

Guardian journalist named Best Cancer Reporter

The Best Cancer Reporter Award 2006, sponsored by the European School of Oncology, has gone to **Sarah Boseley**, health reporter for the UK's *Guardian* newspaper. She was commended for her 'thorough, balanced, informed and articulate' approach to covering cancer from a wide variety of angles. Below we reprint one of her articles, entitled: *Can you catch cancer?*

Within a few years, girls will be vaccinated against cancer. Not every cancer – at least, not yet. But the cervical cancer jab is well on its way.

There are currently 3,000 new cases of cervical cancer a year in the UK. A couple of shots in the arm, perhaps, and young women may never have to think about it again.

That is possible because cervical cancer is spread by a virus called HPV, or human papilloma virus. You can catch it by sleeping with somebody who has it, so women with more sexual partners are more likely to get it. The vaccine does not act against cancer per se, but protects against the virus which causes it. Which makes cervical cancer, effectively, an infectious disease.

Can you really catch cancer? And if cervical cancer is caused by an infection, is it remotely possible that we might also catch breast cancer, or prostate cancer, or bowel cancer? The answer

is yes and no. Certainly, catching cancers is not the same as catching a cold. Human papilloma virus may trigger cervical cancer, but many women infected with it will never develop the disease. There must also be other factors.

Where a virus is involved in cancer, it appears, it is one of many causes – a trigger in a chain of triggers. Along with the virus, there may have to be something in your genes that tips your chances of getting this particular cancer the unlucky way. Diet affects some cancers, alcohol others, smoking is an important risk factor and air pollution is under suspicion. But the remarkable and exciting thing about the involvement of viruses

in cancer is that they are a switch that can potentially be turned off. This is not a bad news story; quite the opposite. If an infection is involved in the onset of some cancers, then there is a way to stop them developing. Potentially, we could invent a vaccine. That is exactly what has happened in cervical cancer





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A good read. In her article *Can you catch cancer?* Boseley doesn't dodge the difficult science, but she also takes a balanced look at the hope vaccines can offer and explores the wider issues – such as parents who take a dim view of being asked to vaccinate their 10-year-old girls against a sexually transmitted disease and environmental campaigners who are unwilling to consider the possibility that cancer clusters may not always be caused by local radiation levels

and there is every reason to think that, one day, it may be possible in other cancers too.

We do not know to what extent viruses are implicated yet, nor in which cancers, but the estimate is that they may play a part in up to 20% of cases. The evidence is slowly accumulating. Just before Christmas, a paper appeared from Newcastle University that offered new evidence that minor viral infections such as colds, respiratory problems and mild flu might trigger childhood cancer. Richard McNally, an epidemiologist, had mapped outbreaks of two cancers – forms of leukaemia and brain tumours – in children under 15 over a period of 45 years from a tumour database in Manchester. He discovered clusters of children who were born around the same time and in the same place – and went on to develop cancer.

Whenever clusters of childhood cancers have been spotted, parents have understandably ascribed them to the man-made environment, assuming that fallout from a power station or radiation from a phone mast must be to blame. But McNally and colleagues have identified a pattern which is exactly like what you would see in infectious diseases.

“We found that place of birth was particularly significant, which suggests that an infection in the mother while she is carrying her baby,

or in a child's early years, could be a trigger factor for the cancer,” says McNally. “These could be minor common illnesses that are not even reported to the GP, such as a cold, mild flu or a respiratory virus.” But no, he hastens to say, you cannot catch cancer. His research suggests that infection is one of the factors in its onset, but it is not the only cause.

Instead, the hypothesis that his research helps to support is a double-whammy theory. Firstly, babies are born with a propensity to leukaemia. Mel Greaves, a professor at the Institute of Cancer Research in London, analysed the blood taken by midwives from the heel-pricks of newborns and found that many already have cell damage that could lead to the disease. But it is now clear that a second thing has to go wrong before a possibility becomes a likelihood. And that could be a viral infection.

This fits with the work of Leo Kinlen at Oxford University, who has been lambasted by anti-nuclear campaigners for his theory, first mooted in 1988, that childhood leukaemia is not the result of radioactive fallout and waste but caused by ‘population mixing’. Cancer clusters occur where whole groups from towns and cities have arrived to live and work in a remotish rural setting, he observed. Look at the oil fields, military installations, the building of new towns

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– and nuclear plants too. The incomers bring with them new viral infections, which could spark cancers among the native local population.

