There’s Always Been a Lot of Mycobacteria Out There

Some 300 million years ago it seems that the common ancestor of today’s species of mycobacteria began to live in close association with a range of animals. These parasitic forms became dependent on their hosts for survival. As is the nature of parasitism, they exacted a toll on the host: disease and/or premature death. Other species retained the ancestral habit, remaining at large in the environment in fresh water, soil, dust, and peat bogs. The majority lived (and live today) as saprophytes—on dead and decaying matter—indeed some environmental mycobacteria may well prove to be important in the future manufacture of biofuels.

Among the parasites, most important for the history of human disease are Mycobacterium leprae and the Mycobacterium tuberculosis complex (MBTC). M. leprae causes leprosy. The Mycobacterium tuberculosis complex includes M. tuberculosis, M. africanum, M. bovis, and M. canettii. All can cause tuberculosis. Each member of the MBTC has a preferred mammalian host
and a slightly different appearance (the phenotype). They appear to be an extremely consistent bunch at the genetic level (the genotype), which usually means they are the clonal descendants of a single successful ancestor. The date for their emergence is currently set at 20,000–35,000 years ago. What was happening before this is actively being explored.¹

The bit players are the non-tuberculosis mycobacteria (NTM), a term used for all the other disease-causing but free-living species. These can produce an extensive range of ailments including inflammation of the lymph node (lymphadenitis), skin diseases, tuberculosis-like pulmonary disease, inflammation of the bones (osteomyelitis), and post-traumatic wound infections. Besides direct illness, historical exposure to these related bacteria might have affected the evolution of the immune response to the tuberculosis-causing bacilli. Non-tuberculosis mycobacteria also cause post-operative infections linked to catheter use and the diverse infections and disseminated diseases in the immuno-compromised. These pathologies reflect new human associations with ancient organisms, the side effects of modern invasive medicine, and the changing nature of the human disease profile—most spectacularly the rise of the HIV/AIDS epidemic in the last thirty years.

It is argued that as today’s vertebrates evolved those harbouring the early mycobacteria carried them along, host and parasite developing together—vertical transmission. It used to be confidently and quite reasonably thought that *M. tuberculosis* mutated from the closely allied *M. bovis* of cattle and goats, spreading to humans by a horizontal transfer when we began domesticating these animals about 9000 BCE in the Neolithic. This is a familiar model in the history of infectious diseases: a zoonosis (one initially spread from animals to people) becomes
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a permanent part of the human disease economy. The specificity of *M. tuberculosis* for humans rather than the more omnipresent *M. bovis* was interpreted as supporting evidence.

Larger groups of humans generally lived more settled lifestyles as members of herding communities. They potentially enjoyed more regular sources of protein from the meat and especially the milk of the ruminants. On the downside, giving up the mobile small-group hunter-gatherer lifestyle brought a greater proximity to other people and to *M. bovis*. In cattle *M. bovis* bacteria caused (and causes) a chronic disease although the animal remained useful. Ingested by humans in milk or meat, this organism produced what we would recognize as tuberculosis, with the gut and the nearby lymph nodes as the primary sites of infection. Cows also cough and out of this germ pool, it was postulated, mutations of *M. bovis* became adapted, as *M. tuberculosis*, to the oxygen-rich environment of the human lung. Once lodged here, the bacteria became capable of spreading directly between one human and the next and, in effect, a new form of disease predominantly targeting the pulmonary system came into being.

So far so good, but recent DNA sequencing of the *M. tuberculosis* genome suggests a rather different evolutionary path for the *Mycobacterium tuberculosis* complex and therefore the human disease’s history. Some of the older model still pertains—*M. bovis* was (and remains) a source of infection; population density plays a significant role in the transmission of pulmonary tuberculosis. But it is now suggested that a human germ has been around rather longer than its cattle equivalent. Instead of *M. tuberculosis* being a mutation of *M. bovis*, analysis of the genomes suggests that *M. tuberculosis* is the older of the two, *M. bovis* evolving via the ancestor of *M. africanum*, one of the
other human tuberculosis-causing species. This ancestral bacterial organism may well have been bothering our hominin precursors in East Africa with tuberculosis-like illness. Thus a hominin-derived rather than animal-derived pathogen could be responsible for much of our modern experience of tuberculosis. As well as potentially explaining the past, this has implications for assisting with current epidemiological puzzles, such as the effective geographical range of the BCG vaccination.

