NATIONAL CANCER SCREENING GUIDELINES
# Table of Contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABBREVIATIONS &amp; ACRONYMS</td>
<td>6</td>
</tr>
<tr>
<td>FOREWORD</td>
<td>9</td>
</tr>
<tr>
<td>ACKNOWLEDGEMENTS</td>
<td>11</td>
</tr>
<tr>
<td>EXECUTIVE SUMMARY OF KEY RECOMMENDATIONS</td>
<td>13</td>
</tr>
<tr>
<td><strong>INTRODUCTION</strong></td>
<td>16</td>
</tr>
<tr>
<td>GUIDELINE OBJECTIVES AND TARGET GROUP</td>
<td>21</td>
</tr>
<tr>
<td>PANEL SELECTION AND COMPOSITION</td>
<td>21</td>
</tr>
<tr>
<td>THE APPROACH</td>
<td>21</td>
</tr>
<tr>
<td>SCOPE OF THE GUIDELINES</td>
<td>21</td>
</tr>
<tr>
<td>REFERENCES</td>
<td>22</td>
</tr>
<tr>
<td><strong>BREAST CANCER SCREENING</strong></td>
<td>24</td>
</tr>
<tr>
<td>INTRODUCTION</td>
<td>24</td>
</tr>
<tr>
<td>RATIONALE FOR SCREENING</td>
<td>24</td>
</tr>
<tr>
<td>RISK FACTORS FOR BREAST CANCER</td>
<td>25</td>
</tr>
<tr>
<td>SCREENING INTERVENTIONS AND FREQUENCY OF SCREENING</td>
<td>27</td>
</tr>
<tr>
<td>TARGET POPULATION</td>
<td>29</td>
</tr>
<tr>
<td>AVERAGE RISK POPULATION</td>
<td>29</td>
</tr>
<tr>
<td>HIGH RISK POPULATION</td>
<td>30</td>
</tr>
<tr>
<td>RISK ASSESSMENT ALGORITHM</td>
<td>32</td>
</tr>
<tr>
<td>SCREENING AT VARIOUS HEALTHCARE LEVELS</td>
<td>33</td>
</tr>
<tr>
<td>BREAST CANCER SCREENING AND REFERRAL ALGORITHM</td>
<td>34</td>
</tr>
<tr>
<td>REFERENCES</td>
<td>35</td>
</tr>
<tr>
<td><strong>CERVICAL CANCER SCREENING</strong></td>
<td>39</td>
</tr>
<tr>
<td>INTRODUCTION</td>
<td>39</td>
</tr>
<tr>
<td>RISK FACTORS FOR CERVICAL CANCER</td>
<td>40</td>
</tr>
<tr>
<td>RATIONALE FOR SCREENING</td>
<td>40</td>
</tr>
<tr>
<td>WHO SHOULD BE SCREENED AND WHEN?</td>
<td>41</td>
</tr>
<tr>
<td>SCREENING METHODS FOR CERVICAL CANCER</td>
<td>41</td>
</tr>
<tr>
<td>HPV TEST</td>
<td>43</td>
</tr>
<tr>
<td>VISUAL INSPECTION METHODS</td>
<td>45</td>
</tr>
<tr>
<td>CYTOLOGY-BASED SCREENING METHODS</td>
<td>48</td>
</tr>
<tr>
<td>SCREENING AT VARIOUS HEALTHCARE LEVELS</td>
<td>49</td>
</tr>
<tr>
<td>RECOMMENDATIONS FOR SPECIAL POPULATIONS</td>
<td>50</td>
</tr>
<tr>
<td>MANAGEMENT OF CERVICAL PRE-CANCEROUS LESIONS</td>
<td>51</td>
</tr>
<tr>
<td>POST-TREATMENT SCREENING</td>
<td>55</td>
</tr>
<tr>
<td>REFERENCES</td>
<td>56</td>
</tr>
</tbody>
</table>
ABBREVIATIONS & ACRONYMS

