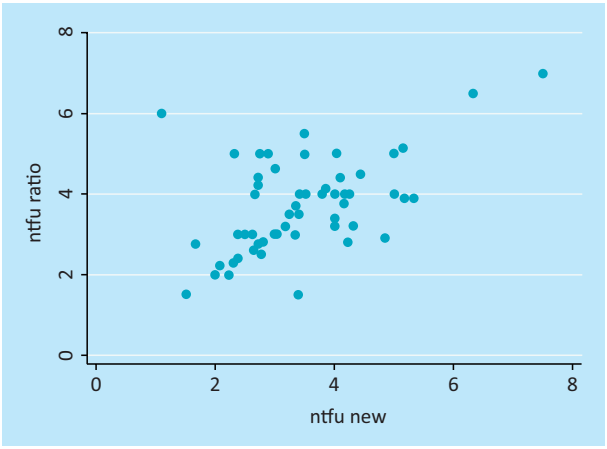

Fig 1. Correlation between actual calculated new to follow-up (ntfu) ratios and those submitted by the respondents.

1 Case-mixes varied markedly between individual units and teams. The type of service was different in different parts of the country, with some having a large soft tissue, neck and back workload, with others providing a service purely for inflammatory arthritis and connective tissue diseases.
2 Nursing input into the new to follow-up ratios varied from unit to unit. Some units provided a DMARD monitoring service with the support of their commissioners, while others moved this activity out into primary care.

The survey and presentation at the AGM uncovered the complexity and controversy of this area. To produce one-size-fits-all BSR-endorsed recommendations on new to follow-up ratios was seen to be counter-productive. The BSR could undertake work in individual diseases or conditions and produce recommendation on appropriate new to follow-up ratios for rheumatoid arthritis (RA), for example. This would involve a considerable amount of work and is still almost certainly controversial. A better approach is to give broad recommendations as per National Institute for Health and Clinical Excellence (NICE) guidelines, patients should have a minimum of specialist unit-led review, even if their disease is quiescent and stable.[3] The following further advice is also recommended:

1 Data are vital, if you can demonstrate that your follow-ups need following up then the argument that your practice is inappropriately reviewing patients can be dismantled
2 Call up your allies. Local GPs may not be aware of the impact of the workload of you discharging inflammatory arthritis back to them, and may be reluctant to take the additional workload and responsibility on.
3 Individual patients and representative groups (eg The Association of Residential Managing Agents, the National Rheumatoid Arthritis Society and Arthritis Care) may feel that their choice which is so much part of the government agenda, including the Next Stage Review[4] is being undermined by being discharged back to primary care.

This exercise has raised more problems than solutions, and the debate at the BSR AGM highlighted the controversies and complexities in this area and recommendations on new to follow-up ratios are still a long way off.

MARWAN BUKHARI
Consultant rheumatologist
Royal Lancaster Infirmary

JOSH DIXEY
Consultant rheumatologist
The New Cross Hospital, Wolverhampton

CHRIS DEIGHTON
Consultant rheumatologist
Rehabilitation Block, Royal Derby Hospital

On behalf of the Clinical Affairs Committee, British Society for Rheumatology, London

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The impact of obesity on cancers of the gastrointestinal tract

Introduction

The current obesity juggernaught affecting the developed world appears unstoppable and has serious implications on the health of the population. The consequences to cardiovascular disease are well appreciated, but the potential links with cancers of the gastrointestinal (GI) tract are less well known. This short review documents the biological mechanisms on how obesity may lead to cancer, the supportive epidemiological data and the potential impact the increased number of cancers will have on general practitioners, hospital doctors and public health physicians.

Biological mechanisms for the effects of obesity

There are several plausible biological mechanisms for how obesity may promote carcinogenesis including: hyperinsulinaemia, hormonal changes, increased inflammation and local physical factors. These may influence several pathophysiological processes including tumour initiation and progression. One of the more developed hypotheses suggests that increased insulin and insulin-like growth factor 1 (IGF-1) levels in overweight and obese individuals promote carcinogenesis.[1] Chronic hyperinsulinaemia is common in obese subjects and bioavailable IGF-1 is also elevated. IGF-1 is a potent mitogen and prevents cell death (anti-apoptotic effect) leading to increased cellular proliferation and

differentiation by stimulating DNA synthesis.[2] Furthermore, both insulin and IGF-1 down regulate sex hormone binding globulin (SHBG) resulting in an increase in serum oestrogens and androgens. Sex hormones are mitogens that may contribute to GI proliferation. Adipose tissue also up regulates inflammatory mediators including cytokines, such as TNF-alpha and interleukin-6 (IL-6). Adipose tissue, particularly visceral tissue, is a source of cytokines with serum leptin and IL-6 correlating well with increasing body mass index. While many GI cancers are associated with pre-malignant inflammatory states it is not yet understood how adipose-derived cytokines may influence this process. Of relevance is that TNF-alpha and IL-6 are potent mitogens. Finally, local physical factors may be important; oesophageal reflux increases with increased abdominal adiposity. The refluxate can promote Barrett's metaplasia and consequently adenocarcinoma.

