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Estimating age, sex and period specific constant tar cigarette consumption in the UK

In a paper published three years ago (September 1990;45:657–65) we examined trends in smoking associated respiratory diseases up to 1985 in relation to trends in cigarette smoking. In that paper we presented graphs for each sex of cumulative constant tar cigarette consumption (CCTCC) by age and year of death, and by age and year of birth. We were approached by the Lung and Asthma Information Agency for permission to use these graphs with the request that they be updated to 1990. While updating the figures an unfortunate error was noted in how the CCTCC estimates had been calculated. Although this did not significantly affect the estimates, or the conclusions of our paper at all, we are writing to present the corrected and updated CCTCC data (table). For each sex, period, and age group, CCTCC is an estimate of the average total lifetime number of manufactured cigarettes of constant tar smoked (using tar factors of 1 for 1961–65, 0·804 for 1966–70, 0·613 for 1971–5, 0·544 for 1976–80, 0·477 for 1981–5 and 0·423 for 1986–90). The CCTCC data are lifetime consumption estimates directly comparable with mortality estimates obtained by aggregating annual age specific data over five year periods. Details of the basic data from which the estimates were derived, and the precise method used to derive them (incorrectly described in our previous paper) are available on request.

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Alveolar atypical hyperplasia in association with primary pulmonary adenocarcinoma: a clinicopathological study of 10 cases

I read with interest the description by Dr FA Carey and colleagues (December 1992;47:1041–3) of nodules of atypical alveolar cell hyperplasia in 10 patients. The lesions as illustrated are identical to those I have reported.[1 2]

The authors make the point that their cases were found in formalin inflated lung but were not grossly apparent in most, if not all, instances. They further state that previous reports of such lesions, including my reports,[1 2] were based on lesions found in formalin inflated lung. In fact the great majority of cases in my series were inflated in Bouin's solution and that point was made in both papers. The issue is of importance since the lesions are not usually grossly apparent in formalin inflated lung but are easy to see in specimens of lung inflated in Bouin's solution, and this difference has been illustrated.[2] The inability to see these lesions grossly will result in an underestimate of frequency in prospective studies, and if the authors are planning such a study, I would recommend that inflation in Bouin's solution be considered.

Dr Carey and coworkers comment that it is premature to view these nodules as bronchioloalveolar cell adenomas rather than atypical hyperplasia since they have not been described except in association with carcinoma. I have, in fact, found such lesions in several resections for other types of primary disease (granulomas and metastatic sarcomas, for example), but I cannot understand how this is relevant to the issue of whether or not these lesions are neoplasms.

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1 Miller RR, Nelems B, Evans KG, Muller NL, Ostrow DN. Glandular neoplasia of the lung. *Cancer* 1988;61:1009–14.
2 Miller RR. Bronchioloalveolar cell adenomas. *Am J Surg Pathol* 1990;14:904–12.

AUTHORS' REPLY We accept Dr Miller's point that lesions of atypical alveolar cell hyperplasia are more easily identified macroscopically in specimens of lung inflated in Bouin's solution. There are, however, disadvantages to the use of this fixative, not least of which is the fact that it severely compromises nuclear DNA analysis of tumour material. Also, since formalin is widely accepted as the routine fixative of choice, a change to Bouin's solution for lung specimens is not likely to be widely accepted. It is, nevertheless, possibly true that inflation in Bouin's solution might be adopted in any prospective search for atypical hyperplasia in pulmonary resection specimens.

Cumulative constant tar cigarette consumption (in thousands) by sex, age and period

