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# Black lungs in the general population: a new look at an old dispute

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Almost from the time that autopsies were first routinely carried out, darkening of lungs with increasing age was described. Different explanations for the origin of the accumulating black pigment arose and by the early nineteenth century three hypotheses had emerged: 1) soot inhaled into the lungs from the air; 2) carbon accumulating in the lungs from abnormal pulmonary carbon dioxide metabolism; and, 3) pigment derived from the blood. In 1813

the English physician and chemist George Pearson published a paper in which he described the recovery of the black pigment from lungs and its chemical analysis. Pearson declared the black pigment to be airborne carbon/soot from the burning of coal and wood. He described these particles depositing in 'black spots' in the terminal airways and accumulating in the peribronchial lymph nodes, forming 'black glands'. Despite Pearson's prescient account, debate continued and the true explanation, given in that paper, was not fully accepted until the late nineteenth century.

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## Introduction

Black pigmentation of the lungs at autopsy in urban adults, in contrast to the pink lungs of children, was a commonplace observation in Britain until recently, allowing pathologists to make informed guesses as to the area in which the deceased patient had lived. The intensity of the blackness also was known to indicate the duration individuals had lived in that area. However, in the early days of pathology the origin of this pigmentation was a subject of dispute. As autopsies were increasingly performed through the eighteenth century, it became evident that the general colour of the lungs changed with the patient's age, from red or pink in childhood, through various shades of grey to a darker hue with advancing age.<sup>1–3</sup> The blackening was initially called melanosis, a descriptive term reflecting ignorance of the origin of the pigment. The most influential figure in medicine in this period, French physician and anatomist René Théophile Hyacinthe Laennec, seems initially to have confused two separate conditions, common melanosis and the uncommon lung tumour, metastatic melanoma. Laennec described black pulmonary matter or (*matière noire pulmonaire*)<sup>1</sup> and claimed that this material was found to some extent in all adults, very markedly in older people. This black pigment was distributed throughout the lung tissue and peribronchial lymph nodes in a more diffuse manner than in secondary melanoma, which was encapsulated in discrete lesions. However, at this time

the pigment was taken to be the same in both conditions, leading to speculation as to its nature.

## Origins of the black pulmonary matter

Eventually metastatic melanoma was recognised as being sufficiently anatomically distinct from the diffuse darkening to be a separate entity and three hypotheses emerged by the early nineteenth century to explain this accumulation of the black pulmonary matter:

- Exogenous soot/carbon deposited in the lung during inhalation of particle-laden air.
- Endogenous elemental carbon secreted in the lungs during abnormal carbon dioxide metabolism associated with respiration and gas exchange.
- Endogenous pigment derived from blood.

The Italian 'father of occupational medicine' Bernardino Ramazzini, in his *De Morbis Artificum Diatriba* of 1713, was aware that inhaled dust could lodge in the lungs, quoting the Dutch anatomist Isbrand van Diemerbroeck (1672) and drawing attention to a stone mason who showed him very fine dust that had accumulated in an ox bladder hanging in his workplace.<sup>4</sup> He was, however, concerned with occupational exposures to stone dust rather than carbonaceous particles. The German physician and anatomist Samuel Thomas von