ACS  American Cancer Society
AFP  Alpha-fetoprotein
AKUH  Aga Khan University Hospital
ASCO  American Society of Clinical Oncology
ASCUS  Atypical Squamous Cells of Undetermined Significance
ASR  Age Standardized Rates
AUA  American Urological Association
BRCA  Breast Cancer Susceptibility gene
BSE  Breast Self-Examination
CA  Cancer Antigen
CBE  Clinical Breast Examination
CEA  Carcinoembryonic Antigen
CHAI  Clinton Health Access Initiative
CHEW  Community Health Extension worker
CHV  Community Health Volunteer
CIN  Cervical Intraepithelial Neoplasia
CIS  Carcinoma In Situ
CML  Chronic Myeloid Leukaemia
CRC  Colorectal Cancer
CT  Computed Tomography
DCBE  Double-Contrast Barium Enema
DNA  Deoxyribonucleic Acid
DNCD  Division of Non Communicable Diseases
DRE  Digital Rectal Examination
EAU  European Association of Urology
EUA  Examination Under Anaesthesia
EBV  Epstein Barr Virus
EFGR  Epidermal Growth Factor Receptors
ER  Estrogen Receptor
ESMO  European Society for Medical Oncology
ESTRO  European Society for Therapeutic Radiation and Oncology
FAP  Familial Adenomatous Polyposis
FELTP  Field Epidemiology & Laboratory Training Program
FIT  Fecal Immunochemical Test
FNA  Fine Needle Aspirate
FOBT  Fecal Occult Blood Test
GSK  Gastroenterology Society of Kenya
HBV  Hepatitis B Virus
HCG  Human Chorionic Gonadotropin
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCP</td>
<td>Healthcare Provider</td>
</tr>
<tr>
<td>HIV</td>
<td>Human Immunodeficiency Virus</td>
</tr>
<tr>
<td>HNPCC</td>
<td>Hereditary Nonpolyposis Colorectal Cancer</td>
</tr>
<tr>
<td>HPV</td>
<td>Human Papillomavirus</td>
</tr>
<tr>
<td>HR-HPV</td>
<td>High-Risk Human papillomavirus</td>
</tr>
<tr>
<td>HRT</td>
<td>Hormone Replacement Therapy</td>
</tr>
<tr>
<td>HSIL</td>
<td>High Grade Squamous Intraepithelial Lesions</td>
</tr>
<tr>
<td>IARC</td>
<td>International Agency for Research on Cancer</td>
</tr>
<tr>
<td>IHC</td>
<td>Immunohistochemistry</td>
</tr>
<tr>
<td>IMCI</td>
<td>Integrated Management of Childhood Illnesses</td>
</tr>
<tr>
<td>JOOTRH</td>
<td>Jaramogi Oginga Odinga Teaching &amp; Referral Hospital</td>
</tr>
<tr>
<td>KAUS</td>
<td>Kenya Association of Urological Surgeons</td>
</tr>
<tr>
<td>KDA</td>
<td>Kenya Dental Association</td>
</tr>
<tr>
<td>KEMRI</td>
<td>Kenya Medical Research Institute</td>
</tr>
<tr>
<td>KENCO</td>
<td>Kenyan Network of Cancer Organizations</td>
</tr>
<tr>
<td>KEPH</td>
<td>Kenya Essential Package for Health</td>
</tr>
<tr>
<td>KESHO</td>
<td>Kenya Society of Hematology &amp; Oncology</td>
</tr>
<tr>
<td>KNBS</td>
<td>Kenya National Bureau of Statistics</td>
</tr>
<tr>
<td>KOGS</td>
<td>Kenya Obstetrical and Gynaecological Society</td>
</tr>
<tr>
<td>LBC</td>
<td>Liquid-based Cytology</td>
</tr>
<tr>
<td>LCIS</td>
<td>Lobular Carcinoma In Situ</td>
</tr>
<tr>
<td>LEEP</td>
<td>Loop Electrosurgical Excision Procedure</td>
</tr>
<tr>
<td>LFT</td>
<td>Liver Function Tests</td>
</tr>
<tr>
<td>LLETZ</td>
<td>Large Loop Excision of the Transformation Zone</td>
</tr>
<tr>
<td>LMICs</td>
<td>Low-and middle-income countries</td>
</tr>
<tr>
<td>LSIL</td>
<td>Low Grade Squamous Intraepithelial Lesions</td>
</tr>
<tr>
<td>M &amp; E</td>
<td>Monitoring &amp; Evaluation</td>
</tr>
<tr>
<td>MOH</td>
<td>Ministry of Health</td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic Resonance Imaging</td>
</tr>
<tr>
<td>MTRH</td>
<td>Moi Teaching &amp; Referral Hospital</td>
</tr>
<tr>
<td>NCCN</td>
<td>National Comprehensive Cancer Network</td>
</tr>
<tr>
<td>NCCP</td>
<td>National Cancer Control Program</td>
</tr>
<tr>
<td>NCCS</td>
<td>National Cancer Control Strategy</td>
</tr>
<tr>
<td>NCDs</td>
<td>Non-Communicable Diseases</td>
</tr>
<tr>
<td>NCI-K</td>
<td>National Cancer Institute - Kenya</td>
</tr>
<tr>
<td>NCR</td>
<td>Nairobi Cancer Registry</td>
</tr>
<tr>
<td>OSCC</td>
<td>Oral Squamous Cell Carcinoma</td>
</tr>
<tr>
<td>PBF</td>
<td>Peripheral Blood Film</td>
</tr>
<tr>
<td>PC</td>
<td>Palliative Care</td>
</tr>
<tr>
<td>PCA</td>
<td>Prostate Cancer Antigen</td>
</tr>
<tr>
<td>PET</td>
<td>Positron Emission Tomography</td>
</tr>
<tr>
<td>PHSK</td>
<td>Public Health Society of Kenya</td>
</tr>
</tbody>
</table>
PR     Progesterone Receptor  
PSA    Prostate Specific Antigen  
RB     Retinoblastoma  
RNA    Ribonucleic Acid  
RT     Radiation Treatment  
SCJ    Squamo-Columnar Junction  
SIOG   International Society of Geriatric Oncology  
STI    Sexually Transmitted Infection  
TBC    Total Blood Count  
TNM    Tumour Node Metastasis  
TRUS   Transrectal ultrasonography  
UEC    Urea, Electrolytes & Creatinine  
UHC    Universal Health Coverage  
UoN    University of Nairobi  
USPSTF United States Preventive Services Task Force  
VIA    Visual Inspection with Acetic acid  
VILI   Visual Inspection with Lugol's iodine  
WHO    World Health Organization
The cancer burden is rising globally, exerting significant strain on populations and health systems at all income levels. In Kenya, cancer is the 3rd leading cause of death after infectious and cardiovascular diseases. The International Agency for Research in Cancer (IARC) GLOBOCAN report for 2018 estimated 47,887 new cases of cancer annually with a mortality of 32,987. This represents close to 45% increase in incidence compared to the previous report that estimated 37,000 new cancer cases annually with an annual mortality 28,500 in 2012.

