him in France in 1779, while he was recovering from a severe gout attack. He wrote that Franklin ate only one meal each day, but that it included "large slices of cold meat," and that he washed down his foods with "two or three bumpers of good wine."³⁰ For Franklin, who was known to drink far more on social occasions, this might have been a concession.

²⁶C. J. Eastmond et al., "Effects of Alcoholic Beverages on Urate Metabolism in Gout Sufferers," *British Journal of Rheumatology* 34 (1995); J. Faller and I. H. Fox, "Ethanol-Induced Hyperuricemia: Evidence for Increased Production by Activation of Adenine Nucleotide Turnover," *New England Journal of Medicine* 307 (1982).

²⁷E. R. Cohn, "Benjamin Franklin and Traditional Music," in *Reappraising Benjamin Franklin*, ed. J.A.L. Lemay, 304–05 (Newark, Del.: University of Delaware Press, 1993).

²⁸*Ibid.*, 312–15.

²⁹Labaree, ed., *PBF*, 1 (1959): 352.

³⁰Emmanuel, duc de Croÿ, "Mémoires du Duc de Croÿ sur les Cours de Louis XV et de Louis XVI (1727–1784)," *Nouvelle Revue Rétrospective* 5 (1889): 339.

Age

The age factor also looms large, because the glomerular filtration rate—an important index of kidney function—drops about 1 percent a year after age forty-five, and declines considerably more rapidly after age sixty-five.³¹ As a result, the kidneys become less efficient at clearing uric acid from the blood, increasing the chances of deposits in the joints. Statistics show that gout is most likely to appear in middle-aged men, and that attacks tend to become more pronounced in subsequent years.³² As for women, they tend to have significantly lower serum urate levels than men prior to menopause.

Franklin's medical history is typical of gout and stone sufferers in the eighteenth century. Although he might have had his first mild attack of gout in his forties, his first surviving letter mentioning his gout was sent to his sister Jane in 1762, when he was fifty-six. His attacks then became more severe, affecting his diplomatic objectives. One gout attack delayed his presentation of his credentials as minister plenipotentiary to Louis XVI of France. A note to the comte de Vergennes dated 14 March 1779 reads, "The Gout having again attacked me, and confined me to my Chair, I find I shall not be able to present myself at Versailles on Tuesday."³³ Three days later, he lamented with some irony, "I don't complain much even of the Gout, [but] there seems however some Incongruity in a *Pleni-potentiary* who can neither stand nor go."³⁴ The term plenipotentiary is derived from the Latin terms *plenus* and *potens*, and means "invested with full power," which obviously did not describe Franklin's physical condition.

As for urinary stones, the longer one lives, the more opportunity there would be for bothersome stones to form and grow. Further, such stones would become more problematic, in part because of the progressive bladder changes that characterize the elderly. These changes include volume shrinkage, voiding difficulties, and muscular weakening.³⁵

Franklin's earliest surviving statement about his stones dates from 1782, when he was seventy-six and working on the peace treaty between

³¹ J. W. Rowe et al., "The Effect of Age on Creatinine Clearance in Men: A Cross-Sectional and Longitudinal Study," *Journal of Gerontology* 31 (1976).

³² Wise, "Crystal-Associated Arthritis in the Elderly."

³³ B. B. Oberg, ed., *PBF*, 29 (1992): 121.

³⁴ *Ibid.*, 143.

³⁵ The prevalence and nature of age-related bladder changes are a matter of some debate. For some recent perspectives, see J. B. Jørgensen, K. M.-E. Jensen, and P. Mogensen, "Longitudinal Observations on Normal and Abnormal Voiding in Men over the Age of 50 Years," *British Journal of Urology* 72 (1993); S. Madersbacher et al., "The Aging Lower Urinary Tract: A Comparative Urodynamic Study of Men and Women," *Urology* 51 (1998); A. D. Rule et al., "Longitudinal Changes in Post-Void Residual and Voided Volume among Community Dwelling Men," *Journal of Urology* 174 (2005).

the new United States and Britain. He had passed some smaller gravel and sensed a larger bladder stone that he could not void. This stone, perhaps beginning in a kidney, and accompanied by smaller bladder stones, continued to grow and trigger inflammatory reactions over the next eight years. In 1787, two years after he returned to America, he wrote to a friend, "As to my malady, . . . I am sensible that it is grown heavier; but on the whole it does not give me more pain than when at Passy, and except in standing, walking, or making water, I am very little incommoded by it."³⁶ It has been suggested that Franklin's bladder stone was probably the size of a small pea when he first complained about it, and that it might have weighed 400 to 500 grams, occupying much of his bladder, when he died.³⁷

PSORIASIS AS A POTENTIAL RISK FACTOR?