Whatever their origin, all mycobacteria have evolved a unique thick cell wall made from glycolipids and lipids. These waxy molecules are part of the group’s evolutionary success rendering them safe from assault. They help the bacteria resist long exposure to acids and alkalis; guard against splitting or lysis by proteins in body fluids; and protect against antibiotics like penicillin, which do battle by destroying the cell coat of many other common disease-causing bacteria. In the bacterial pantheon, mycobacteria are slow growers. *Escherichia coli* takes only twenty minutes to divide itself into two, a further twenty minutes for these two to become four, and so on. *M. tuberculosis* requires twenty to twenty-four hours to divide once. It routinely takes over seven days for a colony to become clearly visible to the naked eye.

Such sluggard division is the result of the cell wall’s complexity. When preparing to divide, the many layers of the cell wall must be synthesized, but the rod-like shape maintained. Then careful management of the breakdown of the existing cell wall components to maintain integrity throughout division is necessary, all the while responding to unfavourable changes in the surrounding environment. The slowness of cell division and its fine control are part of the chronic nature of tuberculosis. So too are the persistent but often subclinical infection and the
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trick of dormancy with the ability to break this stasis when opportune. All these strategies appear to be modulated by sensitive feedback loops. Lipid-rich cell walls have a further utility in the history of tuberculosis—it is likely that this feature makes the bacteria extremely resistant to degradation over very long periods of time. With due care their ancient DNA (aDNA) can be extracted, amplified, and used as proof of the presence of disease in the appropriate context in humans and animals.

The key words for mycobacteria are longevity, resilience, and ubiquity. Welcome to the world of tuberculosis.

A Prehistoric Scourge

Knowledge of the long history of human interactions with mycobacteria comes from a variety of sources. These have been interwoven into histories of disease, human settlement, social development, war, and conquest. Under due scrutiny bones and mummies can provide evidence of what we understand as historical tuberculosis. Some are more unequivocal than others. There has been and remains an understandable temptation to fit the facts to convenient ‘just so stories’ which locate the history of tuberculosis into a wider historical narrative. So bearing this in mind, over to the bones, bodies, and ancient DNA.

Mycobacteria leave certain marks on bones and soft tissues. Visual inspections by palaeoosteologists (bioarchaeologists in the USA) who study ancient bones and palaeopathologists had the bacteria present and interacting deleteriously with their human hosts from at least around 5800 BCE. This is the age (plus or minus 90 years) of the oldest tuberculous skeletal remains among a collection from Liguria, Italy. Often the spine provided proof for infection most dramatically when the degenerated
and fused thoracic (occasionally lumbar) vertebrae collapsed into a sharp angular deformity (kyphosis) and gave its victim a hunchback during life. The unfortunate but now famous Nesperehan, an Egyptian priest of the Twenty-first Dynasty (1100 BCE), had a pronounced hunchback and a psoas abscess. This pus-filled swelling attached to the psoas muscle of the back is highly characteristic of a modern diagnosis of extrapulmonary tuberculosis. Lesions on the ribs—on the internal surface, where the membrane surrounding the bone has become inflamed following contact with tuberculous lung tissue—can be read as markers of the pulmonary form. Less frequently but very usefully, as in the case of an ancient Nubian woman from the island of Hesa, preserved soft tissues—a collapsed lung and fibrous adhesions—alert historians to the presence of what we think of as the most common pulmonary type of tuberculosis.

