Our study shows that the flies from the EMHWCC MDT colony are in fact *L. sericata* and not *L. cuprina*. This is what would be expected, as *L. sericata* is widely used in Europe and the USA for MDT.[1]

The issue of correct identification of these blowflies becomes a medical issue when they are used for MDT, and it is advisable to have adequate quality assurance criteria and protocols in place. The most reliable protocol is to sequence the DNA of these flies for a diagnostic gene.

This study highlights the need for quality assurance protocols for identifying flies for MDT. It demonstrates that the nuclear 28S rRNA gene would be a good choice for this task, and suggests that qualified entomologists who specialise in DNA sequencing of flies assist in this matter.

References

1. Sherman RA, Hall MJR, Thomas S. Medicinal maggots: an ancient remedy for some contemporary afflictions. *Annu Rev Entomol* 2000; 45: 55-81.
2. Cronje FJ, Avenant L. MDT in South Africa today. *Palmnut Post* 2006; 9(2): 8-10.
3. Harvey ML, Gaudieri S, Villet MH, Dadour IR. A global study of forensically significant calliphorids: implications for identification. *Forensic Sci Int* (in press): doi:10.1016/j.forsciint.2007.10.009
4. Holloway BA. Morphological characters to identify adult *Lucilia sericata* (Meigen, 1826) and *L. cuprina* (Wiedemann, 1830) (Diptera: Calliphoridae). *New Zealand Journal of Zoology* 1991; 18: 415-420.
5. Wallman JF. A key to the adults of species of blowflies in southern Australia known or suspected to bred in carrion. *Med Vet Entomol* 2001; 15: 433-437.
6. Aubertin D. Revision of the genus *Lucilia* R.-D. (Diptera, Calliphoridae). *Linn Soc J Zool* 1933; 38: 389-463.

Regional clinical registry data show increased incidence of cutaneous melanoma in Cape Town

S Jessop, H Stubbings, R Sayed, J Duncan-Smith, J W Schneider, H F Jordaan

To the Editor: Cutaneous melanoma is a skin tumour that continues to result in a high mortality rate, particularly in the case of thick tumours and those that are deeply invasive histologically. It occurs in all populations but is most common in fair-skinned individuals, especially those with skin types 1 and 2 that tan poorly or not at all. There is epidemiological evidence for the pathogenetic role of ultraviolet light, particularly intense childhood exposure, although the relationship is complex.[1,2] Genetic factors also play a role, as exemplified by families with both atypical naevi and melanoma.[3]

Division of Dermatology, Department of Medicine, University of Cape Town and Groote Schuur Hospital
S Jessop, MB ChB, FFDerm (SA)

Division of Radiation Oncology, Department of Radiation Medicine, University of Cape Town
H Stubbings, MB ChB, FFRad (T) (SA) (currently at Department of Oncology, Norfolk and Norwich University Hospital, Norwich, UK)
J Duncan-Smith, BOccupTher

School of Public Health and Family Medicine, University of Cape Town
R Sayed, MSc (Stats)

Department of Anatomical Pathology, Stellenbosch University and National Health Laboratory Services, Tygerberg Hospital, Parow, W Cape
J W Schneider, MB ChB, MMed (Anat Path), FCPath (SA)

Department of Dermatology, Stellenbosch University and Tygerberg Hospital, Parow, W Cape
H F Jordaan, MB ChB, MMed (Derm)

Corresponding author: *S Jessop (Susan.Jessop@uct.ac.za)*

The rising incidence of melanoma, noted initially in countries with high levels of UV light, appears to be levelling off or decreasing in some areas.[4] The pattern of these trends is inconsistent, with even European countries showing great variation.

An epidemiological study performed in Cape Town from 1990 to 1995 demonstrated an incidence of melanoma of 24.4 per 100 000 white people per annum.[5] We conducted a methodologically identical study in the same geographical area after a 10-year interval, to identify whether there is a trend in the incidence of melanoma in this area.