The skin disorder that Franklin called the "Scurf" might also have contributed to his chronic hyperuricemia, and therefore to his gout and stones. Franklin's skin rash first appeared in 1774, when he was sixty-eight, and it tended to clear up only to reappear and abate again and again.

Franklin attempted to describe his skin condition to his physicians as objectively as possible. He wrote, "The Scurf appears to be compos'd of extremely thin Scales one upon another, which are white, and when rubb'd off dry, are light as Bran. When the Skin is clear'd in the Bath, it looks red, and seems a little elevated above the sound Skin that is around the Place, but it is not sore: . . . The fine Lamina seem to be formed one under another. . . . In rubbing them off they separate, like Talc, each having a Polish that shines."³⁸

A dermatologist who examined Franklin's notes opined that his scurf was probably some sort of psoriasis, and there seems to be general agreement with this assessment.³⁹ As for the relationship between psoriasis and elevated uric acid levels, this emerged in the second half of the twentieth century.⁴⁰ It is now estimated that at least 30 percent and perhaps as high as 50 percent of severe psoriasis sufferers have uric acid levels that exceed the upper limit of the normal range.

³⁶ Smyth, *WBF*, 9: 560.

³⁷ Corner and Goodwin, "Benjamin Franklin's Bladder Stone," 376.

³⁸ W. B. Willcox, ed., *PBF*, 25 (1986): 79–80.

³⁹ *Ibid.*, 77–78; J. V. Hirschmann, "Benjamin Franklin and Medicine," *Annals of Internal Medicine* 143.11 (2005); Finger, *Doctor Franklin's Medicine*.

⁴⁰ R. R. Baumann and O. F. Jillson, "Hyperuricemia and Psoriasis," *Journal of Investigative Dermatology* 36 (1961).

Psoriasis is increasingly considered as a possible cause of gout and stones, particularly in people with hyperuricemia, because the rapidly dying epidermal (skin) cells release large amounts of purines.⁴¹ The association between psoriasis and hyperuricemia, however, has not been found in every study. To some extent, the variable findings may reflect disease severity and individual differences.⁴²

In retrospect, Franklin's skin disorder could not have predisposed him for his initial attack of gout, because it started about twelve years after his first gout attack. Nevertheless, once he started having attacks of gout, the increase in uric acid caused by epithelial cell turnover could have exacerbated his gouty condition. It also could have increased the probability that calculi would develop and grow in his urinary system, which by this time might have been compromised by his exposure to another risk factor, namely, lead.

SUBTLE LEAD POISONING?

The case for Franklin's body being affected by lead poisoning is indirect, but given his occupation, the pervasiveness of lead in eighteenth-century households, and data showing high levels of lead in the fortified wines he enjoyed, it merits consideration.

Lead poisoning had already been classified into acute and chronic varieties in Franklin's day. The acute variety tends to disable patients in a short time and is more likely to attract the attention of physicians—particularly if it reaches epidemic proportions and is confined to a geographical area. In contrast, chronic lead poisoning is subtler, and its roots are decidedly harder to pin down. George Baker, whom Franklin helped with his landmark treatises on lead poisoning,⁴³ wrote of the chronic variety that “[t]he first beginnings of it are slight . . . it steals on by slow progression,” astutely adding, “It is gradually, and in small quantities, accumulated in the constitution; it acts slowly and lays a foundation for irreparable mischief, before any alarm is taken.”⁴⁴

With either type of lead poisoning, the pernicious metal is carried by red blood cells to the soft tissues of the body, especially the kidneys.⁴⁵

⁴¹ Becker and Jolly, “Clinical Gout and the Pathogenesis of Hyperuricemia,” 2309.

⁴² William J. Koopman and Larry W. Moreland, *Arthritis and Allied Conditions: A Textbook of Rheumatology*, 15th ed. (Philadelphia: Lippincott Williams & Wilkins, 2005).

⁴³ Finger, *Doctor Franklin's Medicine*, 181–96.