Around the world burial sites and preserved bodies hinted at how widespread tuberculosis may have been. In the Old World, material from Poland, Spain, Russia, Greece, Thailand, China, and Denmark followed Italy and Egypt on the tubercular map, as the Neolithic became the Bronze and then the Iron Age. The pattern of spread seemed to follow the developing civilization and urbanization, which stretched out from the Fertile Crescent in the Near East. One by one most of the other countries of Europe and Asia can be added through the centuries of the first millennium CE to the later medieval period. By this time tuberculosis appears to have become endemic, an ever-present smouldering cause of sickness and death. Its effects were masked to some extent by the ravages of the acute infectious diseases. However, what we might be sampling is the development of burial practices over time and place as much as the spread of the disease.
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In the New World tuberculosis left its calling cards too. The increasing numbers of tuberculous mummies apparently dating from the first millennium CE, conserved by hot, dry conditions in Peru and Chile, proved to be something of a conundrum. Tuberculosis, it used to be thought, had not entered continental America in the first wave of human occupation towards the end of the last glacial period perhaps 17,000–10,000 years ago. It was assumed that active cases would be lost in the natural wastage on the initial stages of the journey from the Old to New World and any latent cases would then have succumbed to the ‘cold screen’ endured by the survivors as they crossed the Bering Land Bridge. It was posited instead that tuberculosis came as part of the Columbian Exchange of the late 15th and early 16th centuries when the Spanish and Portuguese accidentally discovered and conquered South America on their way to India. The invaders wreaked havoc with the germs they carried—smallpox, measles, and tuberculosis—among the immunologically naïve locals and took syphilis home with them in return, hence the ‘exchange’.

Preliminary investigations looked merely for the presence of acid-fast bacilli. Acid-fastness refers to a characteristic of some bacteria, when dyed, to increase their visibility under the microscope and crucially includes the mycobacteria. Where acid-fast bacilli were found in the context of the appropriate lesions—tell-tale pathological damage—they gave reasonable evidence for the presence of mycobacteria and hence tuberculosis. The recovery of such organisms in 1973 from a mummy in Southern Peru with a reliable radiocarbon date of 700 CE promoted a (somewhat reluctant) reassessment of the history of tuberculosis in South America. Subsequent developments in aDNA recovery and polymerase chain reaction (PCR) amplification
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substantiated the challenge: *M. tuberculosis* complex bacteria in South America had caused tuberculosis-like illnesses from at least 2000 BCE in Chile. How did the disease originate if it had not been brought by the conquistadors? Did it come with earlier landings on the Pacific coast from Asia and Oceania? More needs to be done here as the history of the peopling of the Americas is revised, but the longevity of the disease is now beyond doubt.

What of North America, where the Native American’s similar decimation by ‘imported’ infectious diseases led to the assumption that they too had no prior exposure to tuberculosis? The skeletal evidence from large settlements at Norris Farms in the Illinois River Valley dated to 1300 CE, for instance, has been sufficient to allow estimates of tuberculosis’s endemicity in a specific population. A supplementary PCR technique—spoligotyping—confirming both the presence and the strain of the *Mycobacterium tuberculosis* complex is providing material for partial answers and it seems that the bovines might be partly to blame after all. Not for passing on *M. bovis* though. Spoligotyping of aDNA from a long-dead (17,000 years BP) American bison with characteristic tubercular lesions reveals the likelihood that this animal was infected with the precursor of the *Mycobacterium tuberculosis* complex. Further sampling of other animals from the same source in Wyoming—bighorn sheep and musk ox—indicates that the animals that wandered over the Bering Strait during the Pleistocene carried the pathogen with them and that it was widespread.

