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SHORT REPORT

Cancer Epidemiology

High-risk human papillomavirus distribution according to human immunodeficiency virus status among women with cervical cancer in Abidjan, Côte d'Ivoire, 2018 to 2020

Simon P. Boni[1,2] | Vanessa Tenet[3] | Apollinaire Horo[4] | Daniëlle A. M. Heideman[5,6] | Maaike C. G. Bleeker[5,6] | Aristophane Tanon[7] | Boston Mian[8] | Isidore D. Mohenou[9] | Didier K. Ekouevi[1,10,11] | Tarik Gheit[12] | Judith Didi-Kouko Coulibaly[13] | Boris K. Tchounga[14] | Innocent Adoubi[2,15] | Gary M. Clifford[3] | Antoine Jaquet[10] | on behalf The IeDEA West Africa Collaboration

Correspondence
Simon P. Boni, PAC-CI Research Program, Treichville, Abidjan, Côte d'Ivoire; National Cancer Control Program, Abidjan, Côte d'Ivoire.
Email: simon-pierre.boni@pac-ci.org

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Abstract

As human papillomavirus (HPV) immunisation and HPV-based cervical cancer (CC) screening programmes expand across sub-Saharan Africa, we investigated the potential impact of human immunodeficiency virus (HIV) status on high-risk (HR)-HPV distribution among women with CC in Côte d'Ivoire. From July 2018 to June 2020, paraffin-embedded CC specimens diagnosed in Abidjan, Côte d'Ivoire were systematically collected and tested for HR-HPV DNA. Type-specific HR-HPV prevalence was compared according to HIV status. Of the 170 CC specimens analysed (median age 52 years, interquartile range: [43.0-60.0]), 43 (25.3%) were from women living with HIV (WLHIV) with a median CD4 count of 526 [373-833] cells/mm^3 and 86% were on antiretroviral therapy (ART). The overall HR-HPV prevalence was 89.4% [95% CI: 84.7-94.1]. All were single HR-HPV infections with no differences according to HIV status ($P = .8$). Among HR-HPV-positive CC specimens, the most prevalent HR-HPV types were HPV16 (57.2%), HPV18 (19.7%), HPV45 (8.6%) and HPV35 (4.6%), with no significant differences according to HIV status. Altogether, infection with HPV16/18 accounted for 71.1% [95% CI: 55.9-86.2] of CC cases in WLHIV vs 78.9% [95% CI: 71.3-86.5] in women without HIV ($P = .3$). The study confirms the major role of HPV16/18 in CC in Côte d'Ivoire and should support a regional scale-up of HPV16/18 vaccination programmes regardless of HIV status.

Abbreviations: ADC, adenocarcinoma; ART, antiretroviral therapy; CC, cervical cancer; CI, confidence interval; H&E, haematoxylin and eosin; HIV, human immunodeficiency virus; HPV, human papillomavirus; HR, high risk; IARC, International Agency for Research on Cancer; IQR, interquartile range; NNRTI, non-nucleoside reverse transcriptase inhibitors; PI, protease inhibitor; SCC, squamous cell carcinoma; SSA, sub-Saharan Africa; WHO, World Health Organisation; WLHIV, women living with HIV.

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However, vaccines targeting additional HR-HPV types, including HPV45 and HPV35, could further decrease future CC incidence in Côte d'Ivoire, both for WLHIV and women without HIV.

KEYWORDS
cervical cancer, Côte d'Ivoire, high-risk human papillomavirus, human immunodeficiency virus

What's new?

HIV favours the persistence of high-risk HPV and progression of cervical intraepithelial neoplasia to cancer, with universal access to antiretroviral therapy also possibly influencing high-risk HPV distribution. This report addresses the timely question of the contributing role of HIV infection in high-risk HPV distribution among women with invasive cervical cancer in Côte d'Ivoire, a country highly affected by both oncogenic viruses. As Côte d'Ivoire and a growing number of other countries today scale up HPV 16/18 vaccination, here the authors confirm that this primary prevention strategy will highly benefit women in this setting, regardless of their HIV status.

