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MULTILEVEL DETERMINANTS AND MECHANISTIC PATHWAYS OF GYNAECOLOGICAL CANCERS IN SUB- SAHARAN AFRICA: A TRANSLATIONAL FRAMEWORK FOR PREVENTION, EQUITY, AND HEALTH SYSTEM STRENGTHENING

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ABSTRACT

Background: Gynaecological cancers remain a leading cause of morbidity and mortality among women in Sub-Saharan Africa (SSA), with cervical cancer being disproportionately represented. The interplay between biological, environmental, and socioeconomic determinants of the component entities or pathologies underpins this burden, yet mechanistic integration across these domains towards a direction of mitigating this obvious burden remains limited. **Methods:** We conducted a narrative integrative synthesis of epidemiological, molecular, and health systems evidences with the aim of constructing a multilevel framework that links the upstream determinants to downstream carcinogenic pathways. **Findings:** Persistent human papillomavirus infection, when localized to the lower genital tract, particularly in sexually active women or those taken within the reproductive age funnel is the necessary cause of most cervical cancers through viral oncoprotein-mediated inactivation of tumour suppressor genes. Metabolic dysregulation and hyperoestrogenism are key among the upstream determinants that drive endometrial carcinogenesis, while defects in DNA repair pathways, particularly involving BRCA1 and BRCA2 genes, underlie ovarian cancer susceptibility. Environmental exposures shape infection dynamics and metabolic risk, whereas socioeconomic determinants critically govern access to prevention, early detection, and treatment of these neoplastic pathologies. **Interpretation and Conclusion:** The gynaecological cancer burden in Sub-Saharan Africa reflects a convergence and real-time interaction of proximal biological mechanisms and distal structural inequities in basic resource access and distribution especially for women. Effective control requires integrated strategies targeting molecular, behavioural, and health system levels of interventions.

KEYWORDS: Determinants, Pathways, Gynecological cancers, Sub-saharan Africa (SSA), Prevention, Health system strengthening.

INTRODUCTION

The epidemiological transition in sub-Saharan Africa (SSA) is currently defined by a profound and widening disparity in gynaecological cancer outcomes especially in the face of progressively abysmal prognoses, where biological susceptibility intersects with systemic failure.[1,2] While high-income countries (HICs) have witnessed a steady decline in cervical cancer mortality due to robust cytological screening and high-uptake Human Papillomavirus (HPV) vaccination programs,

SSA continues to bear a disproportionate share of the global burden.[3,4] Cervical cancer remains the leading cause of cancer-related mortality among women in the region, reflecting a critical failure to translate established molecular insights into scalable public health successes.[5,6] This "African Paradox" in oncology—where highly preventable and treatable malignant diseases continue to claim thousands of lives annually—demands a multi-dimensional or as stated, a multilevel analysis that goes beyond traditional clinical descriptions.[7,8]

The current burden is not merely a consequence of late-stage presentation but is also rooted in the "syndemic" nature of health in the region.[5,9] The convergence of the infectious disease epoch, characterized by the persistent HIV epidemic, and the rising tide of non-communicable diseases (NCDs) driven by urbanization has created a unique carcinogenic environment.[9,10] In this context, gynaecological malignancies are no longer just biological anomalies; they are now indicators of structural inequity and reproductive health fragility.[7,11] For instance, the persistence of high-risk HPV (hrHPV) genotypes 16 and 18, which drive the vast majority of cervical cancers, is significantly exacerbated by the regional prevalence of human immune deficiency viru (HIV), which compromises the host (human) mucosal immunity and accelerates the genomic integration of viral oncoproteins.[12-14]

Furthermore, the "nutritional transition" in SSA's expanding urban centers is fueling a secondary epidemic of obesity-related gynaecological cancers.**[15,16]** Endometrial cancer, once considered rare in the region, is increasing in incidence as energy-dense diets and sedentary lifestyles drive metabolic dysregulation and chronic hyperoestrogenism.[17,18] This shift illustrates that the gynaecological cancer landscape in SSA is no longer static; it is a moving target that requires an integrated framework encompassing viral oncology, metabolic endocrinology, and genomic stability.[19-21] The regional lack of specialized genetic assay or testing services further obscures the role of hereditary factors, such as BRCA1 and BRCA2 gene mutations, which likely contribute to ovarian cancer clusters that remain undiagnosed until terminal stages.[22-24]