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Soemmering was amongst the earliest to suggest, in an essay, an exogenous origin for the age-related blackening of the lungs. He stated, in 1808, that it was 'soot ... particularly among common people who burn bad tallow or coarse-oil which can find its way into the bronchial glands only through the air passages'.<sup>5</sup> Although correct, this was conjecture with no experimental proof in support. In contrast, George Pearson in 1813 argued that the gradual blackening of the lungs with ageing was due to inhaling soot-laden air and, by extracting and analysing the black pigment, provided chemical evidence that the black material was indeed soot/carbon.<sup>2</sup> Pearson (1751–1828) studied medicine at the University of Edinburgh and was awarded an MD in 1771, going on to briefly study at St Thomas' Hospital in London. He then practiced medicine in Doncaster between 1777 and 1783, before moving to London where he was admitted as a licentiate of the Royal College of Physicians in 1784. Pearson then began a course of lectures on chemistry, materia medica and therapeutics in his home. He became a chief physician at St George's Hospital in 1787, where he remained for the rest of his working life. Throughout his life he showed a practical interest in chemistry across a considerable breadth of topics, publishing an English translation of a French chemical nomenclature, a monograph on the chemistry of Buxton Water and another on the nature of Indian steel, whilst also working on the composition of water. In the field of biochemistry, Pearson turned his attention to the composition of renal and bladder stones and the composition of bronchial secretions in a variety of conditions. He was best known, however, for his association with Edward Jenner and smallpox vaccination, where he did a considerable body of research and clinical work. However, the relationship between Pearson and Jenner that had developed, soured when Pearson claimed that he, not Jenner, had discovered vaccination. Following several clashes between Pearson and Jenner regarding Pearson's role, he was publicly repudiated in 1807. This controversy overshadowed the perfectly good research he had carried out on vaccination and he withdrew from the controversy, making no further claims about vaccination. Pearson died after a fall in his home in 1828.<sup>6</sup> After his paper on black spots and glands,<sup>2</sup> discussed at length in the present paper, he seems never to have published again on this topic.

Pearson's paper<sup>2</sup> is entitled 'On the colouring matter of the black bronchial glands and of the black spots of the lungs'. The 'black spots' are now known to be accumulations of particles in macrophages and in the interstitium around the terminal bronchioles at the centre of the pulmonary lobule (centrilobular). Figure 1 shows the cut surface of two different lungs fixed in 1950 and 1959, with black spots clearly present, an appearance familiar to pathologists of that era when urban air pollution was still high in British cities.<sup>7</sup> They are generally called macules if they are large enough to warrant the status of a lesion, as shown microscopically in Figure 2.

Pearson's use of the term 'black glands' refers to the peribronchial lymph nodes, to which particles that have crossed into the interstitium at alveolar level have been

carried (Figure 3).<sup>8</sup> Pearson first reports that 'As hath been repeatedly observed, the lungs generally become more dark coloured proportionately to their age'. He hypothesises that the black particles were derived from combustion and were present in the air: 'In the air it is suspended in invisibly small particles derived from the burning of coal, wood and other inflammable materials in common life'. He then suggests that these particles deposit in the lungs and are interstitialised: 'sooty matter taken in with the air at respiration and accumulated in proportion to the duration of life' and 'retained in the minute ramifications of the air tubes and conveyed by the absorbents to the bronchial glands'.

In his paper Pearson also noted the black marbling pattern of the pleural surface of the lung, comparing it to the reported pattern of the lymphatic vessels in the pleura, taking this as further proof of transport of particles through the lymphatics en route to the peribronchial glands. He states, 'When I compared the black lines and black net-like figures, many of them pentagonal, on the surface of the lungs, with the plates of the lymphatic vessels ... I found an exact resemblance'. This is shown in Figure 4, the pleural aspect of a lung preserved in 1934.

Finally, Pearson also carried out chemical analyses that were simple but persuasive as to the carbon/charcoal nature of the black matter. These were the early days of chemistry, the English chemist and physicist John Dalton having only very recently described his atomic theory. A suspension of the particles in water retained their black colour on treatment with muriatic acid (HCl) or nitric acid and on burning released a gas that was identifiable as carbon dioxide on the basis that it formed carbonic acid, then calcium carbonate in lime water, turning it milky. Some animal organic matter, lung tissue, was present as shown by the detection of 'empyreumatic oil', i.e. the products of the combustion of organic matter and trace amounts of prussic acid. Although post-dating Dalton's descriptions of atomic theory in 1808 and 1810 by a few years, Pearson made no effort to describe his findings in atomic terms. Finally he concluded:

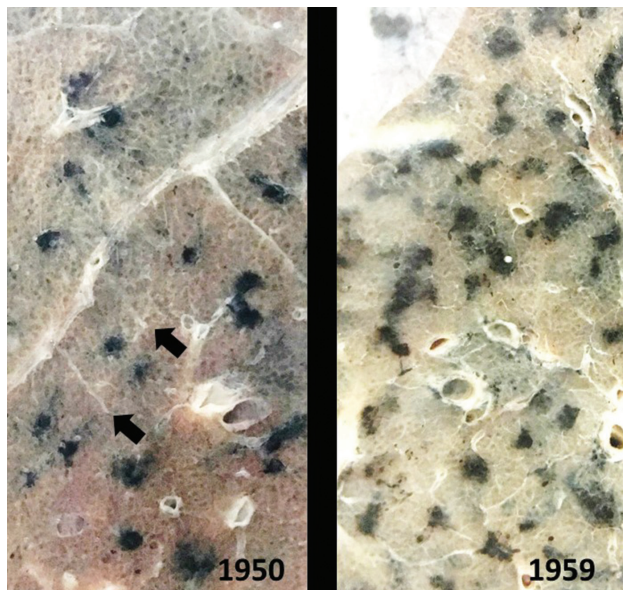
I declare the black matter obtained from the bronchial glands and from the lungs to be animal charcoal in the uncombined state i.e. not existing as a constituent ingredient of organized animal solids or fluids.

In other words it was soot breathed into the lungs.

## Anthracosis

Although the first to describe and explain environmental anthracosis, Pearson did not use the term, even though it has been wrongly suggested that he did.<sup>9</sup> In fact, he did not give any name to the condition he so clearly described. He described the dark pigment in lung as 'coaly matter', but this must be taken to refer to the colour and not the fact that it was coal; he did not deal with coalminer's lungs and his pigment was derived from air pollution. The term 'anthracosis', from the Greek for coal, was coined by

**Figure 1** Black spots. Cut surface of lungs showing ‘black spots’ from two lungs preserved in 1950 and 1959 (collection of the Museums of the Royal College of Surgeons of Edinburgh). The black spots are centrilobular accumulations of black particles and macrophages. In the 1950 lung, two interlobular septa are marked by arrows, indicating the centrilobular position of macules

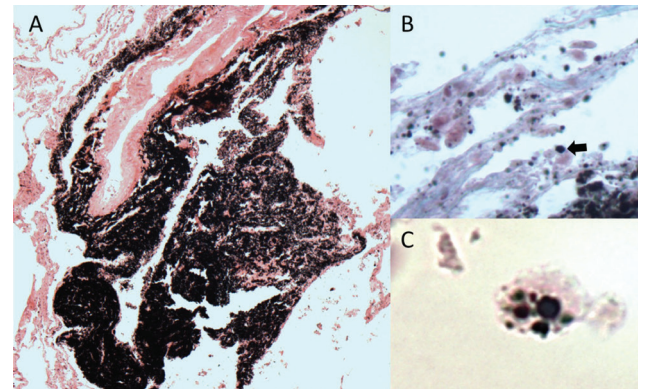


Thomas Stratton (1816–86), an Edinburgh physician who later became a naval surgeon and who considered it to be an appropriate term for the blackening of the lungs seen in coalminers.<sup>3</sup> He reserved the term anthracosis for the very black lungs of coalminers, which he differentiated from the progressive blackening of the lung with ageing and from melanoma. In fact, he specifically excluded the blackening of lungs by ambient particles in the general population from the term anthracosis. However, modern usage argues that carbon deposition from ambient air satisfies the term anthracosis. The blackened lungs of miners are now called coal-workers’ pneumoconiosis (CWP), whilst lungs blackened by carbon in air pollution are described as anthracotic.<sup>9–11</sup> The term anthracosis is used hereafter in this paper to describe the progressive blackening of the lung due to the inhalation and pulmonary deposition of ambient particles, principally soot, from indoor and outdoor air.

