Acta Dermatovenerol APA

Acta Dermatovenerologica
Alpina, Pannonica et Adriatica

2013;22:1-5
doi: 10.2478/v10162-012-0024-1

Human papillomavirus (HPV) prevalence and HPV type distribution in cervical, vulvar, and anal cancers in central and eastern Europe

Mateja Škamperle[1], Boštjan J. Kocjan[1], Polona J. Maver[1], Katja Seme[1], Mario Poljak[1] ✉

Abstract

Introduction: High-risk human papillomaviruses (HPV) play the leading etiological role in the development of cervical, anal, and vaginal cancers and a substantial proportion of penile, vulvar, and oropharyngeal (tonsillar) cancers.
Methods: The article summarizes the results of the most important studies that examined tissue specimens of cervical, anal, and vulvar carcinoma from 16 central and eastern European countries for the presence of HPV DNA.
Results: Twenty-eight eligible studies were identified. Among 2,531 invasive cervical cancers, 86.6% were HPV-positive. The combined prevalence of HPV-16/18 among HPV-positive cervical cancers was 87.5%. The overall prevalence of HPV DNA in six studies of anal carcinomas (43 cases) and vulvar carcinomas (164 cases) was 90.7% and 32.9%, respectively. HPV-16 DNA was detected in 86% of all anal carcinomas studied, and the proportion of HPV-16 positive vulvar carcinomas varied from 10.9% to 27.5%. HPV-18 DNA was not detected in any anal or vulvar carcinoma studied.
Conclusions: HPV prevalence and type distribution among women with cervical cancer in central and eastern Europe is comparable with other European regions. Several gaps in knowledge exist in the region concerning HPV prevalence and type distribution among women with HPV-related cancers other than cervical cancer and in the general population.

Received: 20 February 2013 | **Returned for modification:** 1 March 2013 | **Accepted:** 3 March 2013

Introduction and Methods

Approximately 40 different human papillomavirus (HPV) types from the clinically most important HPV genus, genus alpha, are known to infect the mucosal epithelium, with a subset of 10 to 15 HPV types associated with various anogenital cancers. These high-risk HPV types play the leading etiological role in the development of cervical, anal, and vaginal cancers and a substantial proportion of penile, vulvar, and oropharyngeal (tonsillar) cancers (1).

The burden of main HPV-related cancer, cervical cancer, is substantially higher in central and eastern Europe than in the rest of Europe, with increasing trends of incidence and mortality in several countries (2). However, several gaps in knowledge exist in the region concerning the incidence and mortality of other HPV-related cancers (2). Similar is true for HPV prevalence and type distribution among women with HPV-related cancers other than cervical cancer and in the general female population (2).

The aim of this article is to provide a detailed and critical review of the most important studies from 16 central and eastern European countries that examined tissue specimens of five HPV-related cancers in the anogenital region–cervical, anal, vaginal, vulvar, and penile carcinoma–for the presence of HPV DNA (3–30). We defined central and eastern Europe as the region comprising the 16 countries of Albania, Bosnia-Herzegovina, Bulgaria, Croatia, Czech Republic, Estonia, Former Yugoslav Republic (FYR) of Macedonia, Hungary, Latvia, Lithuania, Montenegro, Poland, Romania, Serbia, Slovakia, and Slovenia.

The published peer-reviewed studies for these 16 European countries were searched through Medline/Pubmed, Web of Science, Scopus, and Google Scholar without language and time limitations. The search was performed by combining different MeSH terms: *cervical, vulvar, vaginal, penile, anal, cervix, vulva, vagina, penis, anus, HPV, human papillomavirus, prevalence, carcinoma, cancer,* and *neoplasia* with each country name. The search was performed in January 2013. The eligibility criteria for the studies were: (i) at least 10 subjects with a clinical condition of interest, (ii) the use of Hybrid Capture 2 (HC2) (Qiagen Gaithersburg, Inc., MD, USA) or polymerase chain reaction (PCR) capable of detecting preferably all high-risk HPV types but at least HPV-16/18, (iii) a detailed methodological description of HPV DNA detection and genotyping methods used, and (iv) prevalence data provided for at least HPV-16/18.