Back in the Old World during the past twenty years many of the previously scrutinized bones and bodies have been reinvestigated using the developing aDNA techniques. As a result some would like to move the earliest case back to 9,000 years
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ago from a Neolithic settlement in Atlit-Yam in the Eastern Mediterranean—a submerged village off the coast of present-day Israel. Here the bones of a mother and child who both died of tuberculosis (determined by the bone lesions and presence of aDNA) were found along with those of extensive animal remains. Here the shift to fishing, farming, and animal keeping apparently supported a population density capable of maintaining a settled village life and endemic as opposed to sporadic tuberculosis.8

If prehistoric tuberculosis seems far away, older still, much older in fact, from near Kocabaş in Turkey part of a fossilised Homo erectus skull dating from between c. 510,000 and 490,000 years ago bears lesions on its inner surface thought to be the result of tubercular meningitis. This is currently the oldest known example of a tuberculosis-like pathology in a fossil hominin.9 If this seems a little early for the presence of modern M. tuberculosis (thought to be c. 35,000 years old), the ancestral progenitor species from which it developed its cunning complexities, is estimated to be perhaps three million years old: plenty of time to dog the proto-human race.

New ways of investigating the history of M. tuberculosis enhance knowledge about the germ’s past. Serendipitous finds of ancient material can drive back the tuberculosis timeline, reconfiguring aspects of the disease’s record. We continue to unpack and then repack a construction of the history of tuberculosis, but it quickly becomes a history of the mycobacteria rather than the experience of disease. Tantalizing as the prehistoric period may be for modern bacteriology, we need to return closer to the present, to the early written records to more fully appreciate the disease in its human terms. Modern bacteriological reinterpretations may provide greater certainty in retrospective diagnosis
but they cannot rewrite the recorded experiences of those who coughed, sweated, and bled from their lungs, suffered disintegrating bones and joints, and shrinking of the flesh. These people explained and experienced such phenomena in different, non-bacteriological ways, which made sense to them. It is time to give up the specificity of the mycobacteria for a while and begin to speak the language of phthisis, consumption, and the humours.

Phthisis: All-Consuming Consumption in the Ancient World

The understanding of disease in the ancient world was fundamentally different from ours, varying both over time and among the diverse cultures of Asia and the Mediterranean basin. The belief that disease was divinely inspired and treatable through supplication of the gods was a shared characteristic. Beginning in the classical period in the 5th century BCE such celestial explanations were challenged by a new naturalism. Temple cults of healing continued, but various groups devised secular explanations for health and disease. The physicians who collected around Hippocrates of Cos (c.460–c.370 BCE) introduced humoral medicine. Based on a theoretical configuration of body fluids, it was rooted in the dominant philosophy of matter. Everything in the sublunary world, including the human body, was made up of the four elements—fire, water, air, and earth. In a healthy body the humours were in a state of balance, but during illness this was upset and the goal of treatment was to restore it where possible. Humoral medicine gained the ascendancy over its competitors and lasted a very long time—effectively until the 18th century in the western medical tradition—largely
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thanks to its greatest exponent and most efficient codifier, Galen of Pergamon (129–c.210 CE).

From the sometimes contradictory Hippocratic writings (not all written by Hippocrates himself) Galen distilled a wonderfully neat scheme of four humours—blood, yellow bile, black bile, and phlegm, which he linked with the four elements and the four qualities—hot, dry, cold, and wet. There was also a time component. The humours followed the seasons of each year and people passed through the same sequence during their lifetime. Thus spring and childhood were dominated by hot, wet blood: winter and old age by cold, wet phlegm. The mutability of illnesses, an important part of humoral pathology, could be explained in part by these progressions.

An individual had an exclusive humoral blueprint and his or her experience of ill health was correspondingly unique. There were diseased individuals rather than diseases, but since there could be only so much variation along the assorted continua of humoral medicine’s framework, distinctive patterns could be distilled by observant doctors into pen-portraits of disease. What we know about the disease experience in this period is essentially dominated by these texts, which articulated the likely cause, course, and outcome of the illnesses the Greeks recognized.