⁴⁴ G. Baker, *Medical Tracts, Read at the College of Physicians Between the Years 1767 and 1785 by Sir George Baker* (London: W. Bulmer and Co., 1818), 125–27.

⁴⁵ D. D. Choie and G. W. Richter, “Effects of Lead on the Kidney,” in *Lead Toxicity*, ed. R. L. Singhal and J. A. Thomas (Baltimore: Urban and Schwarzenberg, 1980).

Although most renal lead is then excreted or redistributed to the bones, where it may accumulate over time, the kidneys may not escape unharmed. These changes were first documented in the nineteenth century.⁴⁶ Today, the effects of lead on the fine anatomy of the kidney are well known and are envisioned as falling along a continuum.⁴⁷ At low concentrations of lead over short periods of time, there can be reversible changes in proximal renal tubule cells, including the formation of “inclusion bodies” in the cell nucleus and swelling of mitochondria, even before there are clinical signs. With greater exposure to lead, chronic nephritis or nephropathy can develop, with decreased clearance of uric acid.⁴⁸ Gilfillan points to impaired renal function as a common consequence of lead slowly entering the body, with gout as a common clinical consequence.⁴⁹

Franklin was aware of the association between lead poisoning and gout, but not the underlying mechanisms. He had examined hospital records in Europe, had read William Musgrave's works on the subject, and had helped George Baker to appreciate the perils of lead, which Baker then wrote about.⁵⁰ A century later, however, the causal relationship between lead and gout was better known.⁵¹ Alfred Baring Garrod found that 25 to 33 percent of the gouty patients he examined at Lon-

⁴⁶ J. M. Charcot and P. H. Gombault, “Note relative à l'étude anatomique de la néphrite saturnine expérimentale,” *Archives de Physiologie Normale et Pathologique* 8 (1881).

⁴⁷ Choie and Richter, “Effects of Lead on the Kidney”; R. A. Goyer, “Lead and the Kidneys,” *Current Topics in Pathology* 55 (1971); K. Cramér et al., “Renal Ultrastructure, Renal Function, and Parameters of Lead Toxicity in Workers with Different Periods of Lead Exposure,” *British Journal of Industrial Medicine* 31 (1974); R. Lilis et al., “Nephropathy in Chronic Lead Poisoning,” *British Journal of Industrial Medicine* 25 (1968).

⁴⁸ D. A. Henderson, “The Aetiology of Chronic Nephritis in Queensland,” *Medical Journal of Australia* 1 (1958); R. Kim et al., “A Longitudinal Study of Low-Level Lead Exposure and Impairment of Renal Function,” *Journal of the American Medical Association* 275 (1997); R. Lilis et al., “Renal Function Impairment in Secondary Lead Smelter Workers: Correlations with Zinc Porphyrin and Blood Lead Levels,” *Journal of Environmental Pathology and Toxicology* 2 (1979); M. Payton et al., “Low-Level Lead Exposure and Renal Function in the Normative Aging Study,” *American Journal of Epidemiology* 140 (1994); J. A. Staessen et al., “Impairment of Renal Function with Increasing Blood Lead Concentrations in the General Population,” *New England Journal of Medicine* 327 (1992); R. P. Wedeen et al., “Occupational Lead Neuropathy,” *American Journal of Medicine* 59 (1975).

⁴⁹ S. C. Gilfillan, *Rome's Ruin by Lead Poison* (Long Beach, Calif.: Wenzel Press, 1990), 85–86.

⁵⁰ G. Musgrave, *De arthride symptomatica dissertatio* (Geneva: G. de Tournes et fils, 1723); G. Baker, *An Essay Concerning the Cause of Endemial Colic of Devonshire* (London: J. Hughs, 1767); Finger, *Doctor Franklin's Medicine*.