In this review, 12 HPV types: HPV-16, HPV-18, HPV-31, HPV-33, HPV-35, HPV-39, HPV-45, HPV-51, HPV-52, HPV-56, HPV-58, and HPV-59 are considered carcinogenic (class I) or high-risk HPV types, as recently suggested by the International Agency for Research on Cancer (1). To allow comparison with previous studies from different parts of the world, HPV-16/18 type-specific positivity was presented in two ways for each carcinoma investigated: (i) as a proportion of HPV-16/18–positive cases among all cancers tested for HPV DNA, and (ii) as a proportion of HPV-16/18–positive cases among all cancers tested positive for HPV DNA (Tables 1–3).

Of the two attribution models that are used to derive estimates of proportions of cancers caused by individual HPV types - the proportional and hierarchical attribution model (31) - this study used the proportional attribution model. In the proportional attribution model, a fraction of each case is attributed to every multiple type present in the lesion. In contrast to the hierarchical attribution model, the proportional attribution model tends to favor less common HPV types, including HPV types with little carcinogenic potential, and provides information about minimum proportions of disease caused by individual HPV types (31).

[1]Institute of Microbiology and Immunology, Faculty of Medicine, University of Ljubljana, Zaloška cesta 4, SI-1105 Ljubljana, Slovenia. ✉ **Corresponding author: mario.poljak@mf.uni-lj.si**

Table 1 | **HPV DNA overall prevalence and HPV-16/18 type-specific positivity in women with cervical cancer.**

Country	HPV detection/genotyping method	No. of women tested	Age (years)	Any HPV positive	HPV-16/18 positive overall	HPV-16/18 positive among all HPV DNA positives	Reference
Bulgaria	HPV-16/18 TS PCR	127	mean = 54.3	98 (77.2%)	98 (77.2%)	98 (100%)	(3)
Croatia	MY09/11 nested with GP5+/6+, and CPI/IIg PCR / HPV-6, -11, -16, -18 TS PCR	15	NA	14 (93.3%)	11 (73.3%)	11 (78.6%)	(4)
	GP5+/6+, E6-E7 consensus and SPF10 PCR / Inno-LiPA® and HPV-16, -18, -33 TS PCR	94	range 26–67	87 (92.6%)	66 (70.2%)	66 (75.9%)	(5)
	E6-E7 consensus PCR / HPV-16, -18, -31, -33, -45, -52, -59, -68 TS PCR	89	NA	81 (91%)	67 (75.3%)	67 (82.7%)	(6)
Czech Republic	MY09/11 / DBH and sequencing	71	mean = 50 range 26–84	71 (100%)	61 (85.9%)	61 (85.9%)	(7)
	MY09/11 / DBH and sequencing	49	mean = 44.8 range 30–83	36 (73.5%)	34 (69.4%)	34 (94.4%)	(8)
	GP5+/6+ PCR / RLB and sequencing	86	mean = 49.7 range 28–87	82 (95.3%)	65 (75.6%)	65 (79.3%)	(9)
Latvia	HPV-16/18 TS PCR and Linear Array®	221	mean = 54.9 range 21–92	183 (82.8%)	154 (69.7%)	154 (84.2%)	(10)
Lithuania	MY09/11 / HPV-16 TS PCR and sequencing	212	range 20–69	195 (92%)	131 (61.8%)	131 (67.2%)	(11)
Poland	HPV-16/18 TS PCR	41	NA	28 (68.3%)	28 (68.3%)	28 (100%)	(12)
	HPV-16/18 TS PCR	31	mean = 49.3 range 29–81	21 (67.7%)	21 (67.7%)	21 (100%)	(13)
	MY09/11 PCR / RFLP	53	mean = 58 range 28–82	28 (52.8%)	25 (47.1%)	25 (89.3%)	(14)
	MY09/11 PCR / pU-1M/2R PCR	79	mean = 49.1 range 32–74	60 (75.9%)	55 (69.6%)	55 (91.7%)	(15)
	GP5+/6+ PCR / IA and RLB	88	mean = 56 range 35–85	87 (98.9%)	70 (79.6%)	70 (80.5%)	(16)
	Inno-LiPA®	27	NA	27 (100%)	24 (88.9%)	24 (88.9%)	(17)
	MY09/11 PCR / HPV-16/18 TS PCR	570	NA	516 (90.5%)	516 (90.5%)	516 (100%)	(18)
	Inno-LiPA®	183	NA	161 (88%)	129 (70.5%)	129 (80.1%)	(19)
Romania	HPV-16, -18, -33 TS PCR	30	mean = 52.1	20 (66.7%)	20 (66.7%)	20 (100%)	(20)
	Linear Array®	18	NA	16 (88.9%)	11 (61,1%)	11 (68.8%)	(21)
	Linear Array®	31	mean = 35.7 range 23–65	28 (90.3%)	28 (90.3%)	28 (100%)	(22)
	Linear Array®	20	NA	18 (90%)	16 (80%)	16 (88.9%)	(23)
Serbia	HPV-16/18 TS PCR	48	NA	30 (62.5%)	30 (62.5%)	30 (100%)	(24)
Slovenia	HPV-16/18 TS PCR	70	NA	42 (60%)	42 (60%)	42 (100%)	(25)
	GP5+/6+ and CPI/IIg PCR / sequencing and/or Inno-LiPA®	278	mean = 49 range 22–92	262 (94.2%)	215 (77.3%)	215 (82.1%)	(26)
Overall	–	2,531	–	2,191 (86.6%)	1,917 (75.7%)	1,917 (87.5%)	–