1 | INTRODUCTION

Worldwide, it is estimated that 604 127 new cases and 341 831 deaths were attributed to cervical cancer (CC) in 2020, with low- and middle-income countries hosting approximately 90% of CC-related deaths globally.[1]

Thirteen high-risk (HR)-HPV types (HPV16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59 and 68) are classified as human carcinogens,[2] and established as causative agents of CC.[3,4] HPV16 and HPV18 are currently considered to contribute to ~70% of the global CC burden, however African populations remain underrepresented in these pooled estimates.[5] In sub-Saharan Africa (SSA), the attributable fraction of HPV45 and HPV35 in CC was slightly higher compared to other regions.[6] Previous evidence has highlighted regional disparities, including the higher relative contribution of HPV16/18 to CC in Eastern/Southern Africa compared to West Africa.[7,8]

Human immunodeficiency virus (HIV), mainly through its immunosuppressive action, is known to favour the persistence of HR-HPV and the progression of cervical intraepithelial neoplasia resulting in a 5- to 6-fold increased risk of CC.[9] National HPV immunisation programmes are increasingly being rolled-out across SSA, including Côte d'Ivoire. It is, therefore, of utmost importance to document HR-HPV vaccine efficacy/effectiveness and the impact on the occurrence of CC at a national level.[10]

Evidence supports the positive influence of antiretroviral therapy (ART) on reducing HR-HPV prevalence, and potential changes in HR-HPV distribution,[11-13] however, the data in West Africa—where HIV is endemic—are scarce.

In a context of increasing access to HPV-based CC prevention strategies using partial genotyping for HPV16/18 or HPV16/18/45 as a triage option, addressing the contribution of specific HR-HPV genotypes on CC while highlighting the effect of HIV co-infection is critical.

The present study aimed to investigate the HR-HPV distribution in women diagnosed with CC in Côte d'Ivoire, as well as the potential impact of HIV co-infection in a time of universal access to ART.

2 | MATERIALS AND METHODS

2.1 | Study design and setting

A cross-sectional survey was conducted by collecting CC biopsy samples from July 2018 to June 2020. During this period, all suspected CC cases in the five major cancer facilities of Abidjan were consecutively and prospectively biopsied and underwent a histopathology examination at a national histopathology laboratory in Abidjan.

All biopsies were processed in paraffin-embedded tissue blocks. Subsequently, haematoxylin and eosin (H&E) stained slides were used to confirm the presence of CC. Paraffin-embedded tissue blocks with cancer were sent to the Amsterdam University Medical Center's Department of Pathology in the Netherlands, for confirmation of CC in the tissue block and HR-HPV genotyping.

2.2 | HPV testing

For HR-HPV DNA detection, H&E-guided core punch biopsies from the tumour area in the FFPE-blocks were taken and DNA was isolated as described previously.[14] Beta-globin PCR analysis was used to assess the quality of the DNA to be submitted to HPV PCR. HR-HPV testing was performed using GP5+/6+ mediated PCR, followed by hybridization of PCR products in an enzyme immunoassay with an oligoprobe cocktail that together detects 13 HR-HPV types (HPV16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59 and 68). Subsequent HPV genotyping was conducted using a microsphere bead-based assay (Luminex).

For histologically confirmed CC cases that were HR-HPV negative by GP5+/6+ PCR, extracted DNAs were additionally tested at IARC, Lyon, France, using a type-specific E7 PCR bead-based multiplex genotyping assay (E7-MPG).[15]

2.3 | Data collection

Before biopsy, a standardised questionnaire for women with suspected CC was completed by oncologists and gynaecologists. Collected data included sociodemographic information (age, educational level, monthly income, health insurance, etc), reproductive health and sexual behaviour history (age at sexual debut, number of lifetime sexual partners) and tobacco use.

HIV status was documented using the national algorithm. During the screening visit, a nationally approved rapid HIV test (Determine HIV-1/2, by Abbott Diagnostics) was systematically proposed to women following a counselling session with a cancer specialist. Capillary blood was collected using a finger prick test at the time of interview. A positive result initiated the collection of a venous blood sample for confirmation purposes, using an Enzyme Linked Immune Sorbent Assay (ELISA) test, according to the national algorithm of Côte d'Ivoire.[16] Proof of a previous positive test was presented by women already in HIV care. For these women, HIV characteristics including date of first HIV diagnosis, ART use, last known CD4 count (and last known HIV viral load measure, only for the 2018-2020 period) were requested and then merged from HIV-programme databases.