In fact, infections associated with cancer have been known for some time. There is a cat virus which causes leukaemia and a vaccine against it, causing people to wonder if there could be a parallel in human leukaemia. But the neatest example of infection as a significant cause is in stomach cancer. This is not triggered by a virus, but by a bacterium called *Helicobacter pylori*. That discovery netted a recent Nobel prize. “Fifteen to 20 years ago,” says Heather Dickenson, principal research associate at Newcastle University’s centre for health services research, “nobody would have taken seriously the theory that stomach cancer was the result of infection.”

Helicobacter pylori is a bacterium that enters the stomach in food and drink, but does not get destroyed by the acid there. Around 30%–40% of us are thought to be infected with it, and it can cause inflammation of the stomach lining, known as gastritis. In a small number of cases (about 3%, which means that other triggers such as diet or smoking have to be involved) that progresses to stomach cancer. But now we know that *H. pylori* is one of the guilty parties, many of these cancers (though not all) can be prevented. Give patients the right antibiotics, and *H. pylori* goes away.

Research into the links between cancer and viruses began around the start of the last century. In 1908, two Danes, Wilhelm Ellermann and Oluf Bang, identified a virus which they found spread leukaemia between chickens. In 1911, Peyton Rous in the United States found another chicken virus which caused sarcoma. The work was ignored for decades, but eventually won Rous a Nobel prize in the 1960s.

In that same decade, the first definitive link between infection and a human cancer was

established. A British scientist called Anthony Epstein, based at the Middlesex hospital, went to listen to a British surgeon called Denis Burkitt, who had identified what is now known to be the commonest childhood cancer in Africa. This was a tumour of the jaw that became known as Burkitt’s lymphoma.

In a remarkable piece of scientific detective work, Epstein mapped the incidence of the tumour across the wet, lowland areas of central Africa and realised he was looking at the malarial belt. He hypothesised that the cancer was caused by an infectious agent, spread by the malarial mosquito, and spent two years staring down an electron microscope attempting to find it.

He and his team had no luck until one tumour biopsy arrived from Uganda in an unfit state for microscopic examination. So Epstein cultured the cells instead. To everyone’s surprise, it grew a previously unknown form of herpes virus, which became known as Epstein-Barr. Epstein-Barr was later found in almost all samples of Burkitt’s lymphoma from Africa.

Almost everyone has this virus. “Ninety-five per cent of us are infected by Epstein-Barr,” says Lawrence Young, professor of cancer biology at the institute of cancer research in Birmingham. “It doesn’t cause us any effect at all. But with certain co-factors it could cause problems.” Malaria was a co-factor in Africa, which is why the pattern of incidence of Burkitt’s lymphoma matched the malarial regions.

The ultimate proof that a virus is a contributory cause of cancer is if you remove it, says Young. “Hepatitis B virus is associated with primary liver cancer. It’s very common in Africa and the Far East. About 25 years ago they introduced a vaccine for Hepatitis B in Taiwan where it had been a very common infection and you would see liver cancer in young adults. The incidence of liver cancer in the population has been significantly reduced.”



Public watchdog. In *The selling of a wonder drug*, also submitted to the award panel, Boseley takes a critical look at drug company marketing techniques. Over the years, she has helped readers build up an understanding of cancer from a medical, social, political and economic standpoint. More than 400 of her articles touching on cancer can be accessed on Guardian Unlimited, the *Guardian's* free Internet site, which is visited by more than 9 million readers



If you have Epstein-Barr and you catch malaria while on holiday, it does not mean you will develop Burkitt's lymphoma. None of this is quite that simple. You would have two of the risk factors – two possible triggers – but because this is mostly an African cancer, there is probably a genetic component involved too. Too little is known about the causes of cancer, for all the noise made about diagnosis and treatment. But if scientists can nail down a particular virus as a risk, they can interrupt the process that can cause disease and death. Young calls the virus “a link in the chain of events. This is not like catching a cold. You can't catch cancer as an acute disease. But if it is a vital link, you can break the chain.”