### The two alternative hypotheses for blackened lungs

The two alternative hypotheses for progressive blackening of the lungs in the general public are only of historical interest, reflecting as they do the understanding of chemistry at the end of the eighteenth and early nineteenth century. Scottish chemist and physicist Joseph Black had discovered the first known gas other than air in 1754, which he called ‘fixed air’, now known to be carbon dioxide. A decade later French chemist Antoine-Lauren de Lavoisier was able to show that oxygen was converted to ‘fixed air’ by animal respiration. However, it was not until the first decade of the nineteenth century that John Dalton described his atomic theory, so it is not surprising that, at that time, it was possible to speculate that the carbon accumulating in ageing lungs

**Figure 2** (a–c) Photomicrographs taken from a lung resected and accessed into the Museums of the Royal College of Surgeons of Edinburgh in 1822, showing an area of lung with a marked accumulation of black particles in the interstitium and around an airway. The age of the specimen and its poor state of preservation for histological analysis makes it difficult to ascertain much else

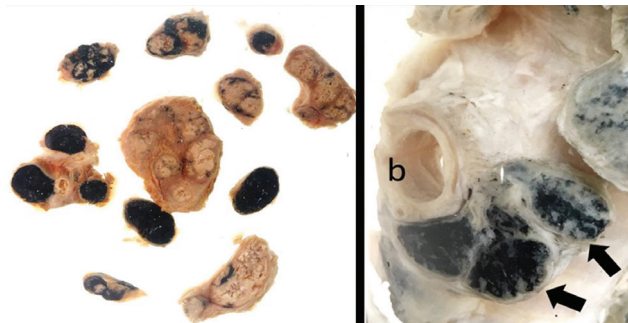


could be derived from carbon dioxide. The theory was that in pathological conditions carbon dioxide could ‘degrade’ to form elemental carbon that would deposit in the lung tissue turning it black. German anatomist Franz Daniel Reissessen in 1808<sup>12</sup> described this process as elemental carbon being ‘exhaled from the blood into the air cells’ and transported to the lymph nodes. American author George Rosen<sup>12</sup> states that, in French anatomist Marie Francois Xavier Bichat’s *Traité D’anatomie*, which was completed by his pupils Francois Buisson and Philibert-Joseph Roux 10 years after his death, this process is said to have occurred in the glands projecting into the bronchi which ‘secreted’ the carbon. Ferdinand Wilhelm Becker in 1826<sup>12</sup> supported Reissessen’s contention that, when there was an oxygen deficit, ‘decarbonisation of the blood’ in the lungs was deficient, resulting in laying down of the carbon in the lungs. Laennec<sup>1</sup> believed his black pulmonary matter was produced in the lungs of all people as they aged and was so common as to be not pathological. He and his compatriot Antoine Laurent Jessé Bayle<sup>13</sup> also thought the black matter might be related to tuberculous infection. British anatomist James Paxton<sup>14</sup> called the mechanism ‘insufficient combustion’, believing that the conversion of organic molecules to carbon dioxide during normal respiratory metabolism is incomplete, resulting in elemental carbon being formed. He also believed that in chronic inflammation, such as occurs in tuberculosis, sufficient disturbance to the normal alveolar function occurs that ‘the air and blood are no longer in contact, the extrication of carbonic acid ceases and free carbon is detained in the lungs’.