This manuscript proposes a translational framework that bridges the gap between molecular mechanisms and the "fundamental causes" of disease—the socioeconomic and health system determinants.[11,25] By synthesizing evidence from across the IMRAD domains, we demonstrate that effective cancer control in SSA necessitates a shift from siloed clinical care to a lifecycle approach.[6,26] This includes leveraging existing HIV and maternal health platforms for "Screen-and-Treat" scale-up, addressing the critical deficit in radiotherapy and surgical oncology workforce, and advocating for "Metabolic Justice" in urban planning and food distribution or management policy.[21,27-29] Ultimately, realizing the right to health for African women requires an oncology paradigm that is as biologically sophisticated as it is socially grounded.[7,25,30]

Section 1: Biological Determinants and Mechanistic Pathways

As shown in Figure 2, Biological mechanisms in the sub-Saharan African (SSA) context are not merely isolated cellular events but are the proximal manifestations of a complex "double burden" of infectious and metabolic drivers. At the forefront is the molecular pathogenesis of cervical cancer, almost universally driven by high-risk Human Papillomavirus (hrHPV), predominantly serotypes 16 and 18.[1,2,12] The mechanistic core of this transformation lies in the integration of the viral genome into the host DNA, which leads to the constitutive overexpression of the E6 and E7 oncoproteins.[31,32]

The E6 viral oncoprotein targets the p53 tumor suppressor gene for ubiquitin-mediated proteasomal degradation, thereby disabling the cell's ability to initiate apoptosis in response to DNA damage.[33,34] Simultaneously, E7 oncoprotein binds to the hypophosphorylated form of the Retinoblastoma protein (pRb), displacing the E2F transcription factor and forcing the cell into the S-phase of the cell cycle.[34,35] In SSA, this viral oncogenesis is profoundly modulated by the HIV epidemic with its immune targeted effects. HIV-induced systemic immunosuppression, characterized by depleted CD4+ T-cell counts and chronic mucosal inflammation, impairs the local immune environment's ability to clear hrHPV, leading to higher viral loads and a significantly accelerated progression from Cervical Intraepithelial Neoplasia (CIN) to invasive carcinoma.[9,13,14]

Parallel to viral drivers, endometrial cancer in the region is increasingly linked to the "nutritional transition." The mechanism is centered on the "unopposed estrogen" hypothesis.[18,36] Obesity, characterized by an expansion of white adipose tissue, leads to elevated levels of endogenous estrogens via the aromatization of androgens by the enzyme aromatase.[17,37] This chronic hyperoestrogenic state, often coupled with low progesterone levels in sedentary urban populations, triggers continuous endometrial stimulation and proliferation.[16,18] Furthermore, obesity-associated hyperinsulinemia reduces the production of Sex Hormone-Binding Globulin (SHBG), thus increasing the bioavailability of estradiol.[36,38] The activation of the PI3K/Akt/mTOR pathway by insulin and Insulin-like Growth Factor-1 (IGF-1) further synergizes with estrogen signaling to inhibit apoptosis and promote genomic instability.[10,38]

Ovarian cancer in SSA presents a distinct challenge involving DNA repair deficiency. While long- term oral contraceptive use and high parity—common in rural SSA—are traditionally protective, the rise in germline mutations in BRCA1 and BRCA2 genes necessitates a

focus on the Homologous Recombination Repair (HRR) pathway.[19,22] Mutations in these genes result in a reliance on error-prone repair mechanisms like Non-Homologous End Joining (NHEJ), leading to the "incessant ovulation" damage seen in the ovarian surface epithelium.[39,40] This genomic instability serves as the final common pathway where biological susceptibility meets or intersects with the regional lack of specialized genetic screening.[23,24]

