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LETTER TO THE EDITOR – OPEN ACCESS

A Histogenetic View of the International Classification of Diseases for Oncology Melanomas

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Dear Editor,

Malignant melanoma (M) can be defined as a malignant neoplasm derived from melanocytes; however, there is a high histological and, consequently, clinical variability from case to case (1). In order to try to overcome this intrinsic difficulty, various classification systems have been proposed over the years; in this regard, the World Health Organization (WHO) introduced its notorious classification approximately half a century ago (2). Currently, the International Classification of Diseases for Oncology (ICD-O), provided by the WHO International Agency for Research on Cancer, distinguishes the in situ forms from invasive ones, recognizing among these four main morphological subtypes: nodular M, superficial spreading M, lentigo maligna M, and acral lentiginous M (3). The ICD-O classification includes further morphological codes, such as balloon cell M, regressing M, amelanotic M, M in junctional nevus, M in precancerous melanosis, desmoplastic M, neurotropic M, mucosal lentiginous M, M in giant pigmented nevus/congenital melanocytic nevus, mixed epithelioid and spindle cell M, epithelioid cell M, spindle cell M (not otherwise specified), spindle cell M (type A), spindle cell M (type B), and malignant blue nevus (3). Along with a strictly morphological classification, a histogenetic model, based on the concept of tumor progression, is regaining ground (4, 5). In fact, at the onset, M is characterized by a non-tumorigenic radial growth phase (RGP), inside the epidermis (intraepidermal) or within the papillary dermis (microinvasive), devoid of metastatic potential, which may be followed, early or late, by a tumorigenic vertical growth phase (VGP), with deeper extension in the dermis or beyond, nodular confluence, mitotic activity, and metastatic capacity (Table 1). The unique exception to this is represented by nodular M, in which either RGP is rapidly overrun by VGP, or the tumor arises directly from dermal melanocytes (6). Today, the Breslow depth remains the single most important prognostic factor for clinically localized primary M; it allows to distinguish M as ultra-thin (≤0.5 mm), thin (≤1 mm), thick (>1 mm), or ultra-thick (>6 mm) (7, 8). The systematic application of the histogenetic model to the Breslow depth allows to explain the debated reason why some thin M behave aggressively because they possess an early tumorigenic VGP inside them (9). Moreover, any diagnostic report should be also accompanied by further well-known microstaging attributes, such as Clark level, mitotic count, lymphovascular invasion, perineural infiltration, ulceration, satellitosis, tumor infiltrating lymphocytes, and, if available, sentinel lymph node status (10, 11). In conclusion, we believe that a renewed histogenetic approach to M diagnosis deserves a wide scientific dissemination for better clinical management of individual cases in the modern era of personalized medicine.

Table 1. The non-tumorigenic radial growth phase encompasses the intraepidermal lesions, namely lentigo maligna and *in situ* melanoma (M), and the microinvasive forms including ultra-thin M and the vast majority of thin M. Only a small quota of the thin Ms, burdened by an aggressive biological behavior, shows an early tumorigenic vertical growth phase (VGP). Conversely, a late tumorigenic VGP is constantly present in all thick and ultra-thick M

Melanoma progression model	
Non-tumorigenic radial growth phase (RGP)	Tumorigenic vertical growth phase (VGP)
Intraepidermal RGP	Early VGP
Microinvasive RGP	Late VGP

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