There was also advice on treatment. Determinedly holistic, humoral therapy was concerned with rebalancing the humours and regulating the six non-naturals: air (surrounding atmosphere), diet, exercise, sleep, wastes (excretions), and emotions. Preventive medicine aimed to maintain an individual’s ideal humoral balance within the body and its relation to the world via the non-naturals. Rebalancing of the humours called for the application of opposites: allopathy. Cooling remedies might be
advised for a fever, the result of an excess of the hot, wet humour—blood. Reduction of the fire in the body, by removal of the humoral excess—letting blood—could be employed. A natural bloodletting—haemorrhage—was therefore not always considered a bad sign. Such active intervention was tempered by the Greeks’ belief in the healing power of nature: sometimes it was better to do nothing but care for or nurse the patient. Accurate prognoses, a significant feature of these texts, helped doctors manage their cases and prepared patients for what they could expect.

 Appropriately sifted, there is sufficient evidence in the Greek canon for the familiar Orwellian symptoms of coughing, haemoptysis, fever, and weight loss and for much of the range of non-pulmonary symptoms that he was spared including the dissipated miliary form of the disease (comparable material has also been extricated from the Chinese, Ayurvedic, Babylonian, and Assyrian texts). It is therefore possible to extrapolate backwards and apply modern knowledge to ancient texts, constructing with some certainty retrospective diagnoses of, say, pulmonary tuberculosis (perhaps with gastric sequelae); Pott’s disease of the spine; tubercular lymphadenitis, or scrofula. All the while we are aware that these are members of the tuberculosis family, each caused by the same infectious agent working on different parts of the body; different manifestations of the same disease. This has no meaning for the Greeks. So what did wasting and haemorrhaging from the lungs, a deformed spine, and chronic, perhaps suppurating, masses in the neck mean some two millennia ago?

 We know from the Hippocratic corpus that the Greeks suffered from phthisis. By the time these texts were composed, phthisis was sufficiently widespread to be included in the
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Epidemics. The title referred less to those explosions of infectious disease we know as ‘epidemics’ than to those that were commonly present everywhere—ubiquitous, established diseases. Such an entrenched position meshes with what we know from etymology. The word that became phthinein as the ancient Greek language developed during the second millennium BCE, had arrived in the Balkan Peninsula with the Indo-European invaders, towards the end of the third millennium BCE. A general word for waning, in its medical garb it came to mean a chronic wasting away (especially the muscles), atrophying, being consumed, declining, rotting. All described what happened to the body of the phthisic (a person suffering from phthisis).

An emphasis on slow bodily consumption—as in the melting away of the flesh or using up of the body—was not restricted to medical texts. In Homer’s Odyssey—the epic telling of the hero’s return from Troy—the poet referred to a ‘grievous consumption’, which took the soul from the body; and to one who ‘lies in sickness…a long time wasting away’. Aristophanes (4th century BCE), among others, used ‘consumptive’ as a term of abuse in his play The Assemblywomen. Wasting diseases, atrophy of the bones and flesh were afflicts the Israelites faced, as recorded in the Old Testament books of Leviticus and Deuteronomy. Again etymology is helpful: schachepheth—the ancient Hebrew word for a wasting disease; its derivative—schachefet—the contemporary Hebrew term for tuberculosis.

It is reasonable to suppose that such references to wasting included Greek phthisis, which in turn may have included modern pulmonary tuberculosis. However, they also incorporated a disparate range of conditions that exhausted and drained the body of its life and flesh. Illnesses that heightened the body’s metabolic demands and/or suppressed the appetite, especially
over a prolonged period—anaemias, cancers, diabetes, low-grade infections—may have been covered by this umbrella term. Consumption could also refer to the dissolution of specific parts of the body: spines, hips, larynxes, and kidneys. If consumption thus compels a breadth of meaning, phthisis itself was not a single disease. Medical authors in the classical period listed the various types of phthisis they recognized, their origins, and how they were differentiated from other diseases. The overlap with pulmonary tuberculosis is extensive but not exhaustive, as samples from the Hippocratic corpus illustrate.

