

The Black Devil Called Ergot

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Professor Mary Beth Norton

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The hypothesis that a certain fungus, known as ergot, might be somehow connected to the events occurring around Salem Village in 1691/2 has been controversial. The article “Ergotism: Satan Loose in Salem,” by Linnda R. Caporael was introduced in April of 1976 with a compelling story of a chemical induced hysteria resulting in the Salem witch trials. However, very shortly afterwards her work was heavily criticized by psychologists Nicholas P. Spanos and Jack Gottlieb in their December 1976 article, “Ergotism and the Salem Witch Trials.” The topic from then on remained muted until 1989 when Mary K. Matossian revisited the subject in her book, *Poisons of the Past: Molds, Epidemics, and History*. Matossian made a compelling argument favoring ergot’s role in Salem gruesome past. Reevaluation of historical documents and also the analysis of previously overlooked climate and agricultural accounts provide a new look at a plant pathogen and its presence in New England. Evidence points to the existence of ergot in Massachusetts and the appearance of convulsive ergotism symptoms in Elizabeth “Betty” Parris and Abigail Williams suggests that they had initially suffered from ergot poisoning.

In order for ergot to be involved at all in this historical event it must first be placed at the scene of the crime, and for the fungus to be present it must first have a host to live on. Ergot is caused by one of the ascomycete fungi, or sac fungi, called *Claviceps purpurea*. It occurs throughout the world on grasses and plants cultivated for cereal grains, most commonly on rye. The fungus reproduces by infecting flowering parts with ascospores and then colonizing the ovary of that particular flower. From there it forms sporodochia which produce sphaecelia conidia. The conidia then enter the “honeydew” stage when atmospheric moisture levels are high. During this stage of the life cycle the rye grain heads will drip what looks like diluted

honey. The conidia then reinfect the flower and produce a hard mass of mycelium called a sclerotium. This mass is black in color and remains on the plant with the other grains until it is harvested. The sclerotia contain alkaloids that are toxic to both humans and animals.¹ Many governments around the world have recognized how important it is to control the amount of ergot allowed within a given amount of grain. The European Union allows only a maximum of 0.05% of ergot alkaloids in grain for human use, that's roughly one milligram of alkaloids per kilogram of food. Grain for animal use is restricted to 0.1%. Similar standards are enforced in the United States.²

The two main types of ergot alkaloid poisoning are called gangrenous ergotism and convulsive ergotism. In gangrenous ergotism the blood supply to the extremities is cut off by action of the ergotamine alkaloid, which in turn causes gangrene and the limbs waste away. Convulsive ergotism is caused by the alkaloids ergine and lysergic acid hydroxyethylamide. The symptoms range “from vomiting, diarrhea, and general lethargy, to a sensation of ants crawling over the body (formication), vivid hallucinations, twitching, grotesque distortion of limbs, and seizures similar to those associated with epilepsy.”³ The amount of ergot alkaloids produced within a sclerotium is dependent upon growing conditions. If weather is not perfectly ideal for ergot production, then the amount of alkaloids produced per sclerotium is lower and therefore symptoms are not as intense. If an individual was to ingest ergot with very low levels of alkaloids it might not seem to cause much harm. The symptoms in a case like this might be noticed as fertility suppression, abortion, or miscarriage in women.⁴

¹ George N. Agrios, *Plant Pathology* (London: Elsevier Academic Press, 2005), 501-503.

² Thomas Haarmann, et al., “Ergot: from witchcraft to biotechnology,” *Molecular Plant Pathology* 10(2009):569.

³ George W. Hudler, *Magical Mushrooms, Mischievous Molds* (Princeton: Princeton University Press, 1998), 72.

⁴ Mary Allerton Kilbourne Matossian, *Poisons of the Past: Molds, Epidemics, and History* (New Haven: Yale University Press, 1989), 12-14.