Inno-LiPA®: INNO-LiPA HPV Genotyping Extra test (Innogenetics NV, Ghent, Belgium) or INNO-LiPA HPV genotyping test (Labo Biomedical Products, Rijswijk, the Netherlands); Linear Array®: Linear Array® HPV genotyping test (Roche Molecular Systems Inc., Alameda, CA, USA); PCR: polymerase chain reaction; TS PCR: type specific PCR; DBH: dot-blot hybridization
RLB: reverse line-blot hybridization; IA: immuno-assay–enzyme-linked oligosorbent assay; NA: not available

Table 2 | **HPV DNA overall prevalence and HPV-16/18 type-specific positivity in patients with anal cancer.**

Country	HPV detection / genotyping method	No. of samples tested	Age (years)	Any HPV positive	HPV-16/18 positive overall	HPV-16/18 positive among all HPV DNA positives	Reference
Czech Republic	GP5+/6+ PCR / RLB and sequencing	22	mean = 64.2 range 47–86	18 (81.8%)	18 (81.8%)	18 (100%)	(9, 27)
Slovenia	GP5+/6+ PCR / Inno-LiPA	21	NA	21 (100%)	19 (90.5%)	19 (90.5%)	(28)
Overall	–	43	–	39 (90.7%)	37 (86%)	37 (94.9%)	–

NA: not available

Table 3 | **HPV DNA overall prevalence and HPV-16/18 type-specific positivity in women with vulvar cancer.**

Country	HPV detection genotyping method	No. of samples tested	Age (years)	Any HPV positive	HPV-16/18 positive overall	HPV-16/18 positive among all HPV positives	Reference
Czech Republic	GP5+/6+ PCR / RLB and sequencing	49	mean = 70.7 range 32–95	18 (36.7%)	12 (24.5%)	12 (66.7%)	(9)
	GP5+/6+ PCR / RLB	69	median = 75.0	29 (42%)	19 (27.5%)	19 (65.5%)	(29)
Poland	Linear Array®	46	range 37–93 median = 70.2	7 (15.2%)	5 (10.9%)	5 (71.4%)	(30)
Overall	–	164	–	54 (32.9%)	36 (22%)	36 (66.7%)	–

Results

A detailed search through Medline/Pubmed, Web of Science, Scopus, and Google Scholar resulted in identification of a total of 28 eligible studies (3–30). Twenty-four studies provide data on overall HPV prevalence and HPV type distribution in cervical carcinomas (Table 1) and three studies each for anal (Table 2) and vulvar (Table 3) carcinomas. No eligible study has been identified in peer-reviewed journals dealing with HPV prevalence and type distribution in vaginal and penile carcinomas in the 16 targeted central and eastern European countries.