Breast, cervix uteri, oesophagus, prostate and colorectum are the leading types of new cancer cases in both males and females across all ages, with oesophageal cancer being the leading cause of cancer deaths, followed by cervical cancer and then breast cancer. It is sad to note that 70-80% of cancer patients in Kenya are diagnosed at an advanced disease when it is not amenable to cure; this is part of the justification for developing these screening guidelines.

These National Cancer Screening Guidelines are in line with the implementation of the National Cancer Control Strategy 2017-2022 Pillar 1, which focuses on Prevention, Early Detection and Cancer Screening. It is also based on current evidence and international best practice and includes cancers recommended for screening by the World Health Organization, namely breast, cervical, colorectal, prostate, oral and childhood cancers. Early detection of oesophageal cancers has also been included in response to the related high mortality in Kenya.

The National Cancer Screening Guidelines have been developed through a multi-stakeholder consultative process involving national, county and civil society experts and reviewers and have an aspirational goal in line with Article 43 of the Constitution of Kenya, 2010, which confers on every person the right to the highest attainable standard of health. Their implementation will strengthen Kenya’s primary health care system and will contribute to the attainment of universal health coverage (UHC) ultimately culminating in the attainment of the President Uhuru Kenyatta’s Big Four Agenda.
The goal of screening is to isolate seemingly asymptomatic individuals in the community who have abnormalities that indicate that they could be having a pre-cancerous condition and link them promptly with the appropriate diagnosis, care and treatment. These National Cancer Screening Guidelines will help in reducing the preventable morbidity and mortality due to cancer by improving early detection and contributing to prompt and accurate treatment.

