

and

Human Papillomavirus

Rights

С

Preface

Preface to the third edition

Since the first edition of the HPV Information Centre, GLOBOCAN, one of the landmark products of the International Agency for Research on Cancer (IARC), serves as the reference source of cancer statistics. GLOBOCAN is a resource that provides on a regular basis the most accurate assessment of global cancer burden in the world. On June 1st 2010, the new edition of GLOBOCAN, GLOBOCAN 2008, was launched and new cancer estimates for 2008 are currently available.

This third edition of the HPV Information Centre incorporates the new burden estimates for all HPV-related cancers. In addition to the publicly available GLOBOCAN 2008, IARC has kindly provided the HPV Information Centre with age-specific estimates for HPV-related cancers which are also presented in this report.

Preface to the second edition

Human papillomavirus (HPV) infection is now a well-established cause of cervical cancer and there

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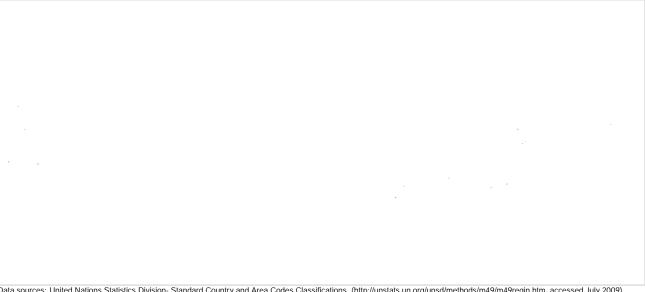
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1 Introduction

Figure 1: World's geographical regions



Data sources: United Nations Statistics Division- Standard Country and Area Codes Classifications. (http://unstats.un.org/unsd/methods/m49/m49regin.htm, accessed July 2009)

The WHO/ICO Information Centre on HPV and Cervical Cancer (HPV Information Centre) aims to compile and centralize updated data and statistics on human papillomavirus (HPV) and HPV-related 1 INTRODUCTION -3-

sis such as the use of smoking, parity, oral contraceptive use and co-infection with HIV.

Section 6 presents sexual and reproductive behaviour indicators that may be used as proxy measures of risk for HPV infection and anogenital cancers.

Section 7 presents preventive strategies that include basic characteristics and performance of cervical cancer screening status, status of HPV vaccine licensure introduction, and recommendations in national immunization programs and the prevalence of male circumcision and condom use.

Figure 3: Estimated population trends of four selected age groups in the World compared to developing and developed regions in

Table 3: Sociodemographic indicators in the World

Indicator	Male	Female	Total
Population in 1000s ¹	3282865 ^a	3229412 ^a	6512276 ^a
Population growth rate (%) ¹	-	-	1.18 ^b
Median age (years) ¹	-	-	27.9 ^a
Population living in urban areas (%) ²	-	-	50 ^c
Crude birth rate (births per 1000 population) ¹	-	-	20.3 ^b
Crude death rate (deaths per 1000 population) ¹	-	-	8.5 ^b

age-standardized rates (ASR) and by age

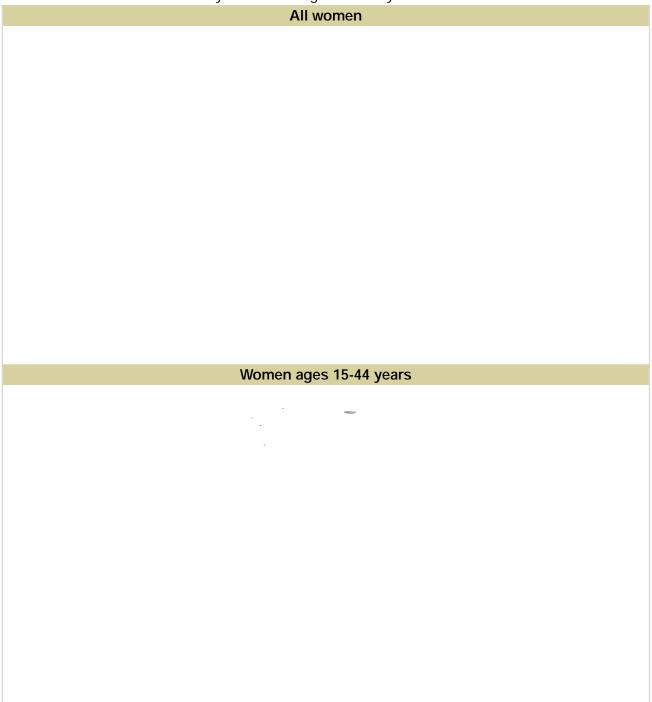
Figure 7: Incidence of cervical cancer compared to other cancers in the World using crude and

Data sources: IARC, Globocan 2008. Age-specific data from GLOBOCAN 2008 were obtained from IARC, personal communication.