HPV prevalence and type distribution in women with invasive cervical cancer

As shown in Table 1, in 24 eligible studies a total of 2,531 cases of invasive cervical cancer were tested for the presence of HPV DNA in nine central and eastern European countries: Bulgaria (3), Croatia (4–6), Czech Republic (7–9), Latvia (10), Lithuania (11), Poland (12–19), Romania (20–23), Serbia (24), and Slovenia (25, 26). The mean age of diagnosis with invasive cervical cancer was 50.2 years (range 35.7–58.0 years in individual studies). The great majority of carcinomas were squamous cell carcinomas and 11% were adenocarcinomas or adenosquamous carcinomas. As shown in Table 1, in individual studies HPV DNA prevalence in cervical cancer ranged from 52.8% to 100%, and the overall prevalence was 86.6%. The country-specific overall HPV DNA prevalence in women with invasive cervical cancer and the type-specific prevalence of HPV-16/18 are presented in Fig. 1. As shown in Fig. 1, the cervical cancers with the lowest proportion of HPV-16/18 positivity were identified in Lithuania (61.8%) and with the highest in Poland (80.9%). Such a wide difference can be attributed to the diversity of the clinical specimens used in the studies (cervical scrape specimens, fresh-frozen tissue, formalin-fixed and paraffin-embedded tissue), molecular methods used to detect HPV (with different sensitivity and specificity, and different genotype coverage), or those less likely to show a real difference in HPV type distribution in particular central and eastern European countries.

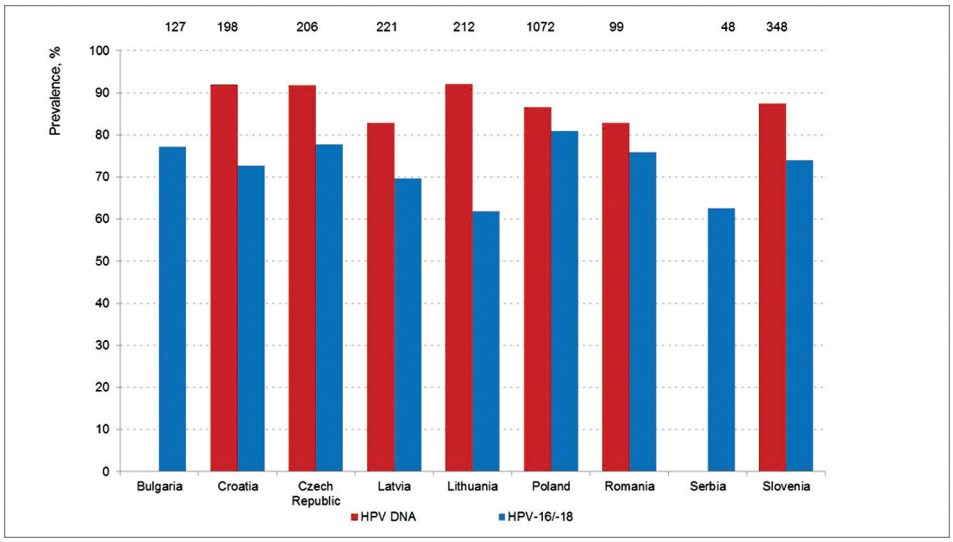

Figure 1 | Overall HPV DNA and HPV-16/18 prevalence in women with invasive cervical cancer from nine central and eastern European countries. Prevalence rates for each country were calculated by pooling data from available published studies: Bulgaria (3), Croatia (4–6), Czech Republic (7–9), Latvia (10), Lithuania (11), Poland (12–19), Romania (20–23), Serbia (24) and Slovenia (25, 26). HPV-16/18 prevalence data include all cases of HPV-16/18–positive cancers (HPV-16/18 present as a single or multiple infection). Numbers above the columns represent the total number of cases of invasive cervical cancers investigated in each country.