These guidelines are meant to standardize cancer screening, provide operational protocols and improve the outcome of cancer screening and treatment by streaming referral along the levels of care in Kenya. It is my appeal to all cancer stakeholders to work together to support the implementation of National Cancer Screening guidelines towards halting and reversing the increasing burden of cancer in Kenya.

Sicily K. Kariuki (Mrs), EGH
Cabinet Secretary
Ministry of Health
ACKNOWLEDGEMENTS

The Ministry of Health appreciate all those who contributed to the successful development of these guidelines. It involved several meetings, workshops and consultations. The support from the top leadership at the Ministry of Health made this process a success. The office of the Cabinet Secretary, Principal Secretary, Director of Medical Services, Department of Preventive and Promotive Health and the Division of Non-Communicable diseases were supportive throughout the process giving much needed guidance.

We appreciate Takeda Pharmaceuticals and Roche Diagnostics for their financial and technical support during the development of this document. Special appreciation goes to all the experts who participated in the writing and/ or review of these guidelines. They demonstrated commitment and passion throughout the entire process. We also appreciate the contribution of various professional associations, including Kenya Society of Hematology & Oncology (KESHO), Surgical Society of Kenya (SSK), Kenya Association of Radiologists (KAR), Kenya Obstetrical & Gynaecological Society (KOGS), Kenya Association of Urological Surgeons (KAUS), Kenya Dental Association (KDA) and Gastroenterology Society of Kenya (GSK).

We are grateful to Dr. Gladwell Kiarie who was the lead consultant and provided strategic direction during the development process. Special thanks go to Dr. Anne Ng’ang’a, the head of the National Cancer Control Program for providing overall leadership to the whole process and ensuring completion of the document in good time and Dr. Joan-Paula Malenya for her coordination role.

The contribution of the following individuals is much appreciated also: Dr. Joseph Kibachio, Lydia Kirika, Dr. Mary Nyangasi, Dr. Eunice Gathitu, Dr. Valerian Mwenda, Hannah Gitungo, Evans Obaga and Linda Ogol. They portrayed tremendous dedication to the process to ensure Kenya achieves the goal of reduced cancer incidence through population-based prevention services, to which these guidelines will contribute.
Early detection of cancer through screening and early diagnosis remains a key intervention in cancer control. These guidelines will provide a structured framework for offering cancer screening services to health facilities and organizations involved in cancer screening. It is my hope that with the launch of these guidelines, a robust cancer screening program will be in place providing quality screening services that will eventually lead to reduced incidence, early detection, reduced morbidity and mortality from cancer.

I encourage all healthcare providers, partners and stakeholders to make use of these guidelines.

Peter K. Tum, OGW
Principal Secretary
Ministry of Health
EXECUTIVE SUMMARY OF KEY RECOMMENDATIONS

This executive summary highlights the key recommendations from the main text of the guidelines.

Main Recommendations

Breast cancer
Promotion of breast awareness and education of women on breast health are important aspects in early detection of breast cancer. Mammography is the recommended mode of screening; breast magnetic resonance imaging (MRI) may be used in selected high-risk populations. Breast Self-Examination (BSE), clinical breast examination (CBE) and ultrasound are not screening modalities but are complementary to mammography and aid in early diagnosis of breast cancer. Age of starting screening as well as frequency will depend on risk assessment and stratification.

Cervical cancer
The target population for screening is women aged 25 to 49 years. Testing for the human papilloma virus (HPV) is recommended as the primary screening method; visual inspection with acetic acid (VIA) alone, or combined with visual inspection with Lugol’s iodine (VILI) can also be used as primary screening methods where facilities for HPV testing are not yet available, while pap smear may be used in some specified circumstances. Ideally, a same day ‘screen & treat’ approach is recommended, with cryotherapy and/or Loop Electrosurgical Excision Procedure (LEEP) as part of the screening programme.