Dr. Gabbai, Dr. Lisbonne, and Dr. Pourquier observed an outbreak of convulsive ergotism in Pont St. Esprit, France in 1951. According to their observations, “many of the patients did not show the signs and symptoms in their entirety.”⁵ Many women were also noted to have menstruated prematurely.⁶ Regarding hallucinations, they noted that “the particular themes were visions of animals and of flames. All these visions were fleeting and variable.”⁷ Also in this case the disorders seemed to develop in children before it developed in adults. Another point of interest they had observed in patients is that in some cases “delirium was the first serious sign to be noted; it then appeared very late—between 10 to 12 days after the first onset of poisoning.”⁸ Gangrenous ergotism was not mentioned in their report, although one woman that did show slight gangrene of her toes. However, she had existing health conditions that would have contributed to that development.⁹ It seems normal, in many cases, that either gangrenous or convulsive ergotism occurs in a particular epidemic; both do not seem to occur simultaneously.¹⁰

Weather conditions from 1690 to 1691 appear favorable for the development of ergot according to various diaries, journals, and records. A cold winter prior to the year of infection followed by a damp summer would increase the chance of infection.¹¹ Samuel Sewall recorded in 1690 the following entries: Nov. 30. “Tis extream cold;” Dec. 1. “Extream cold;” Dec. 16. “Very cold;” Wednesday Decembr 17. “Tis so cold and so much Ice in the Charlestown River.”¹² Later

⁵ Dr. Gabbai, Dr. Lisbonne, and Dr. Pourquier, “Ergot Poisoning At Pont St. Esprit,” *British Medical Journal* 2(15 September 1951):651.

⁶ *Ibid.* 651.

⁷ *Ibid.* 651.

⁸ *Ibid.* 651.

⁹ *Ibid.* 651.

¹⁰ Hudler, *Magical Mushrooms*, 74.

¹¹ Matossian, *Poisons of the Past*, 119.

¹² M. Halsey Thomas, *The Diary Of Samuel Sewall: 1674-1729 Newly Edited from the Manuscript at the Massachusetts Historical Society* (New York: Farrar, Straus and Giroux, 1973), 271-272.

in the year 1691 he mentioned great thunderstorms on July 20 and August 6.¹³ Then on August 28 he mentioned the heat of summer being so great that his cousin had to be buried at night.¹⁴ Tree ring records also indicate that the early growing season in New England was cooler than normal from 1688 through the 1690's.¹⁵ The cool weather in early summer would increase the window of opportunity for infection of rye to occur and then the hot late summer would allow for decent growth. A good deal of the farm land used to grow rye was near swamp land; these areas would be prone to higher humidity throughout the year. Aside from weather conditions, the local landscape can affect the incidence of ergot in an area; fields in low-lying regions or valleys near water were “more heavily ergotised than its neighbours, and led to sporadic outbreaks of ergotism.”¹⁶

There is no definitive mention of the ergot fungus by name in any record in early New England; however, there were a number of mentions of wheat “blasting.” Wheat blast was a disease mentioned numerous times throughout history and even in the Old Testament of the Bible. It was known to cause kernels of grain to turn black in a head of wheat or other cereal crop. A comparison of “blasting” and ergot side by side can demonstrate how easily one might be mistaken for the other;¹⁷ especially since ergot was not “discovered” in North America until 1807.¹⁸ Records of blast are mixed in with other characteristics of disease that are very similar to that of ergot. In an anonymous manuscript sent to England in 1637 discussing the swamp areas

¹³ Thomas, *Diary of Samuel Sewall*, 280.

¹⁴ *Ibid.* 280.

¹⁵ Matossian, *Poisons of the Past*, 120. Presented by Matossian from E. DeWitt and M. Ames, *Tree-ring chronologies of eastern North America* (Tucson: 1978), 19.

¹⁶ George Barger, *Ergot and Ergotism: A monograph Based on the Dohme Lectures Delivered in Johns Hopkins University, Baltimore* (London: Gurney And Jackson, 1931), 99; The Map of Salem Village, 1692. By W. P. Upham in 1866 shows a number of hills intermittent with bodies of water that include brooks, rivers, and ponds. The area around those water bodies would be low land areas.

¹⁷ Agrios, *Plant Path.*, 12 and 502.

¹⁸ Linnda R. Caporael, “Ergotism: The Satan Loosed in Salem?” *Science* 192(2 April 1976):23-24.

of New England it is said that “rye likes it not.”¹⁹ It continued to say “Hay, we have here of the low lands, such as it is, which, in my opinion, is inferior in goodness to our reed and sedge in England,..., that our beasts grow lousy with feeding upon it,..., besides, it breeds among them sundry diseases, which we know not how to cure.” The entry also mentioned about the grass disease that it “brings our cattle so low, and many times to disease of which they hardly ever recover.”²⁰ This record establishes that some disease existed within the native grass population of early New England that poisoned grazing cattle, making them unhealthy or worse.

