industry, was found to have a titre of 1:128 in sera taken on days 20 and 33 after the onset of a 'flu-like illness in March 1978 contracted in Majorca. The illness progressed to pneumonia and he was admitted to hospital in England six days after onset. Recovery was slow. By April 1979 his antibody level had dropped to <1:16. Using class specific antiserum, it was demonstrated that only IgM antibodies were present. No antibodies were detected against *L. pneumophila* strains 2, 3, or 4.

While the serological results do not entirely meet the presently accepted rigid criteria, the high specific IgM titre, the negative IFAT result after 12 months, and the clinical picture all support a presumptive diagnosis of Legionnaires' disease.

A third male patient, aged 63 years, became ill in December 1977 with IFAT titres of 1:32 in two sera taken three weeks apart in March 1978 and a third serum taken a year later. The patient who works as a printer's cutter has a 13-year history of chronic bronchitis.

The prevalence of Legionnaires' disease among patients with sporadic, otherwise undiagnosed, pneumonia was found in this study to be 0.6% (2/340) compared with 1.5% (21/2023) reported in a general population study carried out in Nottingham (Macrae *et al.*, 1979). Other workers have reported a prevalence of 1% (Foy *et al.*, 1979) and 4-10% (Renner *et al.*, 1979; Cohen *et al.*, 1979) in groups of otherwise undiagnosed pneumonias.

ALAN H. BALFOUR

JOHN P. HARFORD

Public Health Laboratory,
Bridle Path,
York Road,
Leeds LS15 7TR

References

- Cohen, M. L., Broome, C. V., Paris, A. L., Martin, W. T., and Allen, J. R. (1979). Fatal nosocomial Legionnaires' disease: Clinical and epidemiologic characteristics. *Annals of Internal Medicine*, **90**, 611-613.
- Foy, H. M., Broome, C. V., Hayes, P. S., Allen, I., Cooney, M. K., and Tobie, R. (1979). Legionnaires' disease in a prepaid medical-care group in Seattle 1963-75. *Lancet*, **i**, 767-770.
- McDade, J. E., Shepard, C. C., Fraser, D. W., Tsai, T. R., Redus, M. A., Dowdle, W. R., and the Laboratory Investigation Team (1977). Legionnaires' disease.

Isolation of a bacterium and demonstration of its role in other respiratory disease. *New England Journal of Medicine*, **297**, 1197-1203.

Macrae, A. D., Appleton, P. N., and Laverick, A. (1979). Legionnaires' disease in Nottingham, England. *Annals of Internal Medicine*, **90**, 580-583.

Renner, E. D., Helms, C. M., Hierholzer, W. J., Jr., Hall, N., Wong, Y. W., Viner, J. P., Johnson, W., and Hausler, W. J., Jr. (1979). Legionnaires' disease in pneumonia patients in Iowa. A retrospective sero-epidemiologic study, 1972-1977. *Annals of Internal Medicine*, **90**, 603-606.

Taylor, A. G., Harrison, T. G., Dighero, M. W., and Bradstreet, C. M. P. (1979). False positive reactions in the indirect fluorescent antibody test for Legionnaires' disease eliminated by the use of formalised yolk-sac antigen. *Annals of Internal Medicine*, **90**, 686-689.

Platelet aggregation and serum prolactin

We recently described enhanced platelet aggregation after insulin-induced hypoglycaemia (Hutton *et al.*, 1979). Increased adrenaline release was thought to be largely responsible for this effect but the influence of other hormones released during an insulin stress test could not be excluded. We have therefore examined the effect on platelet aggregation of stimulating the release of one of these hormones, prolactin, with thyrotrophin releasing hormone (TRH).

Seven patients selected in accordance with the criteria used in the previous study were given 200 µg TRH intravenously. There was no significant change in aggregation induced by either adenosine diphosphate (ADP) or adrenaline during the 2-hour test period, although all subjects showed the expected rise in prolactin (Table).

These results make it unlikely that prolactin alone was responsible for the enhanced platelet aggregation observed

during the insulin stress test and thus add weight to our suggestion that our earlier observations resulted from the synergistic effect of adrenaline released *in vivo* with ADP added as the aggregating agent *in vitro*.