Colorectal cancer
On average, Screening should start at 45 years; the recommended screening tests are fecal occult blood test (FOBT) for people with average risk and colonoscopy for high risk groups. Frequency of screening is 5 years; high risk groups may require more frequent screening. Genetic testing is recommended for familial colorectal cancer.

Oral cancers
The most effective approach to ensure early diagnosis of oral cancers in Kenya is to offer opportunistic screening, targeting all individuals at risk of developing oral cancer. Recommended screening/early diagnosis methods are visual inspection, imaging, exfoliative cytology and incisional biopsy.

Oesophageal cancer
The goal is to detect precancerous lesions and early cancerous lesions. Screening modality of choice is endoscopy (white light endoscopy, Lugol’s chromoendoscopy or narrow band imaging endoscopy). Targeted screening is advised for people with first degree relatives with biopsy proven oesophageal cancer, asymptomatic people living in high-risk areas, patients treated for head and neck squamous cell carcinoma and patients with history of caustic acid ingestion.
Prostate cancer
There is no role for mass screening for prostate cancer. Screening for prostate cancer should be a highly individualized decision between a client and his caregiver, bearing in mind the client’s values and preferences. The client should be well informed about the benefits and harms of screening. Screening should target men aged 40 years and above of African descent; 55 years and above of Caucasian or Asian origin. Men with a family history of prostate cancer should begin screening at 40 years of age. Patients with a PSA >4ng/ml regardless of other parameters, should be referred to a urologist for further management. The final diagnosis of prostate cancer must be histological based on a biopsy report.

Childhood cancers
The majority of childhood cancers are not amenable to screening, apart from retinoblastoma and other rarer heritable conditions. Furthermore, unlike some adult cancers, childhood cancers are not associated with lifestyle. The emphasis therefore, in childhood cancers is early detection as there is high potential for cure. Screening of childhood cancers is recommended mainly for hereditary retinoblastoma, certain genetic syndromes and in childhood cancer survivors.

Tumor Markers
Tumor markers are produced by cancers or by the body in response to cancer. This therefore means that most tumor markers have no role in cancer screening in the general population. However, tumor markers have a very restricted use in early detection of some cancers. PSA & CA 125 are useful for screening high risk individuals with either a strong family history or with specific risk factors for prostate and ovarian cancers respectively.

This Guideline provides evidence-based recommendations for cancer screening in Kenya. In all instances, a patient-centered approach should be employed in the implementation of the stated recommendations.

Dr Kioko Jackson K., OGW, MBS
Director of Medical Services
Ministry of Health
INTRODUCTION

Background

GLOBAL CANCER BURDEN

Cancer incidence and mortality rates continue to rise globally, with an estimated 18.1 million new cases and 9.6 million deaths in 2018. The detailed cancer patterns in different world regions, however, are complex. Majority of cancers occur in low-and middle-income countries (GLOBCAN 2018; WHO, 2017). The number of new cases is expected to rise by about 70% over the next two decades, with significant and rising economic effects. The direct and indirect economic costs related to the prevention and treatment of the cancer globally were approximately $1.16 trillion in 2010 (WHO, 2017).

Lung cancer is the most commonly diagnosed cancer (11.6% of the total cases) and the leading cause of cancer death (18.4% of the total cancer deaths) among both sexes combined. This is closely followed by female breast cancer (11.6%), prostate cancer (7.1%), and colorectal cancer (6.1%) for incidence and colorectal cancer (9.2%), stomach cancer (8.2%), and liver cancer (8.2%) for mortality. Globally, among males, lung cancer is leading in incidence and mortality, followed by prostate and colorectal cancer (for incidence) and liver and stomach cancer (for mortality). Among females, breast cancer is the leading type of cancer in incidence and mortality, followed by colorectal and lung cancer (for incidence), and vice versa (for mortality); cervical cancer ranks fourth for both incidence and mortality (GLOBCAN 2018).

The incidence and mortality for various types of cancer, however, substantially vary across countries and within each country. This depends on the degree of economic development and advancement of healthcare, associated environmental, sociocultural factors, and lifestyle factors, among others.