In the 21 studies included in this review, type-specific prevalence data presented separately for HPV-16 and HPV-18 was available (3–19, 21–23, 26). HPV-16 DNA was detected in 59.6% (1,420.5/2,383; range 30.9–79.6%) of all cervical carcinomas studied and in 67.7% (1,420.5/2,099; range 34–79.6%) of HPV DNA–positive tumors. On the other hand, the prevalence of HPV-18 DNA was significantly lower: 16.9% (404.5/2,383; range 0–44.4%) of cervical carcinomas studied contained HPV-18 or 19.3% (404.5/2,099; range 0–48.8%) of those identified as HPV DNA–positive. Combined data in individual studies showed the presence of HPV-16/18 in all carcinomas studied ranging from 47.1% to 90.5%. Overall, 75.7% of cervical carcinomas studied were positive for HPV-16/18, or 87.5% of those were identified as HPV DNA–positive. In cervical cancers originating from central and eastern Europe, several non-HPV-16/18 types were also identified, most frequently HPV-31, HPV-33 and HPV-45 (as single or multiple infections), while the frequency of other detected HPV types: HPV-35, HPV-39, HPV-51, HPV-52, HPV-56, HPV-58, HPV-59 and HPV-68, was significantly lower.

HPV DNA prevalence and HPV type distribution in cervical cancers originating from central and eastern Europe are consistent with data obtained in a recent large-scale retrospective cross-sectional study (32) as well as in a recent meta-analysis of HPV type distribution in women with cervical cancer worldwide (33).

HPV prevalence and type distribution in anal cancers

HPV prevalence and type distribution in anal carcinomas from three eligible studies - one from Slovenia (28) and two from the Czech Republic (9, 27) - are presented in Table 2. Taking into account that 22 samples were identical in both Czech studies, altogether 43 samples of anal carcinomas obtained from 28 female and 15 male patients were included in this analysis. The great majority of cancers (91%) were histologically defined as invasive squamous cell carcinomas and 9% as carcinomas in situ. HPV DNA prevalence ranged from 81.8% to 100% in individual studies, with an overall HPV DNA prevalence of 90.7% (Table 2). Among 39 HPV DNA–positive anal cancers, HPV-16 was identified in 37 (94.9%) samples. HPV-6 single and multiple infection with HPV-52 and HPV-61 was detected in one sample each. HPV-18 was not identified in any anal cancer tested. HPV prevalence in anal carcinomas from two central European countries was comparable to figures recently described in a meta-analysis of 29 studies, in which 696 anal carcinomas from 13 European studies were tested for HPV DNA (34).

HPV prevalence and type distribution in women with vulvar cancer

As shown in Table 3, three eligible studies in women with vulvar cancer were identified, one from Poland (30) and two from the Czech Republic (9, 29). In both Czech studies, some samples were identical but it was not possible to specify and separate all duplicated cases. Thus, the total number of vulvar carcinoma samples included in this review was 164, and all were histologically defined as squamous cell carcinoma. HPV DNA prevalence ranged from 15.2% to 42% in individual studies, with an overall HPV DNA prevalence of 32.9%. HPV-16 was detected in 22% of all vulvar carcinomas studied and in 66.7% of HPV DNA–positive tumors (Table 3). HPV-18 was not identified in any vulvar cancer tested. HPV-6 was detected as a single HPV type in two vulvar cancers: one each from Poland and the Czech Republic. Individual cancers contained other high-risk types (HPV-33, HPV-45, HPV-58) and some also low-risk HPV types (HPV-42) (29, 30).

HPV prevalence and type distribution in vulvar carcinomas originating from two central European countries was comparable to figures presented in a recent meta-analysis that showed a HPV prevalence in vulvar carcinomas of 40.4% worldwide and 34.7% in Europe, with HPV-16 detected in 32.2% of all vulvar carcinomas tested (34).

Conclusions

HPV prevalence and genotype distribution among women with cervical cancer in central and eastern Europe is comparable with other European regions. Several gaps in knowledge exist in the region concerning HPV prevalence and type distribution among individuals with HPV-related cancers other than cervical cancer (especially vaginal and penile carcinoma) as well as in the general population of both genders. Further research is urgently needed in the region to provide these important and missing data.