Methods

A prospective case-based study was conducted from 1 January 2001 to 31 December 2003. All histopathology reports of melanoma were obtained from all pathologists (private and public sector) serving the greater Cape Town area (as in the previous study).[5]

As the incidence of melanoma is low in dark-skinned individuals, the previous study was confined to white people. To obtain comparable data, the study population was defined in the same way in this study. Population figures for greater Cape Town (the study area as defined in the previous study) were based on the projected 2001 population, which estimated a denominator population of 441 970 whites above the age of 14 years.[6]

Histopathology reports were reviewed for patients' geographical area, gender, age, ethnic group, tumour site, Clark

level of invasion, Breslow thickness (mm), and histogenetic type (when available). Thickness was recorded in 3 arbitrary groups, as in the previous study (0 - 1.49, 1.5 - 3.49, and 3.5+ mm).

Reasons for exclusion from the study included living outside the designated area, if the tumour was not a primary melanoma, and if it was confined to the epidermis (*in situ* melanoma). The exclusion criteria used in this study (age under 14, secondary or *in situ* tumours, skin types IV - VI, residence outside the designated area) were identical to those applied in the previous study.[5]

Ethics approval was obtained from the Research Ethics Committee of the University of Cape Town.

Statistical analysis

The collected data were analysed using Stata statistical software.[7] Age-standardised incidence rates were calculated by applying the world standard population.[8] Incidence rates were considered significantly different if their 95% confidence interval (CI) did not overlap. Chi-squared tests were performed to compare the distribution of categorical data between the two studies. The analysis was performed by gender for tumour site, Clark level and Breslow thickness.

Results

Among the primary melanomas recorded during the 3-year study period, 443 satisfied the inclusion criteria and were further analysed. There were 234 (52.8%), in women, and 209 (47.2%) in men. The overall age-standardised incidence rate per year was 33.5 per 100 000 (95% CI 29.1 - 37.8) for women, and 36.9 (95% CI 31.9 - 41.9) for men. In the 1990 - 1995 study, the age-standardised rate for women was 22.2 (CI 19.6 - 24.9), and for men 27.5 (CI 24.3 - 30.7). Therefore, the age-standardised rates show a significant rise in incidence of melanoma over the 10-year period (1.5-fold among women and 1.3-fold among men). The increase is particularly marked in women under 35 years.

Most tumours were found to invade to Clark level 2 and 3 (71% of women and 69% of men). Deep tumours (level 5) were found in 3% of women and 1% of men (Table I). Breslow thickness of the tumours showed a similar pattern, with thinner tumours predominating, lesions of 0 - 1.49 mm being found in 78% of women and 74% of men. Tumours with a Breslow thickness >3.5 mm were seen in 10% of women and 8% of men. The Clark level and Breslow thickness figures showed no significant change compared with the previous study.

Table I. Percentage distribution of Clark level, comparing 1990 - 1995 and 2001 - 2003 data sets

	1990 - 1995 *N* (%)	2001 - 2003 *N* (%)
Women		
Clark level 2	114 (38.5)	88 (37.6)
3	95 (32.1)	77 (32.9)
4	76 (25.7)	63 (26.9)
5	11 (3.7)	6 (2.6)
Total	296 (100)	234 (100)
Chi-squared test (3 degrees of freedom) = 0.67, $p = 0.880$		
Men		
Clark level 2	88 (29.4)	76 (36.4)
3	108 (36.1)	70 (33.5)
4	98 (32.8)	60 (28.7)
5	5 (1.7)	3 (1.4)
Total	299 (100)	209 (100)
Chi-squared test (3 degrees of freedom) = 2.77, $p = 0.428$		

Discussion

Our study demonstrates a significant increase in the incidence of melanoma among whites in Cape Town over a 10-year period (1.5-fold for women and 1.3-fold for men). The use of population census figures as a denominator might have lead to an error in estimates. However, a consistent increased incidence of melanoma in Cape Town was shown, whether applying the data from either of the Cape Town census periods (1991 and 1996) or the projected population figures for 2001. The projected population figure was applied in this study as it was thought likely to be the most accurate.

A rising incidence in melanoma has been reported in other countries over the last 30 years, although it is levelling off in some areas. Our study identifies Cape Town whites as having one of the highest occurrence rates in the world for new primary melanoma.