	Age													
Period	*15–19*	*20–24*	*25–29*	*30–34*	*35–39*	*40–44*	*45–49*	*50–54*	*55–59*	*60–64*	*65–69*	*70–74*	*75–79*	*80–84*
Men														
1901–05	1	3	4	4	4	4	3	3	2	2	1	1	1	0
1906–10	2	6	9	9	9	8	7	6	5	4	3	2	2	1
1911–15	3	9	14	16	16	15	13	12	10	8	6	5	3	2
1916–20	3	12	21	26	27	26	24	20	18	15	12	9	6	4
1921–25	4	15	27	36	39	39	37	33	28	23	19	14	11	8
1926–30	4	15	30	43	51	53	52	48	41	34	28	22	17	12
1931–35	4	16	32	48	61	69	69	65	59	49	40	32	25	19
1936–40	5	18	35	52	69	82	89	86	79	69	57	45	36	28
1941–45	5	20	39	58	76	95	107	111	104	93	79	64	50	39
1946–50	5	21	42	62	81	101	120	130	132	120	104	87	69	54
1951–55	4	18	41	64	84	104	124	142	151	150	132	112	93	73
1956–60	5	17	37	62	87	107	128	147	164	170	163	141	119	98
1961–65	6	20	38	58	84	110	131	151	169	183	185	174	150	125
1966–70	5	21	40	56	77	104	130	150	169	185	196	196	182	155
1971–75	4	17	36	55	71	93	120	145	164	181	196	205	202	186
1976–80	3	14	29	48	67	83	104	132	156	173	190	202	210	205
1981–85	3	10	23	38	56	75	92	113	140	163	179	194	206	213
1986–90	2	8	17	29	44	63	81	98	118	145	167	183	197	208
Women														
1926–30	0	1	1	1	1	1	1	1	1	0	0	0	0	0
1931–35	0	1	2	2	2	2	2	2	2	1	1	1	0	0
1936–40	1	2	4	4	5	4	4	4	3	3	2	2	1	1
1941–45	1	4	7	9	9	9	9	8	7	6	4	3	2	1
1946–50	1	6	11	15	16	16	16	15	13	11	8	6	4	3
1951–55	1	6	14	20	23	23	24	22	20	17	14	10	8	5
1956–60	2	6	14	24	30	33	34	32	29	25	20	16	12	9
1961–65	2	9	16	25	35	42	45	44	41	36	29	23	18	13
1966–70	3	11	19	26	35	47	54	56	53	48	40	33	25	19
1971–75	3	11	21	29	36	46	58	64	64	60	52	44	35	26
1976–80	3	11	20	30	38	45	55	67	72	71	65	55	46	36
1981–85	2	8	17	27	37	45	52	62	74	78	75	67	57	47
1986–90	2	6	13	23	33	43	51	57	67	79	81	77	69	58

We do not accept Dr Miller's contention that the lesions described in her series and ours ought to be considered as bronchiolo-alveolar cell adenomas. The lesions are generally characterised by nuclear pleomorphism and a high nucleocytoplasmic ratio—features not normally associated with a benign neoplasm. We feel strongly that these features are much more consistent with premalignant dysplastic lesions seen at other sites—including the bronchus—hence our preference for "atypical hyperplasia."

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BOOK NOTICES

Asthma in the Workplace. I Leonard Bernstein, Moira Chan-Yeung, Jean-Luc Malo, David I Bernstein. (Pp 664; $185·00.) New York: Dekker, 1993. ISBN 0-8247-8799-4.

This book is like a fruit cake: solid, full of goodness, digestible after a fashion, and mildly old fashioned. In it you will find a great deal about occupational asthma, yet some readers will remain disappointed.

Occupational asthma is of continuing interest because of its public health implications and because it provides a very useful model for the study of the mechanisms of asthma in general. Much is known about the frequency of occupational asthma in many working populations and this is covered well in a comprehensive chapter on epidemiology by Becklake. Too little is known about the impact of occupational asthma in the population as a whole and more discussion of this would have been welcome. Pathophysiological comparisons between occupational and non-occupational asthma are well covered in a chapter by Fabbri and others, but again these comparisons could have been usefully extended. The importance of evaluating exposure-response relationships is discussed too briefly in an otherwise excellent chapter on surveillance by D Bernstein. Other chapters discuss definitions, clinical matters, and environmental monitoring, and a comprehensive list of specific types of occupational asthma is included.

The four editors are all North American and between them they contribute to 17 of the 27 chapters. The parochialism of some of these contributions is irritating. The section on platinum asthma is, for example, little more than a repetition of the authors' work with no reference to studies carried out on this side of the Atlantic. The chapter on challenge tests describes a procedure very different from the more pragmatic approach used in Britain. On the other hand appropriate regard is given to surveillance systems and legal/compensation matters outside North America.