Global Incidence & Mortality rates (Age-standardized) for top 10 cancers
It is noteworthy that high-quality cancer registry data which would form the basis for planning and implementing evidence-based cancer control programs, are not available in most low- and middle-income countries (LMICs). Even then, the scanty data is enough to trigger action to prevent and control cancer.

KENYAN SITUATION
In Kenya, cancer is the 3rd leading cause of death after infectious and cardiovascular diseases. The annual incidence of cancer was estimated at 47,887 new cancer cases, with an annual mortality 32,987 in 2018. Among men, prostate, oesophageal and colorectal are the leading cancers, while among women, breast, cervical and oesophageal cancers are most common. The leading cause of cancer death in Kenya is oesophageal cancer contributing 13.2% (4,351 deaths) of cancer mortality. Cervical cancer is the second leading cause of cancer death contributing 10% (3,266 deaths) while breast cancer comes in third at 7.7% (2,553 deaths) (GLOBOCAN, 2018).

Estimated number of new cancers in Kenya among all ages, both males & females, GLOBOCAN 2018

Estimated number of cancer deaths in Kenya among all ages, both males & females, GLOBOCAN 2018
Late-stage presentation when cure is difficult to achieve is a common problem here in Kenya as is the case in many LMICs (WHO, 2018b), where diagnostic and treatment services are inadequate or non-existent (WHO, 2017). Data from Kenyatta national Hospital shows that between 2014 and 2016, approximately 64% of cancer patients were diagnosed at stage III or IV, when treatment for cure is difficult to achieve. Cancer registration and surveillance in Kenya has been suboptimal. Currently, there are two (2) established regional population-based cancer registries in Eldoret and Nairobi covering an estimated 10% of the Kenya population. For childhood cancers, low awareness and stigma amongst parents/guardians and caregivers leads to late presentation of patients to cancer treatment centres (Njuguna, 2016). Amongst other factors that contribute to poor outcomes are limited diagnostic facilities with insufficient equipment, personnel and consumables.

While about a third to half of cancers can be prevented, the cancer burden can be significantly reduced through early detection and management of precancerous conditions. However, there is low uptake of screening services in Kenya. For example, uptake of cervical cancer screening is 16% among women aged 30-49 years, which is disproportionate to the awareness on availability of the screening services which is 47% among women (STEPS survey, 2015; Ng’ang’a et al., 2018). This is unfortunate since some of the leading cancers can be detected early through screening.

The first pillar of the Kenya National Cancer Control Strategy (NCCS) 2017-2022 focuses on Prevention, Early Detection and Cancer Screening. Early detection can result in better treatment outcomes, less morbidity and even lower costs of treatment. It can be achieved through early diagnosis and through screening. For certain types of cancer, screening increases the chances of early detection. Screening programmes can be effective for certain cancers when suitable tests are utilized competently with quality assurance incorporated followed by linkage to diagnosis and treatment.

**EARLY DETECTION OF CANCER**

Early detection of cancer greatly improves the possibility of treatment being successful and cure being achieved. Early detection implies detection of disease at an early, pre-symptomatic stage when a client would have no reason to seek medical care – an intervention referred to as secondary prevention.

**There are two major components of early detection of cancer:**

1. Early diagnosis
2. Screening
EARLY DIAGNOSIS
This is an important public health strategy that can have great impact in Kenya where most patients present at advanced disease stages. It focuses on detection of symptomatic patients as early as possible through the recognition of possible warning signs of cancer in order to take prompt action. It can be achieved by increasing awareness of possible warning signs of cancer, among health care providers and among the general public through education. The aim is to improve treatment outcomes by providing care at the earliest possible stage.

Early diagnosis of cancer involves 3 steps:
1. Improving awareness and access to care
2. Building diagnostic capacity and improving referral mechanisms
3. Improving access to timely cancer treatment by addressing the relevant barriers

The following table shows some signs and symptoms associated with certain cancers that can aid in early diagnosis of these cancers. Health care providers should recognize these as possible warning signs of cancer and take prompt action to diagnose these cancers early.