⁵¹ A. B. Garrod, “On Gout and Rheumatism: The Differential Diagnosis and the Nature of the So-called Rheumatic Gout,” *Medico-Chirurgical Transactions*, 2nd ser., 19 (1854); Idem, *The Nature and Treatment of Gout and Rheumatic Gout* (London: Walton Maberly, 1859); Idem, *A Treatise on Gout and Rheumatic Gout (Rheumatoid Arthritis)*, 3rd ed. (London: Longmans, Green & Co., 1876).

don's University College Hospital "had been affected with lead disease." He further correlated elevated blood uric acid levels with gout, and hypothesized that lead somehow caused "deficient elimination by the kidneys." Later research confirmed that Garrod was right.⁵²

Occupational Lead Poisoning

Franklin recognized early on that he was a prime candidate for occupational lead poisoning, because printers, like plumbers, painters, and metalsmiths, had to handle lead on a daily basis. His appreciation of the perils of lead was enhanced by his observation that, after repeatedly handling hot type at Palmer's Printing House in London, he felt a strange, "obscure pain" in the bones of his hands. The details of this incident, which took place when he was eighteen, were spelled out years later in a letter to an old friend.⁵³

Franklin explained that some workers at Palmer's Printing House were in the habit of drying new lead type in front of the fireplace. The warmth of the lead felt so good on their hands that they also heated cold type when their hands became frigid in the inadequately heated building. When he began to emulate this practice, an older workman warned him that it could cause tremors and shaking, and make his hands useless for the job he had been hired to do. He also learned that there was some disagreement about how the lead entered the body. Some people thought that it penetrated the skin or was inhaled, whereas others believed it was ingested, the result of workmen not washing their hands before eating.

Franklin did not give up the printing profession after this incident. As is well known, he continued to work for established printers in Philadelphia and then set up his own printing shop, which grew into an empire.⁵⁴ But after being warned as a young man in London, he handled lead type with greater respect and care than he had previously done.

Household Lead

Lead was clearly more prevalent in eighteenth-century households than it is at the present time with so many governmental regulations. Franklin

⁵²V. Batuman et al., "The Role of Lead in Gout Nephropathy," *New England Journal of Medicine* 304 (1981); B. T. Emmerson, "The Clinical Differentiation of Lead Gout from Primary Gout," *Arthritis and Rheumatism* 11 (1968); G. D. Ludwig, "Saturnine Gout," *Archives of Internal Medicine* 100 (1957).

⁵³Smyth, *WBF*, 9:530–33.

⁵⁴Finger, *Doctor Franklin's Medicine*, 284–90.

tells us that he ate bread and milk “out of a two penny earthen porringer with a pewter spoon” after he settled in Philadelphia.⁵⁵ Storage and drinking vessels, including glazed earthenware, also contained lead, and milk might have been kept in pewter or lead-lined containers at the time. Today we know that acids and alcohol can leach the lead out of such vessels, and there are even sporadic reports of lead poisoning caused by drinking non-alcoholic beverages from pewter mugs.⁵⁶

In addition, cooking pots and saucepans containing abundant lead were routinely used for cooking in the eighteenth century. In 1780, Franklin brought his concerns to the attention of Jean-Baptiste Le Roy, a highly regarded French scientist. Le Roy, in turn, asked the chemist Lavoisier how best to plate copper vessels so they would not turn green.⁵⁷ Lavoisier replied that plating copper pans with tin mixed with a large amount of lead was the equivalent of covering one poison with another. He suggested plating with very high-quality tin, although that would add to the expense. George Baker had said almost the same thing previously, in his second treatise on lead, in which he repeatedly thanked Franklin for his help.⁵⁸ Notably, Franklin did not utilize leaded glass for his new musical instrument, the glass armonica,⁵⁹ which used a treadle to spin different-sized glass cups on a spindle, where several could be touched at the same time with moistened fingers.

Lead in Drink

Lead in spirits and wines might have been the greatest non-occupational cause of lead poisoning in the eighteenth century. Before sailing to England as a diplomat and meeting George Baker, Franklin was well informed about the “West India Dry-Gripes,” a disorder characterized by intense stomach pain and one associated with lead-tainted rum. He never fully realized, however, that small amounts of ingested lead could slowly but steadily affect the kidneys, predisposing even moderate drinkers of certain beverages to gout and stones. Only later would Garrod write unequivocally that the use of “fermented or alcoholic liquors is the most powerful of all the predisposing causes of gout.”⁶⁰

⁵⁵ B. Franklin, *The Autobiography of Benjamin Franklin* (Mineola, N.Y.: Dover Publications, 1996), 62.

⁵⁶ J. D. Scarlett et al., “Lead Poisoning by a Mug,” *Medical Journal of Australia* 163 (1995).

⁵⁷ C.-A. Lopez, “Saltpeter, Tin and Gunpowder: Addenda of the Correspondence of Lavoisier and Franklin,” *Annals of Science* 16 (1960).

