The Geography of Disease in East Anglia

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This is a brief survey of various uncommon diseases that have been associated with East Anglia (comprising the counties of Norfolk, Suffolk, Cambridge and Huntingdon). In some of them the association has been due to obvious geographic factors, but in others no such factor has yet been identified.

HUNTINGTON'S CHOREA

In the case of Huntington’s chorea the association appears to have been due to chance. In 1630, John Winthrop of Groton in Suffolk led a party of young people from East Anglia to the New World. Among them were three men from the village of Bures whose first names were Jeffers, Nichols, and Wilkie. They all had the surname of Haste, and Nichols is known to have been baptised on January 14th 1594 as the ‘base sone of Mary Haste’. As there is no record that Mary Haste ever married, it is probable that these men were half-brothers.

Since that time there have been more than 7,000 cases of Huntington’s chorea in the United States among the descendants of these men, approximately 75 per cent of all the cases of Huntington’s chorea occurring in the USA among those of European stock. The other 25 per cent of cases are descended from a man called Welles who emigrated from Colchester in 1633. He had lived only twelve miles from Bures and, although there is no known relationship between him and the Haste family, it seems possible that they were related.

However, no cases of Huntington’s chorea in England have yet been traced to these sources, although in 1934 Macdonald Critchley discovered proportionately more cases in Suffolk than in other counties.

A coincidence regarding this disease is that George Huntington, who described all its essential features in 1872, was himself descended from an East Anglian family. Eight generations previously an ancestor had been a grocer in Norwich and had emigrated to the New World in 1633.

MALARIA

Although East Anglia has the lowest rainfall of any region in the United Kingdom, it has experience of several diseases that are in some way connected with water.
The first of these diseases is malaria. Seventeenth-century writers had connected a certain type of fever with those living in marshy districts. The term ague was sometimes used for this and other fevers. During the eighteenth century the increasing use of cinchona bark made it possible to distinguish malaria from other fevers. In the 1840s quinine became available and made the diagnosis of malaria more dependable. In 1863, the Medical Officer to the Privy Council ordered Dr George Whitley to survey the incidence of indigenous malaria. Whitley toured the country collecting clinical data from hospitals and medical men. In East Anglia he found evidence of malaria in the marshy and coastal districts of Norfolk, Cambridge, and Huntingdon but was told that the incidence had declined considerably in recent years. Only in the area around Peterborough was the disease still prevalent. In the preceding ten years (1854–1863) 2,827 cases of the ague had been admitted to the Peterborough Infirmary (Fig. 1).

Fig. 1. Indigenous malaria in England (after Whitley, 1863).
There was an appreciable mortality from malaria at that time but it steadily declined throughout the century and had reached negligible proportions in 1901 when Professor Nuttall and his associates compared the distribution of malaria as detailed by Whitley with the incidence of the anopheline mosquito. They discovered that the mosquito was present in many areas where there was no past record of malaria. The disease seemed to be related to marshy districts rather than to the distribution of the anopheline mosquito.

During the First World War soldiers who had contracted malaria abroad brought potential sources of the disease back to England. After the Gallipoli campaign many of the troops who had been victims of the disease were accommodated in areas where the disease had formerly been indigenous, in particular in camps along the Thames estuary. As a result there was a dramatic increase in indigenous malaria. In 1917, 235 cases were notified, and although the incidence later declined, there were 481 cases in the four years up to 1920.

Fig. 2. Indigenous malaria in England (after James, 1929).
As a result, the Ministry appointed a malarialogist, Colonel James, to investigate this epidemic.

The incidence of indigenous malaria then declined rapidly so that there were less than six cases a year in 1925–1929. In 1929, Colonel James published his paper, ‘The Disappearance of Malaria from England’, mapping out the distribution of indigenous malaria during the period 1917–1926 (Fig. 2) to show that this was essentially the same as that found by Whitley in 1863. The maps are slightly misleading since there were far fewer cases in the second period than in the first. In the Peterborough district the incidence had declined from more than 2,800 during 1854–1863 to only 29 during 1917–1926.

After 1929 there were very few cases of indigenous malaria. Since the Second World War there have been less than 50 cases in England, despite the number of soldiers returning from the East who had suffered from the disease. I can discover only one authentic case of indigenous malaria in East Anglia since 1945. This was a three-year-old child who contracted the disease while living on the edge of the Felixstowe marshes in 1947.