3.1.2 Mortality

Table 5: Mortality of cervical cancer by regions and sub-regions in the World

Figure 15: Ranking of cervical cancer to others cancers among all women and women ages 15-44 years according to mortality rates



Highest incidence rate rank 1st. Ranking is based on crude mortality rates (actual number of cervical cancer deaths) in the country/region. Ranking using ASR may differ. Data sources:

Figure 16: World age-specific mortality of cervical cancer in the World compared to developed and developing regions and five continents

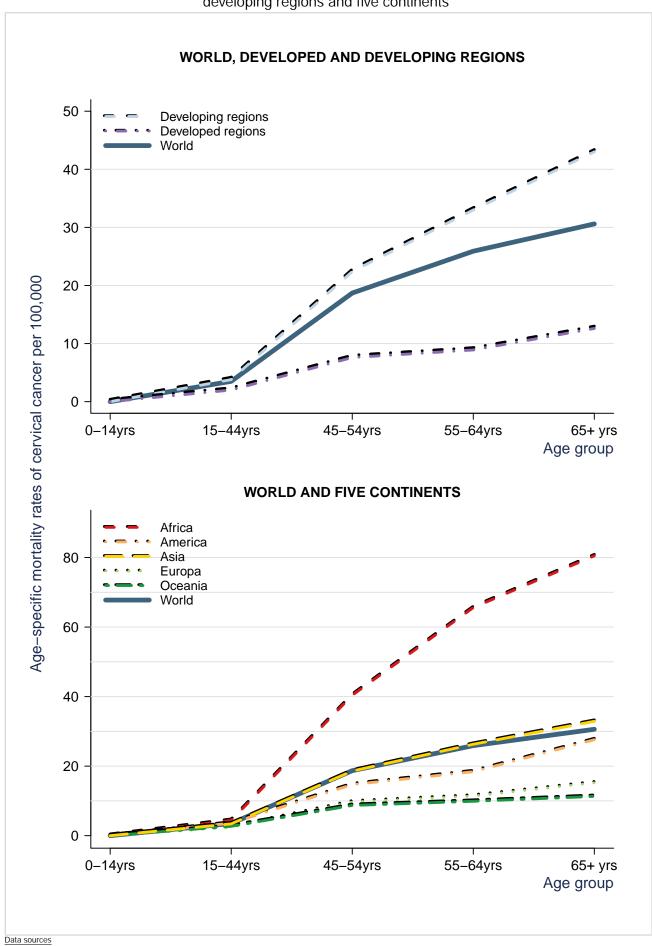
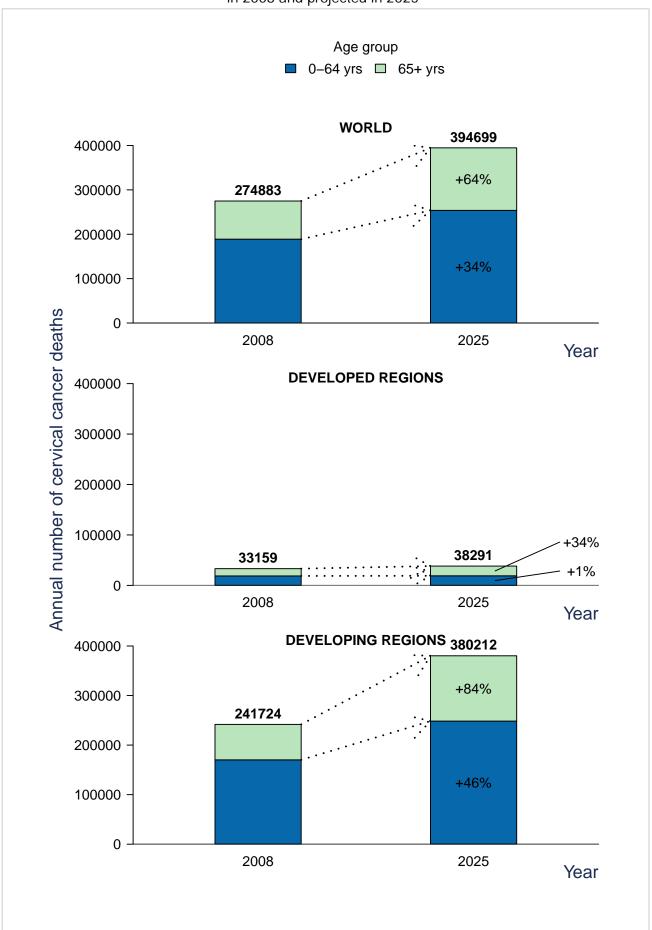