2.4 | Statistical analysis

Categorical variables were described as proportions, and continuous variables as medians and their interquartile ranges (IQR). HR-HPV prevalence was estimated as well as the corresponding 95% confidence intervals (CI). HR-HPV prevalence (overall) and HPV-type distribution (only for CC cases with HR-HPV-positive test) were compared between women living with HIV (WLHIV) and women without HIV. Comparisons were made using Pearson's chi-squared test or Fisher's exact test when appropriate for categorical or dichotomous variables; the Student *t* test or Wilcoxon rank-sum test was used for continuous variables.

3 | RESULTS

Of the 189 suspected CC cases, all consented to participate in the present study and a total of 170 (89.9%) were histologically confirmed and included in the analyses. Of the 170 women with CC, 43 (25.3%) were also living with HIV (Table 1). Of these 170 women, 144 (84.7%) had squamous cell carcinoma (SCC), and 26 (15.3%) adenocarcinoma (ADC). Age at invasive CC diagnosis was 47 years [IQR: 38-52] in WLHIV, and 54 years [IQR: 45-62] in women without HIV ($P < .001$), (Table 1). The reported age at sexual debut was 16 years [IQR: 15-18]. An advanced FIGO stage (III-IV) at diagnosis was reported in 118 (69.4%) women, less commonly in WLHIV (55.8%) compared to women without HIV (74%) ($P = .025$).

Among the 43 WLHIV, 37 (86%) were already on ART before at CC diagnosis (Table S1).

3.1 | HR-HPV prevalence and distribution according to HIV status

In total, GP5+/6+ detected 122 (71.8%) any HR-HPV, while 152 (89.4%) were confirmed HR-HPV positive using both GP5+/6+ and E7-MPG among the 170 samples (Table S2).

Overall HR-HPV prevalence was 89.4% [CI: 84.7-94.1], including 88.4% among WLHIV and 89.8% in women without HIV ($P = .8$).

Among the 152 women with a positive HR-HPV test, the most prevalent types were HPV16 (n = 87; 57.2%), HPV18 (n = 30; 19.7%), HPV45 (n = 13; 8.6%) and HPV35 (n = 7; 4.6%). All were single HR-HPV infections. HPV 56 (n = 3; 2.0%) was only found in WLHIV (7.9%), (Figure 1). No independent association between HPV16, HPV18, HPV45 and HIV status was seen. HR-HPV16/18 was found in 117 (77.0% [95% CI: 70.2-83.7] women: 71.1% [95% CI: 55.9-86.2] in WLHIV and 79% [95% CI: 71.3-86.5] in women without HIV ($P = .32$). Taken together, HR-HPV16/18/45 were identified in 130 (85.5% [95% CI: 79.9-91.2%]) women including 30 (79.0%) WLHIV and 100 (87.7%) women without HIV, ($P = .2$). HR-HPV types: 16, 18, 31, 33, 45, 52 and 58 accounted overall for 90.8% (86.8% in WLHIV and 92.1% in women without HIV; $P = .3$).

In addition, regarding histological types, HR-HPV prevalence was 89.6% in SCC and 88.5% in ADC, ($P = .8$). HPV16 ($P = .018$) and HPV18 ($P = .011$) were more likely to be found in SCC and in ADC, respectively.

4 | DISCUSSION

Our study reported a high prevalence of HR-HPV in women with histologically confirmed CC in Côte d'Ivoire, regardless of HIV status. HIV did not influence the distribution of HR-HPV in women diagnosed with CC, at a time of universal access to ART, providing reassuring results on the potential effectiveness of HPV vaccines in Côte d'Ivoire.

HR-HPV prevalence according to HIV status was consistent with previous estimates (from meta-analysis) in settings highly affected by HIV including reports from SSA.[17]

Our study also reported no difference in HR-HPV distribution according to HIV status contrary to previous estimates from Clifford et al that reported less frequent infection with HPV16 in WLHIV with CC compared to their HIV-uninfected counterparts.[17]

While the absence of differences in HR-HPV prevalence and distribution according to HIV status could be partly explained by our limited sample size, factors such as wider access to ART or starting ART

TABLE 1 General characteristics of 170 women with cervical cancer according to HIV status in Abidjan, Côte d'Ivoire.