Our thanks are due to Dr Jean Ginsburg for access to patients under her care, and to Dr Marion Gore and her staff for the serum prolactin assays.

R. A. HUTTON¹

D. P. MIKHAILIDIS²

E. GEORGIADIS³

A. S. J. BARKLEY³

Departments of ¹Haematology,

²Chemical Pathology, and

³Endocrinology,

Royal Free Hospital, London NW3

Reference

- Hutton, R. A., Mikhailidis, D. P., Dormandy, K. M., and Ginsburg, J. (1979). Platelet aggregation studies during transient hypoglycaemia. *Journal of Clinical Pathology*, **32**, 434-438.

Immunohistochemical demonstration of carcinoembryonic antigen

With reference to several recently published papers on the identification of carcinoembryonic antigen (CEA) in human breast carcinomas, mammary dysplasia, and granular cell myoblastomas using immunoperoxidase techniques and a commercially available antiserum to CEA (Shousha and Lyssiotis, 1978; 1979; Shousha *et al.*, 1979), we feel that some comment should be made regarding the specificity of the immunohistochemistry.

Many anti-CEA sera show cross-reacting activity against related glycoproteins such as non-specific cross-reacting antigen (NCA). These CEA-like materials are found in a large number of

Effect of TRH on prolactin level and platelet aggregation

Time (min)	Serum prolactin (mIU/l)	Platelet aggregation (%)	
		ADP	Adrenaline
Pre-TRH	207 ± 57	59.4 ± 7.4	63.9 ± 22.9
Post-TRH 30	1002 ± 420	52.9 ± 18.7	59.1 ± 15.1
Post-TRH 60	520 ± 218	49.3 ± 12.6	46.4 ± 19.6
Post-TRH 90	359 ± 153	55.7 ± 17.5	56.0 ± 22.5
Post-TRH 120	288 ± 140	63.1 ± 9.4	64.7 ± 24.2

cell types, including cells of the myeloid series, macrophages, and normal colonic mucosa (Burtin *et al.*, 1973; 1975). Similar cross-reacting glycoproteins are also found in breast cyst fluid and normal breast duct mucosa (Kuo *et al.*, 1973; Fleisher *et al.*, 1974).

In view of the widespread presence of these cross-reacting antigens, any claims that tumours contain CEA based on studies using anti-CEA sera must be viewed with caution unless attempts have been made to absorb out unwanted activity against these related substances. The specificity of the immunohistochemical reaction cannot be proved solely by abolition of specific staining after absorption of the anti-CEA serum with CEA since this procedure will also tend to remove the cross-reacting anti-NCA activity (Burtin *et al.*, 1973). In this connection it is perhaps noteworthy that Shousha and Lyssiotis (1979) state that absorption of their anti-CEA serum with CEA before use for immunoperoxidase staining diminished staining but did not abolish the reaction. Pretreatment of tissue sections with periodic acid has been shown to remove unwanted cross-reacting antibody activity with unknown tissue antigens (Isaacson and Judd, 1977), but the antibody used by those authors was already free from anti-NCA activity.

We do not wish to deny that breast carcinomas and possibly granular cell myoblastomas contain CEA but we feel that care should be exercised in the interpretation of results using commercially available antisera unless unwanted cross-reacting activity has been removed. Where this is not the case, the term 'CEA-like material' would perhaps be more appropriate.

J. C. MACARTNEY
ROSEMARY A. WALKER
*Department of Pathology,
The Medical School,
University of Birmingham,
Birmingham B15 2TJ*

References

- Burtin, P., von Kleist, S., Sabine, M. C., and King, M. (1973). Immunohistological localization of carcinoembryonic antigen and nonspecific cross-reacting antigen in gastrointestinal normal and tumoral tissues. *Cancer Research*, **33**, 3299-3305.
- Burtin, P., Quan, P. C., and Sabine, M. C. (1975). Nonspecific cross-reacting antigen as a marker for human polymorphs, macrophages and monocytes. *Nature*, **255**, 714-716.