A manuscript written by a certain Mr. Hubbard²¹ in 1680 said “Many swamps and boggy places,..., such as they call interval land, in level and champain grounds, that oftentimes are overflowed by the channels of water which run beside them, which is supposed to enrich the soil that is so watered.”²² A footnote explained this entry saying that “sometimes, a great freshet²³, in the months of June or July, is prejudicial to the crops upon the mowing and pasture lands, for that reason, making the grass foul and disagreeable to the cattle.”²⁴ Floods in June or July would have been favorable for growth of ergot within grass and cereal crop populations, and also this growth would have made the grass unhealthy for the cattle to eat. It also seems as if this grass disease was able to persist for years.

In Thomas Hutchinson’s *The History of Massachusetts*, another clue to the identity of the “blasting” of grasses is given; “The great discouragement has been the blast... Generally,

¹⁹ Thomas Hutchinson, *The History of Massachusetts, from the first settlement thereof in 1628, until the year 1750* (Salem: Thomas C. Cushing, 1795), 426.

²⁰ Hutchinson, *Hist. of Mass.*, 427.

²¹ *The New England Historical & Genealogical Register, Published Quarterly, Under the Patronage of the New England Historic, Genealogical Society* (Boston: Samuel G. Drake, 1851), 142-143. Accessed via <<http://books.google.com/>>. Mr. Hubbard was the Reverend William Hubbard Jr. of Ipswich.

²² Hutchinson, *Hist. of Mass.*, 428.

²³ A freshet is a flood caused by rain or melting snow.

²⁴ Hutchinson, *Hist. of Mass.*, 428.

between the first and tenth of July it has been observed, that the dew, called the honey dew, falling upon the wheat (the morning after being hot and calm) causes the rust or blast.”²⁵ The description of abundant moisture during July and “honey dew” which led to the later formation of blackened grains, or blast, that make the cattle sick is comparable to the life cycle of ergot and the detrimental effects it can have after consumption by animals.

It was also noted that in New York “a monthly fast was appointed to be observed from September 1691 to June 1692; the special reasons assigned for which were, “a burthensome war, and a blast upon the corn.”²⁶ “Corn” meant cereal grain plants in New England at this time; corn as we know today was called “Indian corn.”²⁷ This evidence of disease not only points to *Claviceps purpurea* as being the probable cause of “blasting,” but also as being present in North America during 1691.

With records that indicate such plant disease in an area relatively near to Salem, and of similar climactic conditions, there is a very real possibility that ergot existed in Massachusetts during the 1691 growing season. Another point to consider is that the ergot fungus has existed within agricultural populations around the world for hundreds, if not thousands, of years. Ergot was first mentioned in the 1582 edition of Adam Lonicer’s *Kreuterbuch*,²⁸ and since then there have been many records of the disease within populations, including the “Great Awakening” of New England in 1741.²⁹ Also, if in fact ergot was being referred to in seventeenth century Massachusetts, then the inoculum to produce infection of grass and cereal plants would still be

²⁵ Hutchinson, *Hist. of Mass.*, 429.

²⁶ Noah Webster, *A Brief History of Epidemic and Pestilential Diseases; With the Principal Phenomena of the Physical World, which precede and accompany them, and observations deduced from the facts stated* (Hartford: Hudson and Goodwin, 1799), 205-206.

²⁷ Matossian, *Poisons of the Past*, 119.

²⁸ Barger, *Ergot and Ergotism*, 7.