Epstein-Barr is also implicated in about half of Hodgkin's lymphomas, but not the other half. In China, Epstein-Barr is in nasopharyngeal carcinoma – but fascinatingly, the extra link in the chain is the salted fish in the Cantonese diet (and probably some genetic propensity as well). “We know because if populations from

China move to the west coast of America, in one generation they lose it,” says Young. “It's the changes in their diet.” Breast cancer, too, appears to have dietary links. The incidence in Japanese women who move to the US soars. “Diet is a major contributory factor to cancer,” acknowledges Young.

Diet we can change. Viruses and bacteria we live with, for the most part harmoniously as long as our bodies' infection-fighting systems are in good order. Epstein-Barr does most of us no harm unless our immune system is suppressed. In the early days of heart transplants, for instance, most patients died not because the heart gave out or was rejected, but of Epstein-Barr-associated lymphomas. They were being given massive doses of immuno-suppressant drugs, which meant that the virus was no longer kept in check, allowing the cancer to develop.

And in the early 80s, the first sign that we were in trouble from a new virus that would wreak havoc across the planet was the arrival of a new cancer in America called Kaposi's sarcoma. It

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had once been a very rare disease in elderly Jewish men from the Mediterranean. Suddenly, young gay men had it, as the HIV virus knocked out their immune systems, allowing the Kaposi's sarcoma herpes virus to flourish.

Viruses are now thought to be implicated in up to one in five cancers. As time goes on, we may find it is more. There are some controversial theories around. Papers have been written that suggest a monkey virus called SV40 is a trigger, with asbestos, for mesothelioma – a cancer of the lining of the chest wall, the abdominal cavity or the lining of the heart. Some have speculated that the monkey virus may have passed to humans through contaminated stocks of polio vaccine. Others are looking for a virus in lung cancer. In Australia, researchers are studying a human virus similar to one called MMTV which is responsible for mammary tumours in mice. They want to know if it could be implicated in breast cancer.

Finding any cause of cancer – even one that plays a small part – is very good news because it means prevention is possible. If a virus is involved, it opens up the possibility of a vaccine to disrupt the chain of events that leads to cancer. That is, in short, a holy grail. The revelations of the excellent results in trials of the cervical cancer vaccine were greeted with euphoria. Gardasil, manufactured by Merck, was 100% effective among the 12,000, mostly young, women who took part. It knocked out the two strains of HPV, 16 and 18, that are implicated in 70% of cervical cancers.

And the vaccine could prove even more useful. The trials showed that some of the other HPV types which are involved in a minority of cervical cancers were also stopped in their tracks. “Because there are beginning to be signs of cross-protection against other HPV types, [the proportion of cancers affected] could go up,” says Anne Szarewski, a clinical consultant

for Cancer Research UK who has been involved in the trial. She thinks the proportion of cancers affected could eventually be as high as 80%. At that point, the vaccine becomes more effective as a prevention tool than the cervical screening programme. As newer vaccines are developed, it is assumed they may hit the rest of the troublesome HPV types too.

So cervical cancer could, in theory, be wiped out, just as smallpox was. This is unlikely to happen, however, since it is only achievable if every girl and boy in the country has the jab. The vaccine is expected to be offered to sexually inexperienced girls who will not have HPV, aged around 10 to 13, but suggesting a vaccination for a young girl that will protect her from a sexually transmitted disease has not gone down well with parents. The Merck vaccine, unlike its GlaxoSmithKline rival, protects also against two types of HPV that cause genital warts. “It has proved a nightmare to promote in the States,” observed Szarewski. Trials in women over 25 who will have the virus – for most pick it up at some point – are only just beginning.

Cancer is the scourge of our times, the most feared disease of the 21st century. It appears to come from nowhere and kill at random. The more we know of the causes, the better we will be able to protect ourselves. At the moment, the best advice we have is generally to live well – to eat a lot of fruit and vegetables, drink in moderation, stop smoking and take exercise. But there are plenty of people who have lived unimpeachably healthy lives and died of cancer. Finding a silent trigger such as a virus that scientists may be able to knock out of the equation with a vaccine is not a reason to panic, but a cause for hope.

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