Characteristics	Total (N = 170) n (%)	Women without HIV (N = 127) n (%)	WLHIV (N = 43) n (%)	*P* value
Age at CC diagnosis, median [IQR[a]], years	52.0 [43.0-60.0]	54.0 [45.0-62.0]	47.0 [38.0-52.0]	<.001
≥25-44	47 (27.7)	29 (22.8)	18 (41.9)	.03
≥45-59	75 (44.1)	57 (44.9)	18 (41.9)	
≥60	48 (28.2)	41 (32.3)	7 (16.2)	
Residency				.8
Rural/Semi-urban	144 (84.7)	108 (85.0)	36 (83.7)	
Urban	26 (15.3)	19 (15.0)	7 (16.3)	
Educational level				.1
No or Primary level	140 (82.4)	108 (85.0)	32 (74.4)	
Secondary level or University	30 (17.6)	19 (15.0)	11 (25.6)	
Having paid employment				.3
Yes	75 (44.1)	52 (40.9)	23 (53.5)	
None	77 (45.3)	61 (48.1)	16 (37.2)	
Retired	18 (10.6)	14 (11.0)	4 (9.3)	
Age at sexual debut	16 [15-18]	16 [15-18]	16 [15.5-18.0]	.9
Number of lifetime sexual partners				
1-2	104 (61.2)	87 (68.5)	17 (39.5)	<.001
≥3	61 (35.9)	37 (29.2)	24 (55.8)	
Do not wish to answer	5 (2.9)	3 (2.3)	2 (4.7)	
Number of pregnancies, median [IQR]	6 [4-8]	7 [5-9]	5 [4-7]	<.001
Parity, median [IQR[a]]	5 [3-7]	6 [4-7]	4 [2-6]	<.001
<5	67 (39.4)	40 (31.5)	27 (63.8)	
≥5	103 (60.6)	87 (68.5)	16 (37.2)	
History of using contraceptives[b] (yes)	25 (14.7)	18 (14.2)	7 (16.3)	.7
Tobacco use				.3
No use	151 (88.8)	111 (87.4)	40 (93.0)	
Current or former use	19 (11.2)	16 (12.6)	3 (7.0)	
Histological types				
SCC	144 (84.7)	106 (83.5)	38 (88.4)	.4
ADC	26 (15.3)	21 (16.5)	5 (11.6)	
FIGO stage				
I-II	52 (30.6)	33 (26.0)	19 (44.2)	.025
III-IV	118 (69.4)	94 (74.0)	24 (55.8)	

[a]Interquartile range.
[b]Oral/injectable contraceptives, implants or intrauterine devices.

earlier might have mitigated the differentiated role of HPV16/18 in WLHIV, compared to women without HIV, as reported in early reviews.[13,18] Furthermore, the relative insensitivity of HPV16 to changes in immunity in comparison to other types,[19] may reduce differences in its prevalence in WLHIV.

In our study, a relatively high proportion (10.6%) of CC samples tested negative for any HR-HPV. While ADC is usually more frequently HR-HPV negative compared to SCC, our present findings report no differences in negative HR-HPV CC according to histological subtypes. It has been reported that sampling errors at clinical and local pathological level as well as suboptimal storage and transportation between facilities and international referral Lab of CC tissues for HPV identification may lead to negative HR-HPV CC.[20] The high proportion of CC tested negative for HR-HPV in our study could also be due to the intrinsic performances in terms of sensitivity of PCR tests. Indeed, using samples previously identified as negative to HR-HPV (7% of HR-HPV negative CC reported by Bosch et al), Walboomers et al demonstrated that they were almost all false negative by applying a combination of generic and type-specific PCR, as well as E7, E1 and L1 sequences amplification that could prevent any deletion or