⁵⁸ Baker, *An Essay Concerning the Cause of Endemic Colic of Devonshire*, 139–53.

⁵⁹ Finger, *Doctor Franklin's Medicine*, 235–50.

⁶⁰ Garrod, *The Nature and Treatment of Gout and Rheumatic Gout*, 260–73.

The best-known fortified wines are port, sherry, Madeira, and Malaga. By adding thick grape spirits (brandy) to common wines before or during the fermentation process, these wines are made firmer and more flavorful, and the stability needed for sea voyages can be achieved. While Franklin was in England, the British were importing an astonishing 3.5 to 6.9 million gallons of port annually.⁶¹ Yet the fortified wines were troublesome for two medical reasons. First, fortification increased the alcohol content by some 6–8 percent, to 17–21 percent. As noted, alcohol can affect uric acid clearance by the kidneys: hence, more alcohol, more serum uric acid. In addition, at least some of these wines had surprisingly high and potentially dangerous amounts of lead.

Some unopened bottles of wine from as early as 1770 were discovered in England in the last century, and were analyzed for lead.⁶² Although the sample was small, a report published in 1971 showed that the lead levels in these wines varied from 320 micrograms per liter in a Malaga to an amazing 1,900 micrograms in an Old Canary wine. A representative port came in at 830 micrograms of lead per liter. By comparison, drinking water with lead exceeding 15 micrograms per liter is considered dangerous in the United States at the time of this writing.

Reflecting on his rather astonishing findings, Gene Ball, the investigator, quipped that “the ‘Springtime’ of gout might really have been more leaden than golden.”⁶³ The lead in the old fortified wines was attributed to the equipment used to distill and store the grape-based spirits. Although unscrupulous vintners were still adding lead acetate to bad wines to make them taste sweeter, that did not seem to be the case for the wines in this sample. Further, these wines were not stored in leaded glass decanters, and the bottles did not have leaded caps.

Franklin thoroughly enjoyed Madeira wines. In 1744, when he went to New York to procure arms for the Pennsylvania militia, he attended a dinner where “there was great drinking of Madeira wine, as the custom of that place then was.”⁶⁴ Later, while he was a diplomat in London, colonist Benjamin Kent wrote to him to recommend “a couple of Glasses” of Madeira as the best way to take a new herbal medicine—for the gout!⁶⁵ Albert Henry Smyth, when describing Franklin’s social life, commented, “He often drank more than a philosopher should. He was particularly partial to the wines of Burgundy, and brought on

⁶¹J. Suckling, *Vintage Port* (San Francisco: Wine Spectator Press, 1990).

⁶²G. V. Ball, “Two Epidemics of Gout,” *Bulletin of the History of Medicine* 45 (1971).

⁶³*Ibid.*, 407.

⁶⁴Franklin, *The Autobiography of Benjamin Franklin*, 87.

⁶⁵Labaree, ed., *PBF*, 13 (1969): 49–50.

access of gout with the copious draughts of Nuits with which Cabanis plied him at Auteuil. But he was also fond of Madeira, and liked to gossip with his friend Strahan over the second bottle.”⁶⁶

Although bottles of Madeira were then about half the size that they are now, Claude-Anne Lopez and Eugenia Herbert might have put it best when they wrote, “Old Madeira had a special place in his affections.”⁶⁷ Most telling are the surviving records of how many bottles of wine of each type were kept in his wine cellar in France. On 1 September 1782, he had some 216 bottles of Madeira, as compared with just 153 bottles of *vin ordinaire*, or common table wine! But Franklin also enjoyed port, then the most popular of the fortified wines. In 1761, while in England, he noted that port is so “universally drank here” and so esteemed, that the enterprising British were now beginning to produce it themselves.⁶⁸

Franklin continued to drink fortified wines after he returned, old and frail, to the United States, albeit mostly when he felt that his gout was under control. He did not seem concerned that George Baker had damned these wines, which were sometimes also laced with lead and stored in lead-glazed vessels.⁶⁹ Baker rightly pointed out that ordinary wines were also sometimes adulterated with lead salts to make them taste sweeter—an offense with ancient roots and one punishable by death in some places.⁷⁰ In his words, “Notwithstanding the severe laws, which are still in force, both in France and in Germany, against the adulteration of wines, by the means of litharge [a lead-based additive], we still frequently find that the small French white wines, and the Rhenish and Moselle wines, bear marks of this most pernicious fraud.”⁷¹

FINAL THOUGHTS

Although it is impossible to give a weight to each of Franklin’s numerous risk factors for gout and stones, their co-occurrence suggests that cumulative effects had to be inevitable. For example, his meaty diet accompanied by a love of Madeira wine might have been more dangerous for him than for others, given the other factors in his medical his-

⁶⁶ Smyth, *WBF*, 10:407.