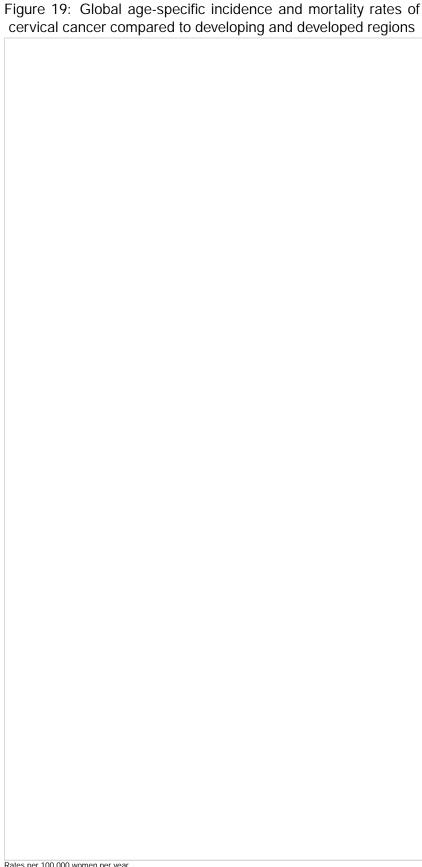
Figure 18: Global estimated number of deaths of cervical cancer by age group compared to regions, in 2008 and projected in 2025



Projected burden in 2025 is estimated by applying current population forecasts for the country and assuming that current mortality rates of cervical cancer are constant over time. Data sources:

IARC, Globocan 2008.

3.1.3 Comparison of incidence and mortality



Rates per 100,000 women per year.

Data sources:
IARC, Globocan 2008. Age-specific data from GLOBOCAN 2008 were obtained from IARC, personal communication.
For specific estimation methodology refer to http://globocan.iarc.fr/DataSource_and_methods.asp.

3.2 Anogenital cancers other than the cervix

Data on the role of HPV in anogenital cancers other than the cervix are limited, but there is an increasing body of evidence strongly linking HPV DNA with cancers of the anus, vulva, vagina, and penis. Although these cancers are much less frequent compared to cancer of the cervix, their association with HPV make them potentially preventable and subject to similar preventative strategies as those for cervical cancer.

(Vaccine 2006, Vol. 24, Supl 3; Vaccine 2008, Vol. 26, Supl 10; IARC Monographs 2007, Vol. 90)

3.2.1 Anal ca

4.1.1 Terminology

Cytologically normal women

No abnormal cells are observed on the surface of their cervix upon cytology.

Cervical Intraepithelial Neoplasia (CIN) / Squamous Intraepithelial Lesions (SIL)

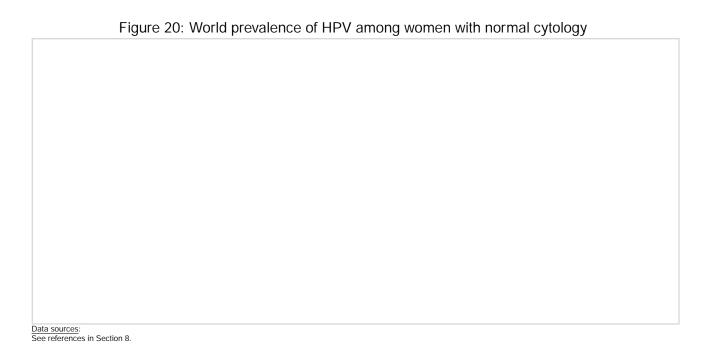


Figure 21: Crude age-specific HPV prevalence in women with normal cytology in the World compared to developing and developed regions and filoped continents

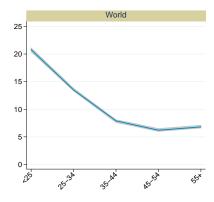


Figure 23: Ten most frequent HPV types among women with invasive cervical cancer in the World