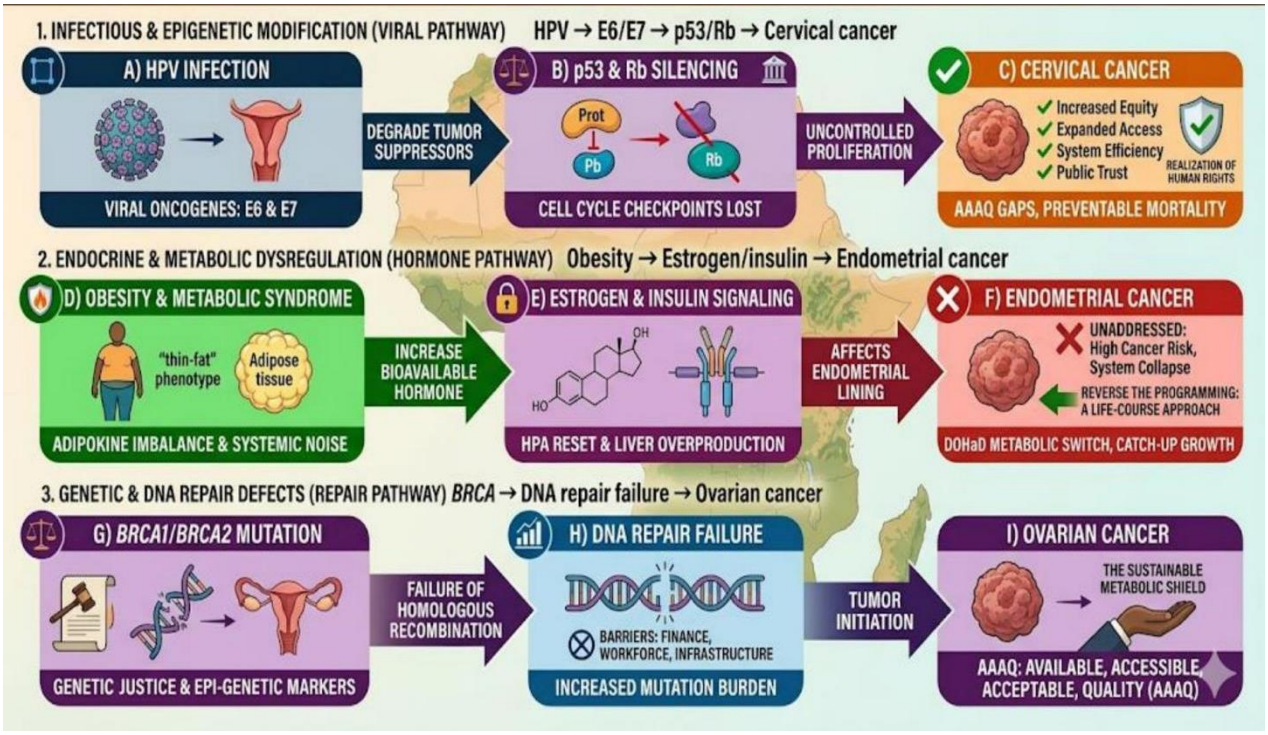

Figure 1: Mechanistic Pathways for Cancer Risk.

HPV → E6/E7 → p53/Rb → Cervical cancer
Obesity → Estrogen/insulin → Endometrial cancer
BRCA → DNA repair failure → Ovarian cancer

Section 2: Environmental and Geographic Determinants

The environmental landscape of SSA acts as a structural modifier of gynaecological cancer risk, dictating the intensity and duration of exposure to biological carcinogens. Urbanization is perhaps the most potent environmental "stressor" or factor currently reshaping regional cancer epidemiology.[10,15] As populations migrate from rural agrarian settings to dense urban centers, there is a marked shift in the "exposome"—the totality of environmental exposures across the life course.[5,41]

This transition is characterized by a shift toward energy-dense, Westernized diets high in refined sugars and saturated fats content, which are primary drivers of the regional obesity epidemic.[15,16] Environmentally, urban centers in SSA often lack the infrastructure for physical activity, creating an "obesogenic" environment that fuels the metabolic pathways of endometrial and breast cancers.[10,17] Furthermore, urbanization alters reproductive environments; we observe a trend toward delayed childbearing and reduced breastfeeding durations in urban professional cohorts, which increases the cumulative number of ovulatory cycles. By extension, this mix of proximal factors induces an oestrogenic milieu, which in turn potentiates the risk for ovarian and endometrial malignancies.[4,30,39]