Book I of Diseases recorded three kinds of phthisis. The first was linked to a previous empyema—a collection of pus in a body cavity, especially the pleural cavity surrounding the lungs—and an excess of the cold, wet, watery humour phlegm. Patients who suffered an inflammation of the lungs, and could not rid themselves of superfluous sputum and phlegm, developed an ulceration there. Choking and difficulty in breathing, typically using only the upper part of the chest, followed. Eventually the overabundant phlegm closed the respiratory passage and death ensued. An empyema might also occur when excess phlegm fell from the brain (the organ identified with this humour) to the lungs. While the patient noticed a slight cough, somewhat bitter sputum, and an occasional, moderate fever, the lungs became increasingly irritated and ulcerated as the phlegm adhered to the tissues, and purulence set in. Heaviness of the chest, sometimes pain (perhaps extreme to the front and back of the thorax), and a heating of the body came next. A raised temperature attracted the phlegm from the rest of the body in a bid to cool the lungs. In health, the lungs served to cool the blood with which the inspired air was mixed; in phthisis this function was impaired. With perceptibly thicker and more putrid matter in
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the lungs, phthisics expelled this as best they could by coughing or ‘vomiting’ it up, but the body was now in a dreadful loop. The more purulent and copious the matter, the higher the temperature rose and the greater the frequency and violence of the cough. Patients lacked appetite and the wasting of the flesh was stark, but so long as they could continue to expel the putrid matter there was hope. If phlegm’s downward tendency spread to the bowels, digestion of what little food could be taken was disordered and death followed.

The second kind of phthisis came on after a burst blood vessel. The blood, transformed into purulent matter, caused ulceration in the lungs and a similar course unfolded. Herodotus, the 5th-century BCE historian, perhaps described such a case when he told of the demise of Pharnunches, riding out in haste from Sardis to meet his command under the Persians. Thrown from his horse, he began to spit up blood and ‘the disease turned into consumption’. The third kind resulted from an acute inflammation of the pleura or a chronic localization of phlegm and blood in the pleura, both of which could lead on to suppuration, ulceration, and a subsequent full-blown phthisis if they lasted for longer than forty days.

In this case, teasing apart the various lung conditions (including dyspnea or difficulty breathing), and determining how they might mutate, challenged the prognostic skills of the Hippocratic doctors and their successors. Help in this task could be found in Diseases, book 2, which delineated the *habitus phthisicus*. That particular people—in this case the tall and thin—tended towards certain diseases—here to phthisis—as a result of their constitution and general physical appearance was part of the holistic conception underpinning humoral medicine. This was a predisposition, not a directly inherited condition, however.
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Besides the general body shape, with its long neck, sloping shoulders, and poorly developed chest region, doctors were to look for a reddish hue in the hollows of the eyes, red cheeks, swollen feet, bent finger nails with the pads of flesh lost beneath, emaciation, languor, debility, and hair loss. Listening, they would expect to hear persistent crepitations or crackling during breathing; coughing; and a dull, hoarse voice, perhaps when the patient spoke of pain in the breast and back. When the patient expectorated the sputum was thick, yellow-tinged, and sweet to the taste. This excessively moist, hailstone-like purulent matter gave off a detestable stench when thrown onto the fire. (A useful test: doctors would be throwing sputum onto hot coals for at least the next two thousand years.) Bloody material might also be brought up with the sputum. Concomitantly disturbed bowels and a general swelling below the diaphragm were exceptionally bad signs. Elsewhere reference is made to highly characteristic wing-like shoulder blades; fever with chills; both extreme perspiration and the need to urinate. Much does seem familiar of late-stage pulmonary tuberculosis but it’s not an exact fit.