Recent figures for Australia show an incidence of approximately 48 and 34 per 100 000, for males and females respectively, but the rate in young women appears to be declining. There is little difference in incidence between Cape Town whites (particularly women) and people in the high-prevalence areas of Australia, according to our current figures.

Where melanoma incidence rates have increased elsewhere, the rise in recent years has tended to be in thin tumours (<1.5 mm). This may be due to earlier diagnosis, possibly as a result of greater awareness among doctors and the public. Education programmes may be contributing to this trend. The Cape Town figures do not demonstrate a disproportionate increase in thin tumours. The proportion of thin (<1.5 mm) and thick (>3.49 mm) tumours has remained constant (71 - 74% thin tumours in initial study, and 74 - 78% in later study, 8 - 10% thick tumours in both studies). These findings tend to suggest that the increased numbers in Cape Town reflect a genuine rise in the incidence of melanoma, rather than earlier diagnosis or inclusion of tumours at an earlier stage.

Gender and age differences in melanoma incidence have been observed in several countries. In Cape Town, the

melanoma incidence rate has risen more among women (1.5-fold v. 1.3-fold in men).

How can the rising melanoma incidence among Cape Town whites be explained?

While incomplete data capture could theoretically have led to a misleadingly low figure in the earlier study, there is no reason to suggest that data capture in the 1990 - 1995 period differed from the present. It is more probable that the figures illustrate a true change in incidence. A possible loss of precision in our estimates might have resulted from the lesser timespan of 3 years of this study, in contrast to 6 years in the previous study, owing to financial constraints (that may be remedied by ongoing data capture).

A significant change in the genetic profile of whites in Cape Town is highly improbable. Increased UV light exposure seems the most plausible explanation for the trend, and may reflect childhood patterns of recreational exposure and/or increasing occupational exposure. Educational campaigns could be undertaken to reduce UV exposure, but their impact on lowering the incidence of melanoma has not yet been clearly established, and there would probably be a considerable lag phase. Further education of the public in Cape Town, targeting children in particular, may reduce the incidence of melanoma in this area in the future.

Melanoma is largely a disease of whites, who represent <20% of the population in South Africa. Infectious diseases, particularly tuberculosis and HIV/AIDS, affect large numbers of people, have a huge impact on individuals and the community, and place an enormous demand on the health budget of South Africa. Infectious disease will clearly require first priority for state health funding. However, in view of the poor outcome of advanced melanoma and the high costs of management, it is important to take appropriate steps to increase the detection of this potentially fatal disease at a curable stage. The increasing incidence of melanoma in Cape Town suggests that the modest resources necessary for such preventive steps can be justified.

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References

1. Bliss JM, Ford D, Swerdlow AJ, *et al.* Risk of cutaneous melanoma associated with pigmentation characteristics and freckling: systematic overview of 10 case-control studies. The International Melanoma Analysis Group (IMAGE). *Int J Cancer* 1995; 62: 367-376.
2. Isaacson C, Ramsay M. Does the sun play a role in the aetiology of malignant melanoma? *S Afr Med J* 2007; 97: 568-571.
3. Bataille V. Genetic epidemiology of melanoma. *Eur J Cancer* 2003; 39: 1341-1347.
4. Marks R. The changing incidence and mortality of melanoma in Australia. *Recent Results Cancer Res* 2002; 160: 113-121.
5. Saxe N, Hoffman M, Krige JE, *et al.* Malignant melanoma in Cape Town, South Africa. *Br J Dermatol* 1998; 138: 998-1002.
6. Dorrington RE. Projection of the population of the Cape Metropolitan Area 1996-2031. 2000. http://www.capetown.gov.za/home/demographics.asp (accessed 3 October 2006).
7. Stata Statistical Software: Release 8.0. College Station, Texas: Stata Corporation, 2003.
8. Waterhouse J, Corres P, Muir C, Powell J, eds. *Cancer Incidence in Five Continents*, Vol. 111. Lyons: International Agency for Research on Cancer, 1976: 456.