Geographically, the "infectious ecology" of SSA remains a dominant environmental determinant.[14,42] The high prevalence of hrHPV is not merely a behavioral outcome but is linked to structural factors such as the lack of early-life vaccination coverage and the environmental persistence of co-infections.[12,43] In regions with high malaria and helminth endemicity, the host immune system is in a state of constant "distraction" or exhaustion, which may further diminish the efficiency of the mucosal immune response to HPV integration.[9,30]

Moreover, geographic disparities in the levels of access to clean energy and exposure to household air pollution (from biomass fuel) have been tentatively linked to systemic oxidative stress, which may exacerbate DNA damage in the presence of existing viral oncogenes.[25,27] While the role of ultraviolet (UV) radiation and vitamin D in cancer protection remains a subject of intense geographic study, the high UV index or exposure in SSA does not appear to offset the risk factors of poor screening and late-stage presentation.[5,37] The environmental determinant framework thus suggests that cancer in SSA is not just a biological failure but a result of "toxic" transitions in which traditional protective

factors are lost faster than modern medical infrastructure can be built to tackle the malignant phenotypes.[7,41,44]

Section 3: Socioeconomic and Health System Determinants

The socioeconomic and health system determinants of gynaecological cancers in SSA represent the "fundamental causes" of the region's high mortality-to-incidence ratios.[5,8] Despite the availability of cost-effective prevention tools, the implementation gap or deficit remains profound (See Figure 2 below). For cervical cancer, the "Prevention Paradox" is evident: while the molecular cause is known and preventable through vaccination, HPV vaccine coverage in many SSA nations remains below 30% due to supply chain fragility and high procurement costs.[6,45]

Health system capacity is the primary mediator or even determinant of survival outcomes. In many SSA countries, gynaecological cancers are diagnosed at stages III or IV, and at such stage curative target is often impossible.[3,25] This late-stage presentation is driven by a critical shortage of "Screen-and-Treat" infrastructure. Many rural primary health centers lack the capacity for visual inspection with acetic acid (VIA) or hrHPV DNA testing.[26,46] Furthermore, the path from a positive screen to a definitive biopsy is often hindered by a lack of competent pathology workforce; Sub-Saharan Africa has some of the lowest pathologist-to-population ratios globally, often leading to diagnostic delays of several months.[24,25]

Financially, the burden of cancer care in SSA is a leading cause of catastrophic health expenditure. In the absence of Universal Health Coverage (UHC), the cost of surgery, platinum-based chemotherapy, and radiotherapy must be borne out-of-pocket by patients, many of whom live below the poverty line.[24,29] The regional deficit in megavoltage radiotherapy machines, where some countries have zero functional units, means that even diagnosed patients often cannot access the standard of care for advanced cervical or endometrial cancers.[27,28]

Socioeconomic status (SES) also modulates risk through the lens of "Health Literacy" and cultural stigma.[11,30] Educational barriers often result in a poor understanding of the relationship between HPV and cancer, or the misconception that a diagnosis is an inevitable death sentence, leading to the use of ineffective traditional healers.[25,30] This is compounded by the "Social Stigma" of gynaecological cancers, where symptoms like abnormal vaginal bleeding are often hidden due to cultural taboos regarding female reproductive health.[7,11] Addressing these systemic failures requires more than just clinical intervention; it demands "Metabolic and Social Justice" or simply put equity in nutritional and resource distribution for women.[1,21,47] Policies must integrate cancer care into existing HIV and maternal health platforms to leverage on existing infrastructure, ensuring that the right to health is realized for the most vulnerable women in the region.[6,9,29]