Many of those with a special interest in occupational asthma have already contributed to this book. Others who will find it useful—and it is undoubtedly a useful book—will be those with an interest in occupational health and in asthma in general, and those with a well funded library.—PC

Lung Vascular Injury. Arnold Johnson, Thomas Ferro. (Pp 368; $135·00.) New York: Dekker, 1992. ISBN 0-8247-8718-8.

This is yet another volume in the series entitled "Lung Biology in Health and Disease" which is rapidly becoming a classic of academic respiratory medicine. There are now over 60 books in the same series and a degree of overlap is hardly surprising. Volumes 24 and 50 were specifically devoted to the adult respiratory distress syndrome (ARDS) and this current work really updates the reader on progress in this important area.

Despite the fact that the mortality associated with ARDS remains depressingly high, research in this area has proved remarkably fruitful. The fashion of the 1980s for lengthy investigations of the pattern of fluid and solute flux through the damaged alveolar capillary membrane seems to be changing, partly through an appreciation by clinicians that, in many cases, lung injury represents only the pulmonary manifestation of a panendothelial insult. The recognition that the endothelium and smooth muscle act in concert to regulate both systemic and pulmonary vascular control, and the burgeoning literature concerning endothelially derived vasoactive products, are reflected fully in the nature of the contributions to this book. Thus, early chapters deal with transmembrane signal transduction and activation of phospholipases in the endothelium and the role of second messengers in the regulation of pulmonary vascular smooth muscle and permeability. There are several contributions touching on the relevance of the oxidant:antioxidant balance and a first class section on impaired vascular smooth muscle function in lung injury.

As in the rest of the series, the illustrations are clear. The referencing just about creeps into the early 1990s, which is probably about as up to date as could be expected. This is a worthy addition to a fine series, even if the current exchange rate puts the price out of reach for most individuals.—TWE

A Colour Atlas of Respiratory Infections. J T Macfarlane, R G Finch, R E Cotton. (Pp 130; £65·00.) London: Chapman and Hall, 1993. ISBN 0-412-38960-6.

This is a book with more than 300 illustrations, most of which are both educational and technically superb. About half reflect an interest in pathology; post mortem specimens are included. Of the 100 or so radiographs only the tuberculous empyema is not convincing and a pleural effusion, with mediastinal shift *towards* it, is a curiosity (figures 4.30 and 4.31 remain a mystery unexplained by the text). The clinical photographs are admirably instructive. However, the spots were difficult to see on the man with a *Varicella* pneumonia and I suppose that tuberculosis is not the only cause of cachexia in a 48 year old man. Red arrows to indicate the portals of infection might relieve the boredom of a sedated, ventilated man in intensive care. The cooling tower (*Legionella*) was worthy of the Tate gallery and parrots were light relief to explain the serious "Psittacines". What a shame that the most helpful figure (seasonal peaks of common respiratory pathogens) was not more glamorous.

The quality of the text was variable. The chapter on the causes of recurrent respiratory infections was excellent. However, guidelines to restrain the adventurous fired up by "Invasive techniques for the investigation of respiratory tract infections" would be helpful. The pot pourri promised by "Uncommon or geographically restricted respiratory tract infections" was a disappointment. The importance of tuberculosis was underplayed: its presentation in the immunocompromised patient would have benefited from an American input, the epidemiology was dated, a clear message on treatment was missing, and the BTS guidelines were misquoted. There were some contentious hidden messages. Is *Haemophilus influenzae* the main cause of infective exacerbations of chronic bronchitis? Is chloramphenicol no longer a good treatment for some respiratory infections? Is a CT scan necessary when an aspergilloma has been well demonstrated by a plain radiograph? Should a highly calcified tuberculoma really be excised?

This is a book for the library with funds to spare at the end of the financial year. I look forward to the second edition—GB.

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NOTICE

Eighteenth International Conference on Lung Sounds

The Eighteenth International Conference on Lung Sounds will be held at Lake Louise, Alberta, Canada on 25–27 August 1993. For further information contact Raymond L H Murphy Jr, Attn: Barbara Keith, Faulkner Hospital, Pulmonary Department, 1153 Centre Street, Boston, Massachusetts 02130, USA. Tel: (617) 522-5800, x1968. Fax: (617) 524-8663.