References

1. Bouvard V, Baan R, Straif K, Grosse Y, Secretan B, El Ghissassi F, et al. A review of human carcinogens—Part B: biological agents. Lancet Oncol. 2009;10:321-2.
2. Kesic V, Poljak M, Rogovskaya S. Cervical cancer burden and prevention activities in Europe. Cancer Epidemiol Biomarkers Prev. 2012;21:1423-33.
3. Todorova I, Ganchev G, Shikova E. Human papillomavirus prevalence in invasive cervical carcinomas in Bulgaria. J Clin Pathol. 2010;63:1121-3.
4. Dabic MM, Hlupic L, Babic D, Jukic S, Seiwerth S. Comparison of polymerase chain reaction and catalyzed signal amplification in situ hybridization methods for human papillomavirus detection in paraffin-embedded cervical preneoplastic and neoplastic lesions. Arch Med Res. 2004;35:511-6.
5. Hadzisejdic I, Simat M, Bosak A, Krasevic M, Grahovac B. Prevalence of human papillomavirus genotypes in cervical cancer and precursor lesions. Coll Antropol. 2006;30:879-83.
6. Hadzisejdic I, Krasevic M, Haller H, Grahovac B. Distribution of human papillomavirus types in different histological subtypes of cervical adenocarcinoma. Coll Antropol. 2007;31:97-102.
7. Tachezy R, Mikyskova I, Salakova M, Van Ranst M. Correlation between human papillomavirus-associated cervical cancer and p53 codon 72 arginine/proline polymorphism. Hum Genet. 1999;105:564-6.
8. Tachezy R, Hamsikova E, Hajek T, Mikyskova I, Smahel M, Van Ranst M, et al. Human papillomavirus genotype spectrum in Czech women: correlation of HPV DNA presence with antibodies against HPV-16, 18, and 33 virus-like particles. J Med Virol. 1999;58:378-86.
9. Tachezy R, Smahelova J, Salakova M, Arbyn M, Rob L, Skapa P, et al. Human papillomavirus genotype distribution in Czech women and men with diseases etiologically linked to HPV. PLoS One. 2011;6:e21913.
10. Silins I, Wang X, Tadesse A, Jansen KU, Schiller JT, Avall-Lundqvist E, et al. A population-based study of cervical carcinoma and HPV infection in Latvia. Gynecol Oncol. 2004;93:484-92.
11. Gudleviciene Z, Didziapetriene J, Ramael M, Uleckiene S, Valuckas KP. Human papillomavirus and p53 polymorphism in Lithuanian cervical cancer patients. Gynecol Oncol. 2006;102:530-3.
12. Bar JK, Harlozinska A, Sedlaczek P, Kasiak J, Markowska J. Relations between the expression of p53, c-erbB-2, Ki-67 and HPV infection in cervical carcinomas and cervical dysplasias. Anticancer Res. 2001;21:1001-6.
13. Stenzel A, Semczuk A, Rozynskal K, Jakowicki J, Wojcierowski J. "Low-risk" and "high-risk" HPV-infection and K-ras gene point mutations in human cervical cancer: a study of 31 cases. Pathol Res Pract. 2001;197:597-603.
14. Dybikowska A, Licznerski P, Podhajska A. HPV detection in cervical cancer patients in northern Poland. Oncol Rep. 2002;9:871-4.
15. Lukaszuk K, Liss J, Wozniak I, Sliwinski W, Emerich J, Wojcikowski C. HPV and histological status of pelvic lymph node metastases in cervical cancer: a prospective study. J Clin Pathol. 2004;57:472-6.
16. Bardin A, Vaccarella S, Clifford GM, Lissowska J, Rekosz M, Bobkiewicz P, et al. Human papillomavirus infection in women with and without cervical cancer in Warsaw, Poland. Eur J Cancer. 2008;44:557-64.