<table>
<thead>
<tr>
<th>CANCER</th>
<th>SIGNS &amp; SYMPTOMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervix</td>
<td>Post-coital bleeding, excessive vaginal discharge</td>
</tr>
<tr>
<td>Breast</td>
<td>Lump in the breast, asymmetry, skin retraction, recent nipple retraction, blood-stained nipple discharge, eczematous changes in areola</td>
</tr>
<tr>
<td>Colorectal</td>
<td>Change in bowel habits, unexplained weight loss, anaemia, blood in the stool</td>
</tr>
<tr>
<td>Stomach</td>
<td>Upper abdominal pain, recent onset of indigestion, weight loss</td>
</tr>
<tr>
<td>Urinary bladder</td>
<td>Pain, frequent and uneasy urination, blood in urine</td>
</tr>
<tr>
<td>Prostate</td>
<td>Difficulty in urination, frequent nocturnal urination</td>
</tr>
<tr>
<td>Head &amp; neck cancers</td>
<td>Lump in nose, throat, or neck (with or without pain), persistent sore throat, difficulty swallowing (dysphagia), persistent cough, hoarseness or change in voice, ear pain or hearing loss, persistent headaches, persistent bad breath not explained by hygiene, nasal obstruction or persistent congestion, difficulty breathing, frequent nose bleeding or unusual discharge,</td>
</tr>
<tr>
<td>Oral cavity</td>
<td>White lesions (leukoplakia) or red lesions (erythroplakia), growth or ulceration in mouth</td>
</tr>
<tr>
<td>Retinoblastoma</td>
<td>White spot in the pupil, convergent strabismus (in a child)</td>
</tr>
</tbody>
</table>
SCREENING
Cancer screening involves applying simple tests or procedures across a healthy population in order to identify unrecognized cancer disease in individuals before they develop any symptoms of the cancer. The goal of screening is to find asymptomatic individuals who have abnormalities that indicate that they could be having a pre-cancerous condition or a specific cancer and then link them promptly with the appropriate diagnostic care and treatment. Successful screening requires having adequate human resource to perform the screening tests and availability of facilities that can undertake subsequent diagnosis, treatment, and follow-up.

Principles for screening
The following are some of the recommended prerequisites that must be in place before establishing screening programmes:

- The disease prevalence must be high enough to justify the expenditure on screening
- The screening programmes must have been proven to be effective
- Availability of adequate resources such as equipment and human resources to cater for most of the entire target population group should be assured
- Availability of confirmatory diagnostic facilities and treatment facilities for those who screen positive.

It is unlikely that the country will realize the full economic benefit potential of screening through encouraging individuals to seek specific tests regularly or through maintaining cancer detection as part of routine medical practice (WHOa, 2018). Screening, by itself, has no actual preventive value and must be linked to treatment. If such a link cannot be implemented, then the screening programme is likely to have no impact on the incidence of cancer. All the activities along the continuum of patient care must be implemented in a coordinated manner. These include community mobilization, screening, diagnosis, referral, treatment and follow up. In addition, functioning of other key components must be assured, including awareness creation, health education, effective collaboration and networking between providers and different levels of the health care system, a well-functioning quality control and quality assurance programme; monitoring and evaluation, advocacy and resource mobilization.

Implementation of a prevention and screening programme can have positive outcomes in terms of the quality of the health-care facilities and services, including improved infrastructure, updated training of health-care providers, increased health awareness, and establishment of a quality control and quality assurance programme (WHOa, 2018).
GUIDELINE OBJECTIVES AND TARGET GROUP
These guidelines are meant to standardize cancer screening, provide operational protocols and improve the outcome of cancer screening and treatment by streaming referral along the levels of care in Kenya. They are to serve as a general guide for health care providers in selecting the appropriate tests for their patients and should be applied through an individualized patient-centered approach. They are not intended to be a basis upon which patients seeking screening services are denied their right to be screened.

They are designed for use by all cadres of health care providers (doctors, nurses and clinical officers –among others) across the KEPH levels of the health care system. Strengthening of the referral systems is important to ensure optimal services to the clients.

PANEL SELECTION AND COMPOSITION
The guideline development panel was constituted by the Ministry of Health - National Cancer Control Program (NCCP) and comprised program officers from NCCP and subject-matter expert teams from academic institutions, health research organizations, relevant civil society organizations as well as cancer specialists from various cancer treatment centers and representatives from the county departments of health. The panel held several consultative meetings and workshops that culminated in the development of the working draft, which subsequently underwent external review and validation, before publication of this final version of the guidelines.