The text *Internal Affections* also listed three kinds of phthisis. The first arose—familiarly now—from an excess of phlegm falling downwards from head to lungs. The second was due to exhaustion and excess: in young males this often referred to venery. The third involved the sufferer initially becoming black and swollen with yellow-tinged skin under the eyes, before a pattern of sputum, cough, fever, and wasting emerged. Patients usually lived for a year with the first form, three years with the second, and as long as nine with the third, when in a wasted state life might yet continue, but the prognosis was very poor. There was therefore much for subsequent medical authorities
to work upon. Each tinkered with these composite pictures of phthisis in the light of their own experiences, placing due emphasis on this or that aspect of the consumption and the nature of the original disturbance in the body. Aretaeus of Cappadocia’s (fl. ?50/100–200 CE) causal triumvirate were abscesses in the lungs, a chronic cough, or haemoptysis. Whatever the details, the causes and course of phthisis were always in the plural.

Galen, the master systematizer of Roman times, ordered, organized, amended, and added to his inheritance of medical knowledge. From a modern vantage point, seeking a continuous trajectory over time, one must look under a multiplicity of Galenic headings—Phthisis, Tabes, Marasmus, Atrophy, Phthoe, Phyma, Struma, Haemorrhage, and Hectic Fever—if we want to piece together his understanding of a range of conditions that have been interpreted as tuberculous. For Galen these were separate, if sometimes linked, illnesses. His bequest of the modifier ‘hectic’ for the fever of phthisis is worth picking out. He described this as a specific disease, which also had the potential to become a phthisis. In contradistinction to other fevers (all of which were diseases, not symptoms of something else, at this time) the heat in a hectic fever was evenly distributed over the body—making it difficult for the patient to detect, the body feeling the same to the touch everywhere—and it did not increase. There was little cough and nothing came up if coughing occurred. Should marasmus (literally withering) occur along with the heat of a hectic fever then phthisis was the likely diagnosis. The seeds of the ‘hectic flush’ or a ‘hectic’ beloved by Romantic aesthete male poets and delicate swooning ladies had been sown for the future.

The pathologist Galen commented on the presence of phûma or tubercles in phthisics. He was not the first to see these small hard swellings in their lungs and preached the accepted wisdom
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that these were the result of the bodily disturbances of phthisis, not its cause. In a phthisic’s body coction or ‘cooking’ of the body’s juices, such as the excess phlegm in the lungs, was impaired. Tubercles were far from being an exclusive feature of phthisis, however, for this was a general term for hard tumours, which could be abscesses, cancers, water-filled cysts. They occurred in conjunction with a gibbosity or humped spine and its attendant deformities of the associated bones and flesh of the chest. The Greeks knew lung tubercles were found with spinal curvatures, but did not relate the two other than to see their formation as flawed coction in both cases. Bent spines may have been a potent inspiration for the hunchbacked figures in Egyptian and Greek art, but we can be sure of only what was left to posterity not what it represented.

Less dramatic but also easily visible in the living were swellings on the neck: struma or strumous disease. Just as ‘consumption’ was a general term for the wasting of the body and phûma for tumour, struma implied a tendency to preternaturally hard glands or to a swelling of the glands of the neck and ears. Again there is no neat overlay with the specificity of tubercular lymphatic glands or the more nebulous medieval scrofula. It is reasonable to suppose that among the possible if rare cases of goitre in the Greek world and the chronic inflammations such as mastoiditis, some of these swellings were tuberculous. Rich, inclusive categories of disease functioned in this highly individualized medical cosmology.

What to Do with the Phthisic Patient?

As is so often the case today, Greek texts urged early intervention for the best results. They also cautioned that full-blown
cases were likely to end in death. The Hippocratic author of *Internal Affections* knew one way to classify phthisis (in retrospect) was how long his patients survived their symptoms. Nevertheless, doctors did what they could, offering a range of dietary counsel, drugs, lifestyle advice, and surgery. Given the individuality of disease, the more options the better for rebalancing the body. Doctors were required to unite knowledge of therapeutics, past experience of similar afflictions, and their understanding of the patient. Blindly following empirical prescriptions was a greatly inferior medical art.