Armand Trousseau and Urbain LeBlanc in 1828 proposed a mechanism for the colour of various tissues based on altered blood pigment. By this argument the black pulmonary matter was just another site where altered blood pigment imparted colour to a tissue or organ, like hair and skin.<sup>15</sup> The influential Polish pathologist Rudolf Virchow and others proposed pathways for the formation of a family of pigments derived from the metabolism of haemoglobin, called variously haematin, haematoidin, etc.<sup>15</sup> They believed that these were



**Figure 3** Black glands. On the left are peribronchial lymph nodes from a patient who died of tuberculosis in 1909. Although enlargement and caseation typical of tuberculosis are seen in some nodes, especially those in the centre, other nodes have various amounts of anthracotic pigmentation. On the right is a dissection of trachea and bronchus preserved in 1831, showing anthracotic peribronchial lymph nodes (arrows), attached to a major bronchus (b)



formed in pathological conditions, such as extravasation of blood or coagulation, and that all endogenous pigment, including that in the lungs, was derived from haemoglobin. By 1866 American physician Francis Oppert, in a paper mostly considering occupational black lungs,<sup>16</sup> proposed a compromise, where the black matter might sometimes be a result of endogenous pigment and sometimes inhaled dust, as in the case of coalminers. He briefly referred to environmental blackening of lungs, but attributed this to natural pigment made in the lungs. Other authorities attempted to integrate the three hypotheses by suggesting that blackened lungs might arise from different mechanisms under different circumstances, exogenous particles providing the explanation in some cases of blackening, as in miners, but endogenous sources being the explanation in others.

### Resolution of the argument

A major stumbling block to the acceptance of Pearson's view of the extrinsic origin of anthracosis, which we now know was the correct one, was the resistance of Virchow, the single most influential figure in the development of modern pathology. Wedded to his theories on blood pigments, he did not believe that the blackening of the lungs over time in the general population was due to inhaled extraneous soot, nor even that the blackening of lungs in coalminers was due to inhaled coalmine dust.<sup>17</sup> With regard to the latter, Virchow had been sent preparations of coal miners' lungs by Swiss anatomist and histologist Rudolph Albert von Kolliker in the late 1850s,<sup>18</sup> who was at that time a guest of the anatomist, Dr John Goodsir and the surgeon Dr James Young Simpson, in Edinburgh.<sup>17</sup> By then, the mid-nineteenth century, Scottish physicians were advanced in their understanding of the role of the accumulated lung burden of coalmine dust in the development of disease in coalminer's lungs,<sup>19</sup> but Virchow remained unconvinced. In his paper on the pathology of coalminers' lungs in 1858, which described the preparations sent to him by von Kolliker and Goodsir, he wrongly concluded that it was 'extremely probable that we have here to do with pigmentary

**Figure 4** The pleural aspect of the upper lobe of a lung preserved in 1934. The marbling effect, 'black lines and net-like figures', alluded to by Pearson are evident. They arise because the lymphatics that outline the lobular structures at the pleural surface are picked out by the presence of black pigment



changes, resulting from extravasations of blood and the subsequent transformation of haematin, and not with an absorption and deposit of carbonaceous material inhaled into the lungs'.<sup>18</sup> It was not until particles of charcoal were incontrovertibly identified by German physician Ludwig Traube in human lung specimens in 1860, on the basis of their morphology,<sup>19</sup> that Virchow finally stated in 1866, 50 years after Pearson's paper, that: 'Traube's well-known case first convinced me that large fragments of vegetable coal can reach the alveoli and collect there ... The very characteristic shape of charcoal completely excluded any possibility of error and from that moment I was convinced that there is a true pulmonary anthracosis'.<sup>12</sup> The entity of environmental anthracosis was generally accepted by 1916, when Canadian pathologist Oskar Klotz could finally write: 'Admitting that but few individuals today can escape the accumulation of carbon particles in the respiratory system, it may be suggested that the condition should be looked upon as a normal process'.