17. Szostek S, Klimek M, Zawilinska B, Kosz-Vnenchak M. Genotype-specific human papillomavirus detection in cervical smears. Acta Biochim Pol. 2008;55:687-92.
18. Kwasniewska A, Korobowicz E, Zdunek M, Skoczynski M, Kwasniewski W, Danilos J, et al. Prevalence of Chlamydia trachomatis and herpes simplex virus 2 in cervical carcinoma associated with human papillomavirus detected in paraffin-sectioned samples. Eur J Gynaecol Oncol. 2009;30:65-70.
19. Nowakowski A, De Sanjose S, Alemany L, Klaustermeier JE, Lloveras B, Kotarski J, et al. HPV prevalence and genotype distribution in cervical cancer in Poland. In: Epidemiology/public health abstract book. Abstract of the 27th papillomavirus conference and clinical workshop; 2011 Sep 17–22; Berlin. Berlin (Germany); 2011. p. 52.
20. Pavai Z, Fule T, Horvath E, Mathe M, Pap Z, Denes L, et al. Comparative detection of high-risk HPV (16, 18, 33) in cervical bioptic material of county hospital of Tg. Mures. Rom J Morphol Embryol. 2006;47:229-34.
21 Goia-Rusanu CD, Iancu IV, Botezatu A, Socolov D, Huica I, Plesa A, et al. Mitochondrial DNA mutations in patients with HRHPV-related cervical lesions. Roum Arch Microbiol Immunol. 2011;70:5-10.
22. Botezatu A, Goia-Rusanu CD, Iancu IV, Huica I, Plesa A, Socolov D, et al. Quantitative analysis of the relationship between microRNA124a, -34b and -203 gene methylation and cervical oncogenesis. Mol Med Report. 2011;4:121-8.
23. Iancu IV, Botezatu A, Goia-Rusanu CD, Stanescu A, Huica I, Nistor E, et al. TGF-beta signalling pathway factors in HPV-induced cervical lesions. Roum Arch Microbiol Immunol. 2010;69:113-8.
24. Malisic E, Jankovic R, Jakovljevic K, Cavic M. HPV infection and TP53 codon 72 polymorphism in cervical carcinoma patients from Serbia. In Vivo. 2011;25:541.
25. Ursic-Vrscaj M, Kovacic J, Poljak M, Marin J. Association of risk factors for cervical cancer and human papilloma viruses in invasive cervical cancer. Eur J Gynaecol Oncol. 1996;17:368-71.
26. Jancar N, Kocjan BJ, Poljak M, Lunar MM, Bokal EV. Distribution of human papillomavirus genotypes in women with cervical cancer in Slovenia. Eur J Obstet Gynecol Reprod Biol. 2009;145:184-8.
27. Tachezy R, Jirasek T, Salakova M, Ludvikova V, Kubecova M, Horak L, et al. Human papillomavirus infection and tumours of the anal canal: correlation of histology, PCR detection in paraffin sections and serology. APMIS. 2007;115:195-203.
28. Komlos KF, Kocjan BJ, Kosorok P, Rus T, Toplak J, Bunic M, et al. Distribution of HPV genotypes in Slovenian patients with anal carcinoma: preliminary results. Acta dermatovenerol Alp Panonica Adriat. 2011;20:141-3.
29. Skapa P, Zamecnik J, Hamsikova E, Salakova M, Smahelova J, Jandova K, et al. Human papillomavirus (HPV) profiles of vulvar lesions: possible implications for the classification of vulvar squamous cell carcinoma precursors and for the efficacy of prophylactic HPV vaccination. Am J Surg Pathol. 2007;31:1834-43.
30. Kowalewska M, Szkoda MT, Radziszewski J, Ptaszynski K, Bidzinski M, Siedlecki JA. The frequency of human papillomavirus infection in Polish patients with vulvar squamous cell carcinoma. Int J Gynecol Cancer. 2010;20:434-7.
31 Sahasrabuddhe VV, Castle PE, Follansbee S, Borgonovo S, Tokugawa D, Schwartz LM, et al. Human papillomavirus genotype attribution and estimation of preventable fraction of anal intraepithelial neoplasia cases among HIV-infected men who have sex with men. J Infect Dis. 2013;207:392-401.
32. de Sanjose S, Quint WG, Alemany L, Geraets DT, Klaustermeier JE, Lloveras B, et al. Human papillomavirus genotype attribution in invasive cervical cancer: a retrospective cross-sectional worldwide study. Lancet Oncol. 2010;11:1048-56.
33. Li N, Franceschi S, Howell-Jones R, Snijders PJ, Clifford GM. Human papillomavirus type distribution in 30,848 invasive cervical cancers worldwide: Variation by geographical region, histological type and year of publication. Int J Cancer. 2011;128:927-35.
34. De Vuyst H, Clifford GM, Nascimento MC, Madeleine MM, Franceschi S. Prevalence and type distribution of human papillomavirus in carcinoma and intraepithelial neoplasia of the vulva, vagina and anus: a meta-analysis. Int J Cancer. 2009;124:1626-36.