- Fleisher, M., Oettgen, H. F., Breed, C. N., Robbins, G. F., Pinsky, C. M., and Schwartz, M. K. (1974). CEA-like material in fluid from benign cysts of the breast. *Clinical Chemistry*, **20**, 41-42.
- Isaacson, P., and Judd, M. A. (1977). Immunohistochemistry of carcinoembryonic antigen: characterisation of cross-reactions with other glycoproteins. *Gut*, **18**, 779-785.
- Kuo, T., Rosai, J., and Tillack, T. W. (1973). Immunological studies of membrane glycoproteins isolated from human breast carcinomas. *International Journal of Cancer*, **12**, 532-542.
- Shousha, S., and Lyssiotis, T. (1978). Correlation of carcinoembryonic antigen in tissue sections with spread of mammary carcinoma. *Histopathology*, **2**, 433-447.
- Shousha, S., and Lyssiotis, T. (1979). Granular cell myoblastoma: positive staining for carcinoembryonic antigen. *Journal of Clinical Pathology*, **32**, 219-224.
- Shousha, S., Lyssiotis, T., Godfrey, V. M., and Scheuer, P. J. (1979). Carcinoembryonic antigen in breast-cancer tissue: a useful prognostic indicator. *British Medical Journal*, **1**, 777-779.

Skin as a source of *Acinetobacter*/*Moraxella* species

Like Drs Al-Khoja and Darrell (*Journal of Clinical Pathology*, 1979, **32**, 497) we too have studied the skin as a source of *Acinetobacter*/*Moraxella* species. Studies on inpatients with diseases of the skin show that both *Acinetobacter* and *Moraxella* can be isolated from normal or involved skin in about one-quarter of patients. We found that 26 of 98 patients with 'eczema', 20 of 53 with psoriasis, 7 of 15 with urticaria, and 4 of 26 with other miscellaneous diagnoses yielded isolates of these organisms. Carriage on involved skin was more common; 30 patients had these organisms on involved skin only, 15 from normal and involved skin, and 13 on normal skin (usually forehead or chest) only. Although clearly common on the skin, these organisms seldom cause skin infection (unpublished observations; Glew *et al.*, 1977).

Acinetobacter and *Moraxella* are easily isolated on Cysteine Lactose Electrolyte Deficient Medium (Oxoid) but identification by micromethods such as API (20E) is unsatisfactory owing to their general lack of biochemical activity. (The absence of a pathogenic role makes it undesirable to use the more sophisticated tests such as cell wall composition, as routine tests show that the only regular difference is the oxidase reaction.) About half our isolates are oxidase-positive and

are therefore classed as *Moraxella* (Buchanan and Gibbons, 1974). On this basis, however, one patient sampled five times yielded three 'acinetobacter' and five 'moraxella'. It seems that more accurate speciation is needed.

Retailliau *et al.* (1979) reported *Acinetobacter calcoaceticus* as a hospital pathogen with a high summer prevalence.

The climate in the UK is not sufficiently constant to make a comparison of 'summer' and 'winter' samples meaningful, but we suggest that the summer maximum observed by these authors may be related to increased sweating in summer producing a more favourable environment for the proliferation of these skin inhabitants.

MICHELLE GAUGHAN
PAMELA M. WHITE
W. C. NOBLE
*Institute of Dermatology,
London*

References

- Buchanan, R. E., and Gibbons, N. E. (eds) (1974). *Bergey's Manual of Determinative Bacteriology*, 8th edition, pp. 427-433. Williams and Wilkins, Baltimore.
- Glew, R. H., Moellering, R. C., Jr., and Kunz, L. J. (1977). Infections with *Acinetobacter calcoaceticus* (*Herellea vaginicola*): clinical and laboratory studies. *Medicine*, **56**, 79-97.
- Retailliau, H. F., Hightower, A. W., Dixon, R. E., and Allen, J. R. (1979). *Acinetobacter calcoaceticus*: A nosocomial pathogen with an unusual seasonal pattern. *Journal of Infectious Diseases*, **139**, 371-375.