### Airborne soot in the early nineteenth century

Pearson's view that blackening of the lungs with ageing was a result of the accumulation of inhaled ambient particles into the lungs implies that environmental air was polluted with soot at that time. In fact, particulate pollution was

high both outdoors and indoors in the early nineteenth century as a consequence of coal burning in multiple small factories, causing the well-documented smogs and the use of candles and oil lamps indoors. John Evelyn described the highly polluted, sooty nature of outdoor air in cities in the seventeenth century, a consequence of coal burning, in his pamphlet 'Fumifugium'.<sup>20</sup> This continued into the mid-nineteenth century as noted by James Johnson in his pamphlet 'Change of air',<sup>21</sup> in which he described London as having a 'dense canopy of smoke that spread itself over her countless streets and squares, enveloping a million and a half human beings in murky vapour'. There were no contemporary measurements of the extent of air pollution, although pictorial representations show the severity of the problem.<sup>22</sup> Indoor air was also very sooty since candles, lamps, wood and coal were all burned indoors for lighting, heating and cooking. Outdoor air pollution also permeates or is drawn indoors when fires are lit. British author and environmental chemist Peter Brimblecombe<sup>22</sup> has noted that smoky interiors caused considerable damage to hangings and paintings, books and leather chairs in the eighteenth century and that in 1725 the French Ambassador reported that hangings were quite uncommon in London interiors as they were so rapidly ruined by smoke/soot. Brimblecombe has described many published studies where, preserved by mummification, ancient lung tissue showed signs of blackening: 'Anthracosis is the rule rather than the exception in mummified lung tissue from Alaska to Peru'.

The introduction of chimneys would, by the mid-nineteenth century, have reduced much indoor pollution from coal fires, conducting the particles outdoors to join the ambient cloud. However, the introduction of mineral oil for lamps from the 1860s, unlike the tallow or whale oil previously used, would have produced indoor pollution that contained not only soot but also the metals associated with the mineral, usually paraffin refined from shale and later from liquid oil. Candles and cooking still produce carbon particles indoors,<sup>23</sup> but prior to the 1860s would not have contained much metallic matter, whilst the outdoor air would contain all the metallic elements associated with coal combustion.

## Conclusion

Pearson's prescient paper on 'black spots and black glands', in 1813, solved the puzzle of age-related blackening of the

lungs in the general population. In true post-enlightenment style, confronted with a black pigment of unknown origin in lungs at autopsy, he invoked empiricism, collected the pigment and carried out the chemical experiments that showed it to be charcoal/soot and, therefore, breathed in from environmental air. Nineteenth-century air, both indoors and outdoors, was loaded with soot particles from a range of sources. His descriptions of black spots and black glands predate any real physiological understanding of particle deposition and clearance, but these processes are now well understood. The centrilobular accumulations of particles (black spots) are now understood in terms of the deposition of particles elutriated to very small size by passage to the terminal airways. There they deposit in the centrilobular position, beyond the muco-ciliary escalator, at the point where air attains zero velocity and there is highly efficient deposition of these small particles by diffusion. The accumulating pigment in peribronchial nodes (black glands) reflects the interstitialisation of such particles around the terminal bronchioles, where the sumps of the lymph system draw them in, there being no lymphatic drainage distally in the alveolar septa. From this position the fraction of interstitialised particles that enter the lymph, drains cranially and is retained in the first nodes that it encounters, those in the peribronchial position. However, perceptive as Pearson's paper was, arguments for alternative explanations for the accumulating black pulmonary pigment continued well into the second half of the nineteenth century.

The issue of any adverse impact of anthracosis is too complex to be addressed in full here. However, in brief, increasing inhalation exposure to particulate air pollution, which causes anthracosis, is now known to have profound and large-scale adverse human health impacts. These are subtle and complex but include both worsening and increased mortality from chronic lung disease and coronary heart disease; there is also reduced lung growth rate in children.<sup>24,25</sup> In addition, there are clear links between exposure to fine ambient particles expressed as fine particulate matter centred about an aerodynamic diameter of 2.5 microns (PM 2.5), which contains the soot component, and the development of lung cancer.<sup>26</sup> The impact of these associations on longevity and health in the early nineteenth century are unknown, given the many conflicting risks that abounded in the era before the biomedical revolution, when average life expectancy was one-half of what it is today. **1**

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