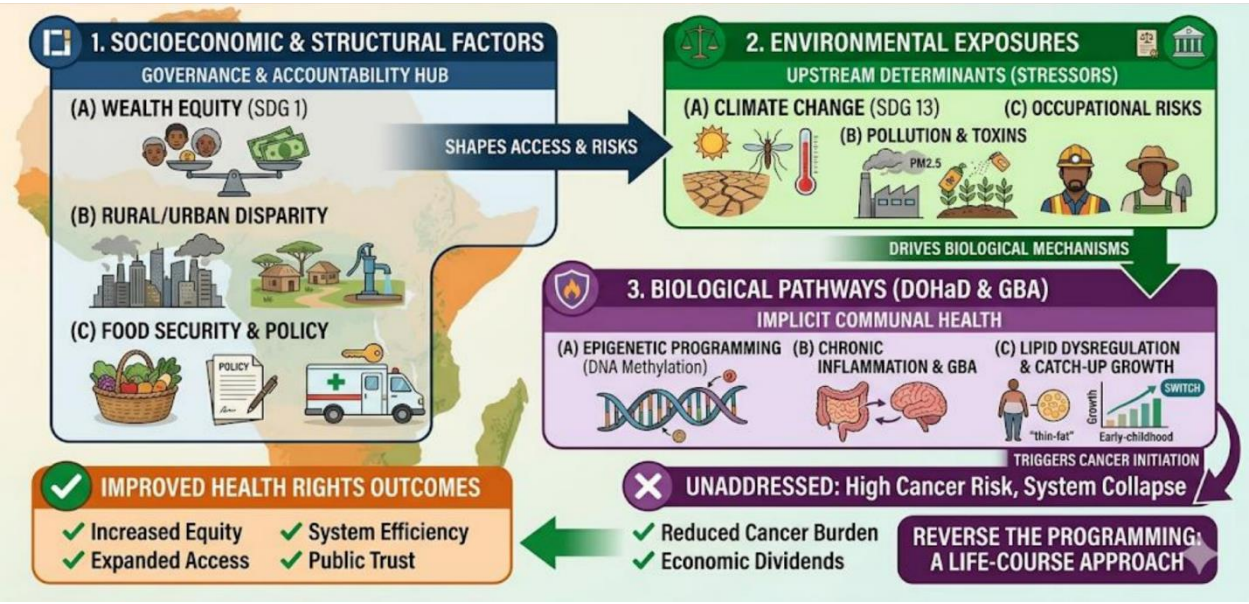

Figure 2: Multilevel Determinants Framework For Cancer Risk.

Socioeconomic → Environmental → Biological → Cancer

Policy and Health System Tables

The following tables and Figure synthesize the translational strategies required to bridge the mechanistic and systemic gaps identified above.

Table 1: Policy Framework for Prevention and Risk Reduction.

Domain	Intervention	Mechanistic Target	Policy Priority
Vaccination	National HPV Immunisation Scale-Up	Viral oncogenesis (E6/E7)	High[6,45]
Metabolic	Obesity and Diabetes Prevention	Hormonal/IGF-1 signaling	High[16,37]
Literacy	Sexual and reproductive health education	HPV transmission Dynamics	Medium[7,11]
Infection	Integrated HIV/HPV co-management	Immune-mediated clearance	High[9,14]

Table 2: Health System Strengthening for Gynaecological Care.

Component	Strategy	Mechanism of Impact	Expected Outcome
Screening	HPV DNA testing as primary screen	Early lesion identification	Reduced mortality[26,46]
Treatment	Regional Radiotherapy Hubs	Tumor volume control	Improved survival[27,28]
Workforce	Specialized Oncology/Nursing/Path0logy workers	Improved diagnostic accuracy	Increased access[24]
Equity	Universal Health Coverage (UHC)	Financial Risk Protection	Reduced Disparities[21,29]