Epidemiological evidence

The experimental work is supported by data from multiple epidemiological studies reporting positive associations between obesity and cancers of the oesophagus, pancreas and large bowel. For colorectal cancer, the most common cancer of the digestive tract, a meta-analysis of 31 epidemiological studies assessed the effects of obesity in 70,000 incident cases.[3] The estimated relative risk of colorectal cancer was 1.19 (95% confidence interval (CI) = 1.11 to 1.29) in obese people (BMI >30 kg/m^2) compared to those with a normal weight (BMI <25 kg/m^2). A dose-response existed with every 2 kg/m^2 increase in BMI (approximately 5 kg of extra weight) increasing the risk by 7% (95% CI = 4 to 10). If the association is causal, then a 2kg/m^2 reduction in BMI in the population would prevent approximately 2,600 colorectal cancers annually in the UK.[4] In pancreatic cancer, a tumour with an extremely poor prognosis, the effect of obesity was studied in a meta-analysis of 6,391 cases.[5] The summary relative risk per unit increase in BMI was 1.02 (95% CI = 1.01 to 1.03), as in a 2% increase risk for each unit of BMI. This translates for those with a BMI of over 30 kg/m^2 into a relative risk of 1.19 (95% CI = 1.10 to 1.29) compared to those with a BMI of 22 kg/m^2. A reduction of two units in the population's BMI would reduce the annual number of cases of pancreatic cancer in the UK by 300. For oesophageal adenocarcinoma, whose incidence has risen faster than any other cancer of the digestive tract, a meta-analysis of six case-control studies reported a 54% (95% CI = 39% to 71%) increased risk with each 5 kg/m^2 rise in BMI.[6] Based on these figures, a relatively large reduction in BMI of one standard category (5 kg/m^2) could prevent approximately 3,100 cases of oesophageal adenocarcinoma in the UK.

Conclusions

While the link between obesity and cardiovascular disease is well established there is now emerging evidence supporting a causal association with cancers of the GI tract. A more detailed understanding of the biological mechanisms is still needed to confirm the link, but the potential rise in these cancers should prompt action to encourage a reduction in the BMI of the population. A decrease in the BMI by two units may prevent approximately 4,000 cases of digestive cancers annually in the UK. This strategy would be of relevance to general practitioners and public health physicians to help prevent cancer, to medical and surgical gastroenterologists and oncologists to reduce the number of patients referred with cancer, and to managers who would need to deal with the increased resources required. Perhaps in this century, obesity will be identified as the main risk factor for cancers of the digestive tract, just as in the last, smoking was for lung cancer.

ANDREW R HART
Senior lecturer in gastroenterology
School of Medicine, Health Policy and Practice, University of East Anglia, Norwich

MICHAEL PN LEWIS
Consultant gastrointestinal surgeon
Department of Upper Gastrointestinal Surgery, Norfolk and Norwich University Hospital NHS Trust

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What is happening to English neurology: an update

In 2008, *Clinical Medicine* kindly published my analysis of the availability of neurology outpatients by primary care trust (PCT) of residence from 2003 to 2006.[1] The information was also given to the Department of Health (DH). I have extended this study to April 2009 and the trends are essentially the same. The number of appointments in neurology has continued to rise (by 11% per year, compared with 3% per year for general medicine) with no apparent ceiling. The follow-up to new appointment ratio has dropped from 1.6 to 1.46 over the same time. The lottery in regional access has not changed (Fig 1).

I recently approached the DH to ask for the agreed outpatient tariffs between PCTs and providers in secondary care. Neurology is unusual in that there has been no nationally agreed tariff; instead, local tariffs have been negotiated between hospitals and PCTs. The DH held no record of the agreed tariffs. Using freedom of information legislation I then contacted all 152 PCTs to ask to which providers their patients were referred and at what cost. In total, 104 PCTs responded, giving the tariffs for 121 providers in England; other PCTs used the exemption that the information was commercially sensitive. The information that has been made available