It appears that, in the United Kingdom, indigenous malaria has occurred only in marshy districts. In the last hundred years there has been a steady decline in the disease, except for a short period after the First World War. In East Anglia this decline has coincided with a steady increase in the drainage of the marshland.

**Well-water cyanosis of infants**

East Anglia is a rural area and has had to rely on shallow wells for much of its water supply. Pollution of these wells has caused disease of various types, one rare form of which was caused in this manner in Suffolk during 1951. Called ‘well-water cyanosis of infants’, the disease had previously been described in the USA. It is caused by using well-water contaminated with nitrates to make up dried milk feeds for bottled-fed infants. The nitrates are reduced in the baby’s bowel to nitrites, which are then absorbed and cause methaemoglobinaemia. During the early 1950s there were at least 16 such cases in East Anglia. The disease cannot be prevented by boiling the water, hence it was necessary to supply distilled water whenever it seemed likely that well-water might be used to make up babies’ feeds. A piped water supply has now reached the rural areas and the disease has disappeared.

**Stone in the bladder**

Another condition that is associated with water is ‘stone in the bladder’. Throughout the eighteenth and nineteenth centuries the population of Norfolk experienced a higher incidence of this condition than other areas of
Britain, a prevalence that was acknowledged at the time (Table 1) and led to the development of a ‘School of Lithotomists’ at the Norfolk and Norwich Hospital. This epidemic of bladder stones continued into the twentieth century, when it gradually declined. The Norfolk and Norwich Hospital still possesses the 1,453 stones removed by operation at the hospital during the period 1712–1909. Four hundred and forty-three of these stones were from children under 10 years old.

The local prevalence of the disease and its high incidence in children suggest that some local factor was responsible. Most of the lithotomists who worked in Norfolk advanced their theories as to its cause, suggesting features of the terrain, climate, diet or way of life. None of these theories seems convincing now because they offer no adequate explanation for the disappearance of the disease. Its cause remains a mystery.

**Plague**

East Anglia is bounded to the north and east by the sea. In the past, various infectious diseases have been brought into the region in ships from overseas and in the early years of this century an unusual epidemic was introduced in this way. In September 1910 four people in the village of Freston died in rapid succession from a fulminant bronchopneumonia. As the local doctor had never seen any disease like it, he asked two local consultants to assist him. As they failed to obtain bacteriological evidence from the first two cases they used rather more heroic methods on the last two. In addition to sputum specimens and blood cultures, they also obtained some blood-stained fluid from lung punctures. From all these specimens they grew *Pasteurella pestis*.

At first it was believed that this was an isolated incident occurring in a farm cottage about five miles from the nearest town, Ipswich. Thirteen days after the death of the last patient a rat and a hare from the same district were found to have died of the plague. As a result, an intensive investigation was carried

### Table 1. Incidence of bladder stone in hospital admissions (Dobson, 1779)

| Place     | Incidence |
|-----------|-----------|
| Cambridge | 1 in 1,650|
| Manchester| 1 in 557  |
| Newcastle | 1 in 287  |
| Norwich   | 1 in 55   |

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out in that district and was continued up to the First World War and for a short period after.

In addition to the four cases at Freston, it was discovered that there had been a local outbreak of pneumonic plague in 1906–1907 and one of bubonic plague in 1909–1910. Three further cases occurred: a naval rating at Shotley Barracks, in 1911, and two women in a pair of very isolated cottages, in 1918. In all, there were 23 human cases of plague in the period 1906–1918 (Fig. 3). Special surveys were organised to look for infected rodents. Between 1911 and 1914 infected brown rats were found in 25 districts comprising 22 parishes, two urban districts, and the County Borough of Ipswich. The area involved (Fig. 4) stretched from the Essex border to a point north of Woodbridge, a

Fig. 3. Distribution of human cases of plague, 1906–1918, in south-east Suffolk.

Fig. 4. Distribution of rodent plague, 1910–1914, in south-east Suffolk.
distance of 25 miles, and from the North Sea in the east to Ipswich in the west, about 12 miles.