REFERENCES

1. Bray F, Ferlay J, Soerjomataram I, Siegel RL, Torre LA, Jemal A. Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide. CA Cancer J Clin, 2018; 68(6): 394–424. doi:10.3322/caac.21492
2. Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A, et al. Global cancer statistics 2020. CA Cancer J Clin, 2021; 71(3): 209–249. doi:10.3322/caac.21660
3. Arbyn M, Weiderpass E, Bruni L, de Sanjosé S, Saraiya M, Ferlay J, et al. Estimates of cervical cancer incidence and mortality. Lancet Glob Health, 2020; 8(2): e191–e203. doi:10.1016/S2214-109X(19)30482-6
4. Parkin DM, Bray F. Cancer burden in Africa. Cancer Epidemiol Biomarkers Prev, 2006; 15(5): 925–929. doi:10.1158/1055-9965.EPI-05-0676
5. Torre LA, Islami F, Siegel RL, Ward EM, Jemal A. Global cancer disparities. CA Cancer J Clin, 2017; 67(2): 93–115. doi:10.3322/caac.21357
6. WHO. Global strategy to eliminate cervical cancer. Lancet Public Health, 2020; 5: e197. doi:10.1016/S2468-2667(20)30065-0
7. Ginsburg O, Bray F, Coleman MP, Vanderpuye V, Eniu A, Kotha SR, et al. Women's cancer in LMICs. Lancet, 2017; 389(10071): 847–860. doi:10.1016/S0140-6736(16)31392- 7
8. Farmer P, Frenk J, Knaul FM, Shulman LN, Alleyne G, Armstrong L, et al. Cancer control in LMICs. Lancet, 2010; 376(9747): 1186–1193. doi:10.1016/S0140-6736(10)61152-X
9. UNAIDS. HIV global report. Lancet, 2019; 393: 2423. doi:10.1016/S0140-6736(19)31138- 7
10. Ng M, Fleming T, Robinson M, Thomson B, Graetz N, Margono C, et al. Global obesity trends. Lancet, 2014; 384: 766—781. doi:10.1016/S0140-6736(14)60460-8
11. Singh GK, Azuine RE. Socioeconomic disparities. J Community Health, 2016; 41(3): 449–460. doi:10.1007/s10900-015-0101-3
12. de Martel C, Plummer M, Vignat J, Franceschi S. Worldwide burden ofcancer attributable to HPV. Lancet Glob Health, 2017; 5(2): e110–e118. doi:10.1016/S2214-109X(16)30377- 4
13. Palefsky JM. HPV in HIV patients. J Natl Cancer Inst Monogr, 2010; 2010(42): 176—178. doi:10.1093/jncimonographs/lgq004
14. Piot P, Bartos M, Larson H, Zewdie D, Mane P. HIV prevention. Lancet, 2008; 372: 845—859. doi:10.1016/S0140-6736(08)60883-4
15. Popkin BM. Nutrition transition. Nutr Rev. 2017; 75(4): 267—281. doi:10.1093/nutrit/nuw056
16. Colditz GA, Bohlke K. Obesity and cancer. CA Cancer J Clin. 2014; 64: 50—62. doi:10.3322/caac.21205
17. Renehan AG, Tyson M, Egger M, Heller RF, Zwahlen M. BMI and cancer incidence. Lancet, 2008; 371(9612): 569–578. doi:10.1016/S0140-6736(08)60269-X
18. Key TJ, Pike MC. The role of estrogens in endometrial cancer. Eur J Cancer Clin Oncol. 1988; 24(1): 29–43. doi:10.1016/0277-5379(88)90279-4
19. Bowtell DD. Biology of ovarian cancer. Nat Rev Cancer. 2010; 10(11): 803–808. doi:10.1038/nrc2946
20. Jemal A, Bray F, Forman D, O'Brien M, Ferlay J, Center M, et al. Cancer burden Africa. Lancet, 2012; 379(9827): 123–131. doi:10.1016/S0140-6736(11)61347-5
21. WHO. Cancer control planning. Geneva: WHO; 2017. doi:10.1016/S0140- 6736(17)33326-3
22. King MC, Marks JH, Mandell JB. BRCA mutations and cancer risk. Science, 2003; 302(5645): 643–646. doi:10.1126/science.1088759
23. Kuchenbaecker KB, Hopper JL, Barnes DR, Phillips KA, Mooij TM, Roos-Blom MJ, et al. Risks associated with BRCA mutations. JAMA. 2017; 317(23): 2402–2416. doi:10.1001/jama.2017.7112
24. Vanderpuye V, Hammad N, Martei Y, Hoppe RT, Adewole I, El Saghir NS. Cancer care Africa. Lancet Oncol, 2017; 18: e359—e368. doi:10.1016/S1470-2045(17)30272-2
25. Sankaranarayanan R, Swaminathan R, Brenner H, Chen K, Chia KS, Chen JG, et al. Cancer survival LMICs. Lancet Oncol, 2010; 11(2): 165—173. doi:10.1016/S1470-2045(09)70335- 5