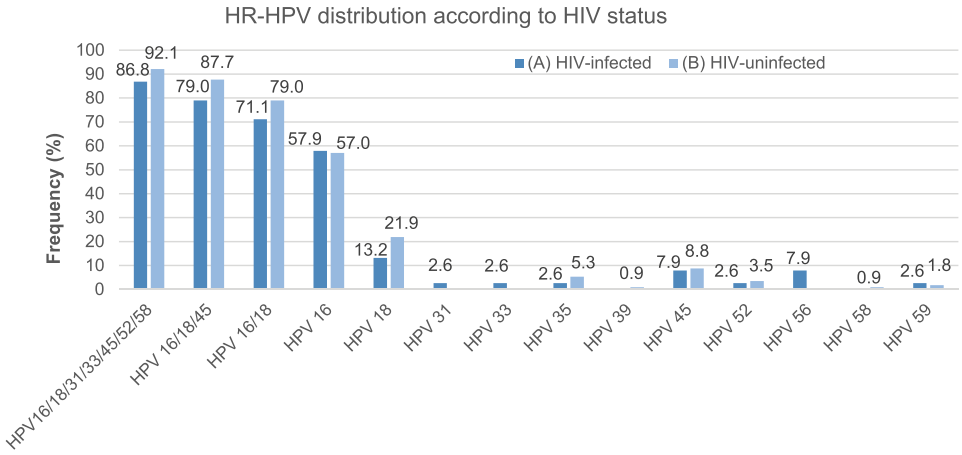

FIGURE 1 High-risk HPV genotypes distribution according to HIV status among 152 women with HR-HPV positive test with cervical cancer in Abidjan, Côte d'Ivoire 2018 to 2020.

disruption from viral integration.[21,22] Although marginally reported, true HPV-negative CC deserves a particular attention in terms of identification as their clinical features and prognosis seem worse than their HR-HPV positive counterparts.[20]

Unlike most other studies, our study reported only single HR-HPV infections in both WLHIV and without HIV with CC.[17,23] This is expected to be related to our choice of HPV testing protocol. We took small punch biopsies from the tumour area, first using GP5+/6+ PCR which is validated to detect clinically relevant HPV infections, and we then only applied a more analytically sensitive HPV test (E7-MPG) to GP5+/6+ PCR-negative tumour samples. This approach, is considered of particular importance as it strengthens the causal association between HR-HPV types and CC.[24]

As expected, HPV16 and HPV18 were the most prevalent HR-HPV in our study, consistent with global patterns.[5] These two genotypes were also associated with histological types but in opposite directions: HPV16 was more common in SCC, while HPV18 was more likely to be found in ADC cases. Despite the underrepresentation of ADC cases in this study, similar associations have been previously reported.[24] This phenomenon of opposition in HPV-associated tumours is well established. HPV16 and others alpha-7 species such as HPV45, HPV59 and HPV68 were strongly associated with ADC compared to SCC.[25]

Based on our reported HR-HPV type distribution, HPV vaccination programmes targeting HPV16 and 18 could prevent up to 77% (potentially more considering cross-protection) of future CC in Côte d'Ivoire. While HPV vaccination uptake is expanding globally, the success of immunisation programmes is hampered by a relatively low uptake of the second dose in these settings.[10] A single-dose instead of two or three doses, as recently recommended by the WHO's Strategic Advisory Group of Experts on Immunisation (SAGE) should help to remove barriers to HPV vaccination, and reduce the predicted health and economic costs of the CC burden.[26] In the absence of long-term post-vaccination surveillance programmes, data from this present study should encourage health decision-makers in West Africa to scale-up population-based strategies to increase vaccine availability and uptake by leveraging funds, increasing supplies, raising awareness and building regional collaborations with stakeholders.

Furthermore, even though some cross-protection is obtained through HPV16/18 vaccination, nonavalent HPV vaccines which cover other frequently detected HR-HPV in women with CC (of which seven are HR-HPV16, 18, 31, 33, 45, 52 and 58), should be supported and made available and accessible.[27] Our study revealed that—regardless of HIV status—at least 90.8% of CC might be prevented if women received a nonavalent vaccine. It is therefore essential to promote second-generation vaccines. Widespread vaccination in SSA with a single-dose nonavalent vaccine is the way to tackle low uptake and support national efforts in the post COVID-19 era.

Finally, efforts to accelerate CC screening implementation based on HPV testing are essential in a health service integration perspective. Findings from this study confirm the benefits for both sexually active women aged 30 to 49 years, and adolescents who are the target of HPV-based prevention strategies. These programmes are now ongoing in many resource-limited areas, emphasising the importance of the WHO's global strategy to accelerate the elimination of cervical cancer as a public health problem.[28] The large proportion of HR-HPV16/18/45 combined, irrespective of HIV status, supports partial genotyping of these three genotypes and represents a triage option to treat positively tested women through HPV-based CC screening programmes.

Potential limitations of our study include a limited sample size of our population that has potentially shaded any effect of HIV infection on HR-HPV distribution through a lack of sufficient statistical power. In addition, the relatively low number of WLHIV that contributed to this analysis was not sufficient to show the association between immunologic status and HR-HPV distribution in WLHIV. Because of their sensitive nature, information such as income, sexual behaviour and tobacco use might have been underreported to treating physicians. To mitigate this risk, oncologists and gynaecologists in charge of data collection were previously sensitise to this risk during training sessions prior to study enrolment and asked to remind all participating women that responses to these questions will have no impact on their access to care. In addition, these data were collected as class or categorical format, rather than discrete values.