Although infected rats were found over this wide area and plague bacilli must have been present within it for at least 12 years, only 23 human cases of plague occurred. Contrary to expectation, the disease did not behave like the great plague epidemics of the past. Instead it remained locally enzootic, involving human beings only occasionally and then probably as the result of accidental contact. This type of plague is called sylvatic or campestral plague and normally occurs in remote upland districts of the world where plague is always present.

The origin of this Suffolk plague was traced to grain ships in the River Orwell. It was established that ships from at least five plague-infested ports had arrived in the river in the period immediately prior to 1906.

**TETANUS**

East Anglia has experience of diseases such as tetanus, which are traditionally associated with a rural way of life. Unfortunately, tetanus was not notifiable until 1968; before that date its incidence could be estimated only from registered deaths and calculations based on its case mortality. It is believed that there were 150 to 250 cases a year in the United Kingdom in the period 1930–1955. As this gives an incidence of 3 to 5 cases per million of population per year, most physicians would have seen very few cases. At Cambridge, Dr Cole collected and recorded 43 cases between 1929–1940. From careful observations he was able to develop a technique for estimating the prognosis, based on his definition of the ‘period of onset’. Although larger series of cases have since been collected on a regional basis this method of estimating prognosis is still the best available. As shown in Table 2 the incidence of tetanus remained relatively high in the Cambridge area up to 1964. Recently, fewer cases have been notified in Cambridge whereas there have been 30 cases in the Norwich area in the last decade.

**Table 2. Incidence of tetanus in the Cambridge area**

| Locality              | Period   | Cases Total | Per year |
|-----------------------|----------|-------------|----------|
| England and Wales     | 1930–1960| 150–200 (estimated) | 4–8      |
| East Anglia (expected)|          |             |          |
| Addenbrookes (after Cole) | 1929–1940| 43          | 3.9      |
|                        | 1952–1958| 24          | 3.8      |
|                        | 1955–1964| 34          | 3.4      |
In a recent book on tetanus (Adams et al., 1969) the authors say that the disease is more common in those liable to injure themselves in rural areas where the soil is fertile and cultivated and the population of both man and animals is substantial. This description fits East Anglia, but the authors also add 'a warm climate and a low material standard of living' as further factors, which do not apply to East Anglia.

The principal epidemiological puzzle about tetanus in a rural area is why so few people contract the disease. Tetanus bacilli and spores abound, and all agricultural workers injure themselves with thorns, etc., yet less than 1 per 100,000 develop the disease.

**Gangrenous Ergotism**

Finally, there is one agricultural disease that has been recorded in Britain on only one occasion. This is gangrenous ergotism, which is caused by eating corn that has been infested by the fungus *Claviceps purpurea*. In 1762, 'six people lost their feet by a mortification not to be accounted for' in the village of Wattisham, Suffolk. There are several contemporary accounts of this episode, including a tablet in Wattisham Church, a written account in the Parish Register, and letters to the Royal Society. The diet, water supply and habits of the victims are known because Dr George Baker wrote on behalf of the Royal Society asking the Vicar of Wattisham pertinent questions on these subjects. In his reply the Vicar explained that the sufferers had eaten bread made from 'poor raddled wheat' which had lain on the fields for many weeks and had become discoloured.

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A Great Victorian
The spoken word, New York, 1862, during the Civil War. Young English doctor to recruiting officer: ‘I want to be a surgeon in your army.’ Officer: ‘If you can mend arms, come for examination tomorrow.’ Doctor retires to night of revision, then presents himself to examiner who says: ‘Strip’. Confusion on both sides. ‘But’, said the officer, ‘you wanted to be a sergeant, you must have a medical.’ Untangling differences in accent, the doctor serves as a surgeon, deeply distressed by the sufferings of the wounded. He leaves the service to become an actor, playing first with John Wilkes Booth whose brother Edwin assassinated Abraham Lincoln.

So started the theatrical career of that great actor-manager of the Criterion Theatre, Sir Charles Wyndham. Stage-struck son of a Liverpool doctor, he obeyed his father’s wishes, studying at King’s College in London and qualifying as a doctor in Dublin. His only spell of medical practice was in the American Civil War. When he came to found his own Wyndham’s Theatre in 1899 he gave the proceeds of the first performance to the Soldiers’ and Sailors’ Family Association. He never forgot what a war did to those who fought and their families.