26. Sankaranarayanan R. Screening in developing countries. Int J Gynecol Obstet, 2006; 95: S71—S80. doi:10.1016/S0020-7292(06)60017-0
27. Atun R, Jaffray DA, Barton MB, Bray F, Baumann M, Vikram B, et al. Radiotherapy needs globally. Lancet Oncol, 2015; 16(10): 1153–1186. doi:10.1016/S1470-2045(15)00222-3
28. Allemani C, Matsuda T, Di Carlo V, Harewood R, Matz M, Nikšić M, et al. Global cancer survival. Lancet, 2018; 391(10125): 1023—1075. doi:10.1016/S0140-6736(17)33326-3
29. Knaul FM, Arreola-Ornelas H, Méndez O. Cancer financing. Lancet, 2012; 379: 1661—1663. doi:10.1016/S0140-6736(12)60555-3
30. Denny L. Cervical cancer Africa. Cancer Epidemiol Biomarkers Prev, 2012; 21: 1434—1438. doi:10.1158/1055-9965.EPI-12-0331
31. Schiffman M, Castle PE, Jeronimo J, Rodriguez AC, Wacholder S. HPV and cervical cancer. Lancet, 2007; 370(9590): 890–907. doi:10.1016/S0140-6736(07)61416-0
32. Walboomers JM, Jacobs MV, Manos MM, Bosch FX, Kummer JA, Shah KV, et al. HPV is necessary cause of cervical cancer. J Pathol. 1999; 189(1): 12–19. doi:10.1002/(SICI)1096-9896
33. McLaughlin-Drubin ME, Münger K. Oncogenic activities of HPV proteins. Virus Res, 2009; 143(2): 195–208. doi:10.1016/j.virusres.2009.06.008
34. Bosch FX, Lorincz A, Muñoz N, Meijer CJ, Shah KV. HPV and cancer. J Clin Pathol, 2002; 55(4): 244—265. doi:10.1136/jcp.55.4.244
35. Moody CA, Laimins LA. HPV life cycle and cancer. Nat Rev Microbiol, 2010; 8(8): 550–560. doi:10.1038/nrmicro2336
36. Kaaks R, Lukanova A. Energy balance and cancer risk. Nat Rev Cancer, 2002; 2(8): 605–615. doi:10.1038/nrc845
37. Calle EE, Kaaks R. Overweight, obesity and cancer. Nat Rev Cancer, 2004; 4(8): 579–591. doi:10.1038/nrc1408
38. Gallagher EJ, LeRoith D. Insulin and cancer. Endocr Rev., 2010; 31(2): 242–264. doi:10.1210/er.2009-0011
39. Fathalla MF. Incessant ovulation hypothesis. Lancet. 1971; 2(7716): 163. doi:10.1016/S0140-6736(71)92335-X
40. Kurman RJ, Shih IeM. Origin of ovarian cancer. Am J Surg Pathol, 2010; 34(3): 433–443. doi:10.1097/PAS.0b013e3181cf3d79
41. Lortet-Tieulent J, Soerjomataram I, Ferlay J, Rutherford M, Weiderpass E, Bray F. Cancer incidence Africa. Int J Cancer, 2013; 132(4): 863—870. doi:10.1002/ijc.27771
42. Forman D, de Martel C, Lacey CJ, Soerjomataram I, Lortet-Tieulent J, Bruni L, et al. HPV global burden. Vaccine, 2012; 30: F12—F23. doi:10.1016/j.vaccine.2012.07.055
43. Denny L, Herrero R, Levin C, Kim JJ. Cervical cancer prevention in low-resource settings. Vaccine, 2012; 30(Suppl 5): F71–F80. doi:10.1016/j.vaccine.2012.05.091
44. Vaccarella S, Lortet-Tieulent J, Plummer M, Franceschi S, Bray F. Worldwide cervical cancer trends. Lancet Glob Health, 2013; 1: e20—e28. doi:10.1016/S2214-109X(13)70001- 1
45. Drolet M, Bénard É, Pérez N, Brisson M. HPV vaccination impact. Lancet, 2019; 394: 497-509. doi:10.1016/S0140-6736(19)30298-3
46. Sankaranarayanan R, Ramadas K, Qiao YL. Screening strategies. Lancet Oncol, 2014; 15(2): e67–e75. doi:10.1016/S1470-2045(13)70530-9
47. Ferlay J, Colombet M, Soerjomataram I, Mathers C, Parkin DM, Piñeros M, et al. Estimating cancer incidence. Int J Cancer, 2019; 144: 1941—1953. doi:10.1002/ijc.31937
48. WHO Cancer control planning. Geneva: WHO, 2017. doi:10,1016/S01406736(17)33326-3