4 HPV RELATED STATISTICS -33-

Table 9: Type-specific HPV prevalence among invasive cervical cancer cases in the World, by histology

4.2 HPV burden in anogenital cancers other than cervix

Table 10: Prevalence of HPV in anogenital cancers other than the cervix

4 HPV RELATED STATISTICS -35-

Table 11: Pooled estimate of HPV prevalence among anal cancer cases, by sex

4.2.2 Vulvar cancer

4 HPV RELATED STATISTICS -38-

4.2.3 Vaginal cancer

Vaginal and cervical cancers share similar risk factors and it is generally accepted that both carcinomas share the same aetiology of HPV infection although there is limited evidence available. Women with vaginal cancer are more likely to have a history of other ano-genital cancers, particularly of the cervix, and these two carcinomas are frequently diagnosed simultaneously. HPV DNA is detected among 91% of invasive vaginal carcinomas and 82% of high-g 0 -rade vaginalasneoplasias (VAIN3).In a case series of vaginal cancers, HPV-16 is the most common type in at least 70% of HPV-positive carcinomas.

(Vaccine 2006, Vol. 24, Supl 3; Vaccine 2008, Vol. 26, Supl 10; IARC Monog0-raphs 2007, Vol. 90

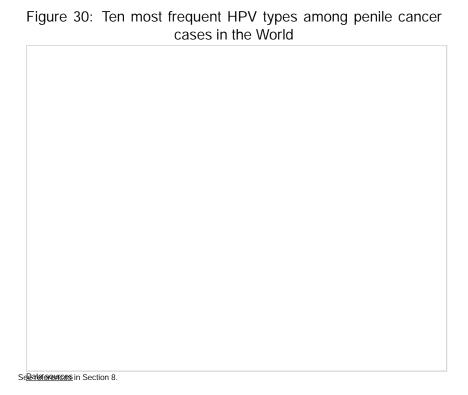
In this section, the HPV burden among cases of vaginal cancers in the World is presented.

	Table 13: Studies	on HPV preva	lence among ca	ses of vaginal	cancer in	the World HPV prevalence
					140.	The v prevalence
Study		HPV dete	ction method	Histology	tested	% PV prevalence

Figure 28:	Ten most fred	quent HPV tases in the \	g vaginal car	icer

4.2.4 Penile cancer

The geographical correlation between the incidence of penile and cervical cancers and the concordance of these two cancers among married couples suggested the common aetiology of HPV infec-



4.3 HPV burden in men

The information to date regarding penile HPV infection is primarily derived from studies that examined husb63eivinfderfdervcross-s0(ectiallev)226(studiesv)22einedl(matiiesv)22suchm-

5 Factors contributing to cervical cancer

HPV is a necessary cause of cervical cancer, but it is not a sufficient cause. Other cofactors are

6 Sexual and reproductive health behaviour indicators

Sexual intercourse is the primary route of transmission of genital HPV infection. Information about

7.2 HPV vaccination

Figure 37: Global HPV vaccine licensure as of March 2009

Quadrivalent HPV vaccine			
Quaurivalent HPV vaccine			
Bivalent HPV vaccine			
Bivalent nev vaccine			

8 REFERENCES -50-

8 References

HPV-related statistics were gathered from specific databases created at the Institut Català d´Oncologia and the International Agency for Research on Cancer.

Systematic collection of published literature from peer-reviewed journals is stored in these databases. Data correspond to results from the following reference papers as well as updated results from continuous monitoring of the literature by the HPV Information Centre:

Cytologically normal women

Data have been compiled by the HPV Information Centre in the Unit of Infections and Cancer at the Institut Catala d'Oncologia and have been published as a meta-analysis in:

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8 REFERENCES -52-

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Côte d'I La Ruche G, Int J Cancer 1998; 76: 480 ¶⋅ ₃

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	Baldwin SB, J Infect Dis 2003; 187: 1064	3/4
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	Chan JK, Br J Cancer 2003; 89: 1062	
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	Yu MY, Int J Cancer 2003; 105: 204	1

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	Lont A, Int J Cancer 2006; 119: 1078			1/2
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	Prinsen CF, BJOG 2007; 114: 951	٠		
	Reesink-Peters N, Eur J Obstet Gynecol Reprod Biol 2001: 98: 199	٠ .		
	Resnick RM, J Natl Cancer Inst 1990; 82: 1477			

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