²⁹ Matossian, *Poisons of the Past*, 123-141.

present every year even if only in limited amounts. The people that lived in Massachusetts at this time would have certainly not practiced modern agricultural techniques, such as deep tilling, to eradicate, or at least suppress, inoculum levels year after year. After all, these people did not know about ergot, so why would they attempt to manage it? Another point to consider is that many farmers would have used stored seed from the previous year to plant their new fields. If ergot had been present at the previous harvest then there would be a very good chance that the farmer would have planted the grain crop and the ergot sclerotia together in the first inch or two of soil the next year. Development of the ergot at this point would depend on weather and location to determine whether or not the incidence of infection was high or low.³⁰

The Salem Witchcraft crisis started in the home of Samuel Parris in January 1691/2, by the Julian calendar, with Elizabeth “Betty” Parris and Abigail Williams.³¹ The time at which their symptoms first appeared coincided with the onset of symptoms described within the oldest English account of ergotism. The account revealed that those with the disease would suffer “every year in the month of December and January.”³² The ergot alkaloids could have been introduced into the two girls systems through ingestion of infected rye bread or possibly through inhaling the fine particulates of infected rye flour while in the kitchen of Samuel Parris’ house. It is often believed that the girls lingered in the kitchen with Tituba.³³

After the initial accusations of witchcraft by Betty Parris and Abigail Williams, it becomes difficult to distinguish actual cases of illness and suffering from those simply acting out in fear. Many of the other accusers gave depositions that were very similar to one another.

³⁰ Agrios, *Plant Path.*, 501-504.

³¹ Mary Beth Norton, *In the Devil’s Snare* (New York: Vintage Books, November 2003), 3 and 18.

³² Barger, *Ergot and Ergotism*, 32 Quote from translation of *Sennertus: De Febribus [Of Agues and Fevers, 1658]* The author of this thought the ergotism was a continuing disease because he did not understand that reinfection had been occurring year after year.

³³ Thomas, *Diary of Samuel Sewall*, 290.

Records dealing with medical history are practically non-existent at the time because the focus had turned to the supernatural rather than the natural as an explanation. The circumstances surrounding these accusations became increasingly complex as the trials continued through 1692, and that complexity has led to much speculation. At that point the series of accusations may have been influenced by social cues rather than a feeling of true suffering, as suggested by Nicholas Spanos and Jack Gottlieb.³⁴

However, the two girls were suffering under strange distempers, which the town physician was unable to understand, before social cues would have influenced them. They were “bitten and pinched by invisible agents; their arms, necks, and backs turned this way and that way, and turned back again, so as it was impossible for them to do of themselves, and beyond the power of any Epileptick Fits, or natural Disease to effect. Sometimes they were taken dumb, their mouths flopped, their throats choaked, their limbs wracked and tormented.”³⁵ The symptoms described here by John Hale illustrate an array of characteristics like those found in a person suffering from convulsive ergotism.

Samuel Parris would likely have felt great pressure from his own followers and those who were opposing him when it was observed that the devil had infiltrated his own house. The pressure he experienced would lead to increased pressure on the girls for the source of their affliction. With no other idea about what the cause could be the town physician suggested witchcraft. This suggestion to the girls would provide a source of fear and imagery of witchcraft. Sermons given by Samuel Parris soon after would have served only to stimulate their

³⁴ Nicholas P. Spanos, and Jack Gottlieb, “Ergotism and the Salem Village Witch Trials,” *Science* 194(24 December 1976), 1391.

³⁵ John Hale, *A modest enquiry into the nature of witchcraft, and how persons guilty of that crime may be convicted: and the means used for their discovery discussed, both negatively and affirmatively, according to Scripture and experience* (Boston: B. Green and J. Allen, 1702), 23-24.

imagination. When symptoms of hallucinations arrived some weeks later in February,³⁶ the earlier suggestions and pressures would help to start the chain reaction of accusations in Salem.

The alkaloid lysergic acid hydroxyethylamide, in ergot, is the chemical from which LSD, or lysergic acid diethylamide, is derived. People under the influence of LSD, or similar compounds like those in ergot, become highly susceptible to suggestion and may create images in their mind of people, animals, or even religious scenes.³⁷ The girls, whose minds were currently under the influence of LSD-like substances, would have been open the suggestions and fears brought before them by Samuel Parris, the town physician, or others who witnessed their behavior and persistently questioned them.

Information of someone, such as Tituba, seeking help against the devil through the devil would have made its way to the girls and then shortly thereafter, in their easily influenced state of being, they claimed that she was the one causing their pain. John Hale wrote of this and said that Tituba made a witch cake and “after this, the Afflicted persons cried out the Indian Woman, named Tituba”³⁸ tormented them. After the first complaints of witchcraft had been made at the end of February 1691/2 the number of the afflicted began to increase.³⁹ The traumatic episodes of fits and contortions witnessed in the girls would have scarred the minds of the people at the time; with no other explanation for the disorder, it was surely demonic.