The reported distribution of HR-HPV genotypes among CC cases in Côte d'Ivoire 2018 to 2020, confirms the major role of HPV16/18 in CC and should serve to support the ongoing scale-up of HPV16/18 vaccination programmes regardless of HIV

status in a time of universal access to ART. Vaccines targeting additional HR-HPV types, as well as HPV16/18/45-focussed screening programmes, could further reduce future CC incidence in Côte d'Ivoire, both for WLHIV and without HIV.

AUTHOR CONTRIBUTIONS

Simon P. Boni: Collected data under the supervision of Innocent Adoubi, Apollinaire Horo, Aristophane Tanon, Didier K. Ekouevi, Judith Didi-Kouko Coulibaly, Boston Mian, Boris K. Tchounga; Assembly of data, data management and logistical support; Interpretation; Wrote the draft of the article. **Vanessa Tenet:** Assembly of data, data management and logistical support. **Daniëlle A. M. Heideman:** Responsible for histological readings, and/or HPV DNA testing. **Maaike C. G. Bleeker:** Responsible for histological readings, and/or HPV DNA testing. **Isidore D. Mohenou:** Responsible for histological readings, and/or HPV DNA testing. **Tarik Gheit:** Responsible for histological readings, and/or HPV DNA testing. **Gary M. Clifford:** Conceived the study; Interpretation. **Antoine Jaquet:** Conceived the study; Interpretation. All the authors revised and approved the article for a final version, achieved by Simon P. Boni, Gary M. Clifford and Antoine Jaquet. The work reported in the article has been performed by the authors, unless clearly specified in the text.

AFFILIATIONS

[1]PAC-CI Research Program, Abidjan, Côte d'Ivoire
[2]National Cancer Control Program, Abidjan, Côte d'Ivoire
[3]Early Detection, Prevention and Infections Branch, International Agency for Research on Cancer (IARC/WHO), Lyon, France
[4]Gynecology and Obstetrics Department, University Hospital of Yopougon, Abidjan, Côte d'Ivoire
[5]Department of Pathology, Amsterdam UMC, Vrije Universiteit Amsterdam, Amsterdam, The Netherlands
[6]Cancer Center Amsterdam, Imaging and Biomarkers, Amsterdam, The Netherlands
[7]Infectious and Tropical Diseases Department, University Hospital of Treichville, Abidjan, Côte d'Ivoire
[8]Gynecology and Obstetrics Department, University Hospital of Cocody, Abidjan, Côte d'Ivoire
[9]Pathology Department, University Hospital of Cocody, Abidjan, Côte d'Ivoire
[10]University of Bordeaux, National Institute for Health and Medical Research (INSERM) UMR 1219, Research Institute for Sustainable Development (IRD) EMR 271, Bordeaux Population Health Centre, Bordeaux, France
[11]Faculté des Sciences de la Santé, Département de Santé Publique, Université de Lomé, Lomé, Togo
[12]Epigenomics and Mechanisms Branch, International Agency for Research on Cancer (IARC/WHO), Lyon, France
[13]Alassane Ouattara National Center of Oncology and Radiotherapy (CNRAO), Abidjan, Côte d'Ivoire
[14]Elizabeth Glazer Paediatric AIDS Foundation, Yaoundé, Cameroon
[15]Oncology Department, University Hospital of Treichville, Abidjan, Côte d'Ivoire

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CONFLICT OF INTEREST STATEMENT

DH is minority shareholder of Self-screen B.V., a spin-off company of VU University Medical Center (currently known as Amsterdam UMC, location Vrije Universiteit Amsterdam). Self-screen B.V. develops, manufactures and licences high-risk HPV and methylation marker assays for cervical cancer screening and holds patents on these tests. All other authors have no conflict of interest to declare.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

This research has been performed in accordance with the Declaration of Helsinki. Approval from The National Ethics Committee for Life Sciences and Health in Côte d'Ivoire (CNESVS) was obtained (no. 041-18/MSHP/CNESVS-kp), and all enrolled women provided informed and written consent before participating in the study.

ORCID

Simon P. Boni https://orcid.org/0000-0002-0630-9135
Antoine Jaquet https://orcid.org/0000-0002-3426-9492

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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