⁶⁷ C.-A. Lopez and E. W. Herbert, *The Private Franklin* (New York: W. W. Norton & Co., 1975).

⁶⁸ Labaree, ed., *PBF*, 9 (1966): 399–401.

⁶⁹ Baker, *Medical Tracts, Read at the College of Physicians*, 187–89.

⁷⁰ J. Eisinger, “Lead and Wine: Eberhard Gockel and the *Colica Pictonum*,” *Medical History* 26.3 (1982): 279–302; J. Nriagu, *Lead and Lead Poisoning in Antiquity* (New York: John Wiley & Sons, 1983); Skovenborg, “Lead in Wine through the Ages.”

⁷¹ Baker, *Medical Tracts, Read at the College of Physicians*, 131.

tory. That Franklin's brother John suffered from stones, suggests that a meaty diet or alcohol levels marginally safe for other men might have been unusually hazardous for this Founding Father.

The interaction of genes and heavy wine drinking is particularly interesting because Franklin's friend Benjamin Rush had specifically brought this matter to his attention in 1773. The influential American physician—whom Franklin had once helped to get into Edinburgh's medical school—had written the following to him: "Dr. Gaubius in his Pathology speaks of certain remote Causes of Diseases which act only on what he calls the 'predispositis.' Thus a few Glasses of Wine will bring on a Fitt of the Gout upon a man who inherits a gouty Constitution, provided he drinks them at the usual seasons of that Disorder's attacking him. The same Quantity of Wine will have no Effect upon a Man who is not predisposed, or subject to the Gout."⁷²

As for the possible interaction of ingested lead with a meaty diet, the previously mentioned gout historian Gene Ball points out that "lead nephritis would have been especially apt to be associated with serious hyperuricemia in this group of [gouty] patients whose solid diet was meaty."⁷³ Aging, too, can act synergistically with lead. It can increase the kidneys' susceptibility to lead's toxic effects and raise serum uric acid levels well above those found with advancing age by itself or with lead exposure in younger subjects.⁷⁴

Franklin, with his connections to the medical community and love of books, was at least as knowledgeable as most eighteenth-century physicians about the causes of gout and stones. He even conducted experiments on his own body, hoping to find safe ways to treat these conditions.⁷⁵ But given the host of risk factors he faced, his body was caught in a vital struggle that it had little chance of winning—a war in which a great many insidious things conspired against it, much like a tragic hero surrounded by many strong and determined enemies.

Although Franklin's gout and large bladder stone reduced his mobility and caused him severe pain and inconvenience, he remained good-natured and somehow retained his legendary sense of humor. Three years before his death, while hounded by pain and increasingly affected by the advancing weakness and inanition of chronic urinary system damage, he retained his gift for self-palliation with metaphor. He wrote, "People who live long, who will drink of the cup of life to the very bottom, must expect to meet with the usual dregs, and when I

⁷²Willcox, ed., *PBF*, 20 (1976): 404.

⁷³Ball, "Two Epidemics of Gout," 407.

⁷⁴Lilis et al., "Renal Function Impairment in Secondary Lead Smelter Workers."

⁷⁵Finger, *Doctor Franklin's Medicine*.

reflect on the number of terrible maladies human nature is subject to, I think myself favoured in having to my share only the stone and the gout.”⁷⁶

Franklin’s cup of life, a cup brimming with fortified wines, sometimes accompanied by lavish meals, clearly contained some hidden “dregs” that would have affected his physiology and indirectly contributed to his demise. While it was a lung abscess (a vestige of older infections) that ultimately killed him, his death can also be associated with the fragile state of his body and especially the debilitated condition of his urinary system—in turn the result of multiple, sometimes stealth-like pathological forces, a number of which have been elucidated here.

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⁷⁶Smyth, *WBF*, 9: 560–61.