Dr. Nicholas P. Spanos and Jack Gottlieb attempt to discredit the notion of ergot playing any role in the Salem witch trials. However, the evidence they seemed to have used was not completely correct. A first point made by them was that George Barger, author of *Ergot and*

³⁶ It was noted with interest in the Pont St. Esprit ergotism episode that hallucinations appeared 10-12 days after the initial set of symptoms.

³⁷ Matossian, *Poisons of the Past*, 116.

³⁸ Hale, *A modest enquiry*, 23-24.

³⁹ Norton, *Devil's Snare*, 21.

Ergotism, claimed that convulsive ergotism occurred in populations with vitamin A deficiencies.⁴⁰ This was based on circumstantial evidence as Barger formulated his hypothesis. However, more than 80 years after Barger's hypothesis was made, no direct evidence has ever linked convulsive ergotism and vitamin A deficiencies to support this claim.⁴¹

A second point made by Spanos and Gottlieb was that gangrenous ergotism did not occur. They said, "the absence of any instance of gangrenous symptomatology makes it highly unlikely that ergot played any role in the Salem crisis."⁴² Records of ergotism throughout history show that convulsive ergotism and gangrenous ergotism do not seem to manifest in the same epidemics.⁴³

Spanos and Gottlieb also claimed that ergotism was unfounded because the girls were too old.⁴⁴ Young children are noted to be more susceptible, especially those under fifteen years of age.⁴⁵ Betty Parris was nine years old at the time and Abigail Williams was only about eleven, which is well below fifteen.⁴⁶ They also argued that everyone in a family should have developed the symptoms of illness if one person in the household had them. That statement is false; the amount of ergot ingested by one of the girls might not have had the same effect on another individual of the same household. It was observed by Dr. Gabbai that not all symptoms occur and that intensity of symptoms varies in a population.⁴⁷

⁴⁰ Spanos, *Ergotism*, 1390.

⁴¹ Mervyn J. Eadie, Convulsive ergotism: epidemics of the serotonin syndrome?" *The Lancet* 2(July 2003):433.

⁴² Spanos, *Ergotism*, 1390.

⁴³ Barger, *Ergot and Ergotism*, 21; Hudler, *Magical Mushrooms*, 75; Eadie, *Convulsive ergotism*, 431.

⁴⁴ Spanos, *Ergotism*, 1390.

⁴⁵ Barger, *Ergot and Ergotism*, 39.

⁴⁶ Hale, *A modest enquiry*, 23.

⁴⁷ Gabbai, *Ergot Poisoning*, 651.

A number of factors were responsible for the events that took place in Salem Village during 1692. There has been much speculation into the cause of the abrupt change in how witchcraft cases were handled in New England, but most are not entirely supported by the very limited evidence available. Many records, especially those regarding early agriculture, are quite rare. Even if the records did exist, most of those in that society would not have known of it to report it. However, when looking at the history of a plant pathogen and its inoculum availability all over the globe and specifics about its disease cycle that appear in occasional records, it makes for a plausible case that ergot and convulsive ergotism influenced the first accusations by Elizabeth Parris and Abigail Williams.

Glossary⁴⁸

Ascomycete: A group of fungi that produce their sexual spores, ascospores, within sac like cells of hypha, called asci.

Ascospore: Sexually produced spore borne in an ascus.

Conidium (pl. Conidia): An asexual fungus spore formed at the end of a conidiophore.

Mycelium: The hypha or mass of hyphae that make up the body of a fungus.

Sclerotium (pl. Sclerotia): A compact mass of hyphae with or without host tissue, usually with a darkened rind, and capable of surviving under unfavorable environmental conditions.

Sporodochium (pl. Sporodochia): A fruiting structure consisting of a cluster of conidiophores woven together on a mass of hyphae.

⁴⁸ Agrios, *Plant Path.*, 888-899

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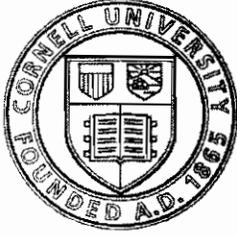
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