A change of air or atmosphere could help. This was no two-week pick-me-up in the sun, but the sombre search for a dryer, lighter climate with gentle, favourable winds. Egypt or Libya were popular in the Roman period because they necessitated long journeys from Italy and getting there could in itself be curative. Sea voyages were considered intrinsically healing because of the motion of the boat, including the nausea (purging) and exposure to the sea air, particularly when patients were spitting up blood. If this was beyond the patient’s strength (or purse), a sojourn to the nearest coast should be attempted or perhaps a gentle trundle by the sea in a litter could be tried in a severe decline. Should the patient be moribund, the nature of air in the bedroom must be assessed and ventilation keenly attended to.

A change of diet was also recommended. If consumption wasted the body, then according to allopathy, a phthisic’s diet called for replenishment. The line between diet and drugs, predominantly herbal at this time, was fluid. Since proper cooking of food in the stomach was an essential component of health, ordinary dietary components—milk, eggs, meat, and wine—took on a more medicinal character under doctor’s orders. Milk from various sources—wolves, asses, cows, goats, human
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breasts—was especially favoured: all to be taken as fresh as possible. Liquid preparations, taken as drinks, might be hyssop and fleawort boiled in sour wine. More solid preparations included combining horehound, pine nuts, parsley, and pepper with honey in a variety of ways. The fumes of ivy could be inhaled. Galen advocated a pound-weight of the mountain squill plant to be steeped for thirty days in strong vinegar, the resulting potion to be taken early each morning when patients had to be brought back from the point of despair. The skill was to know what to give and when.

Bathing was significant—forming in part a useful means of applying external medicines. Myrrh oil infused with a potion of lupines could be applied to the feet, removed, and replaced with butter, the procedure to be repeated three times to draw down the humours. Exercise should be gentle and overexcitement avoided. A successful doctor would also strive for the involvement and commitment of the phthisic to their treatment regime. The patient must remain positive—how else could the doctor be expected to try to combat the inevitable ups and downs during the course of a chronic disease?

Surgical interventions tended to be tried after the diet and drugs had been given a go. If a patient was spitting blood—evidence of excess—venesection, or bleeding, was recommended. Letting blood removed the surplus and cooled the body, helping the impaired lungs do their job. Fasting did the same but while less drastic it was less immediate too, and had to be balanced against the propensity to wasting. Acacia, plantain juice, or other herbal drugs thought to lessen the blood’s overabundance might be tried. If the application of glowing hot irons to the chest in order to dry up the superfluous cold and moist humours was too severe, gentler poultices and purges might be tried to
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the same ends. Modulation of the possible, to meet the needs of the individual, was key.

The diversity and abundance of remedies can be read as evidence for the generalized nature and frequency of consumption. But knowing exactly who and how many suffered from it in antiquity is much harder. One can of course return to the modern analysis of bodily remains using the increasingly sophisticated sampling techniques, but this only answers questions about the epidemiological profile of the much more specific idea of tuberculosis. Qualitative evidence for consumption, while equally bitty, is certainly poignant. The 3rd-century BCE Smyrna funerary stele recounting the long-running misery endured by a 4-year-old child afflicted first in the testicles, then the feet, and finally the intestines—‘I, doomed to a sad end… have left the hated consumption as an heritage to those who begat me’—reminds us that not only adults were affected. 12 The Greeks knew that those aged between 18 and 35 were most at risk and included women who were thought to be particularly vulnerable during pregnancy or after amenorrhea—the retention of blood being another potential cause of bodily unbalancing. Such patterns held for centuries—until the 20th century in effect.

There was a firm awareness that consumption could occur in groups of people. What happened apparently was a shared exposure to the correct external conditions of those in a population who had the necessary constitutional predisposition. More directly—but not by way of a living infectious agent—close contact with phthisics could result in a healthy individual developing a phthisis. The risk increased if the victim had stinking breath: an unpleasant and frequent occurrence in those with ulcerated, disintegrating lungs but also other late-stage,