THE APPROACH
The process started with a desktop review to identify available cancer screening guidelines, both locally and globally and especially in contexts similar to Kenya. Relevant research publications, existing international guidelines and guidance statements from several cancer care organizations globally were also obtained and reviewed in order to formulate these guidelines. Local context was considered and expert opinion employed where there was paucity of evidence. The information is stated in clear, concise statements that can guide cancer screening activities at all levels of healthcare provision in Kenya.

SCOPE OF THE GUIDELINES
These guidelines cover cancer types that are major contributors to cancer morbidity and mortality in Kenya and are amenable to screening as per currently available evidence. These include cervical, breast, colorectal, prostate, oral, childhood and esophageal cancers. They address the early detection of these cancers, including aspects of early diagnosis where relevant.
REFERENCES


World Health Organization 2017: Cancer Control Knowledge into Action-WHO Guide for Effective Programmes: Module 3
BREAST CANCER SCREENING

Introduction
Globally, breast cancer is ranked 2nd in cancer incidence with 2,088,849 cases (accounting for 11.6% of all new cases), while it is 4th in mortality with 626,679 [6.6% of all cancer deaths] (GLOBOCAN, 2018). Epidemiologic studies reveal marked geographic variation worldwide in observed breast cancer burden. Age-standardized incidence rates are higher in North America, Northern Europe, Australia, and New Zealand averaging 84.8 – 94.2 cases per 100,000 persons, compared to estimates of 40.3 per 100,000 women in Kenya (GLOBOCAN, 2018). Some of the variation in breast cancer impact that is observed among countries may be related to differences in their contemporary ethnic composition, such as in Southern Africa (Fregene, 2005).


Overall, breast cancer is the leading cancer in Kenya in incidence with 5,985 new cases, accounting for 12.5% of all new cancer cases, and 20.9% in women alone (GLOBOCAN, 2018). In the same period, it accounted for 9.2% of all cancer deaths, making it the third leading cause of all cancer deaths in the country. Available data shows that majority of breast cancer patients present in late stage, contributing to higher mortality and low overall survival. A study at Kenyatta National Hospital showed that 7.4% were diagnosed in tumor stage I, 33.7% in stage II, 29.7% in stage III, and 21% in stage IV. The study showed that breast lump is the commonest presentation (79.4%), followed by breast pain (26.8%). The average age at presentation was 48 years [range 21-84] (Othieno-Abinya et al, 2018).

RATIONALE FOR SCREENING
The incidence of breast cancer is on the rise, with 5,985 cases in 2018 compared to 4,465 in 2012 (GLOBOCAN, 2018). Screening for early detection therefore is an important aspect in the control of breast cancer. The primary goal of screening is to increase detection of breast cancer in its early stages and hence improve prognosis and reduce mortality.

The goal of these guidelines is to provide guidance on the appropriate use of screening tools for breast cancer and to help physicians, clinicians and women make informed decisions about screening for breast cancer.
Risk Factors For Breast Cancer

- Heredity & Family History
- Mutations
- Previous abnormal biopsy
- Chest wall radiation
- High breast density
- Reproductive history- nulliparity
- Early menarche
- No breastfeeding history
- Hormone Replacement Therapy (HRT)
- Lifestyle – obesity, physical inactivity, tobacco & alcohol

Heredity and Family History:
Having one or two affected first-degree relatives is associated with a higher risk of breast cancer, with a lifetime excess incidence of breast cancer of 5.5% and 13.3% respectively. The increase in risk is greater for younger women and also when the relative was affected at a younger age (Collaborative Group, 2001).

Known Mutations:
Women with BRCA1 and BRCA2 mutations have a cumulative lifetime risk of breast cancer of 57%; these genes are hereditary. It is therefore important to assess cancer history from both the paternal and maternal side (Collaborative Study Group, 2000). Breast cancer and ovarian cancer may also occur in other genetic syndromes. Assessment, counselling and potential genetic testing for these syndromes will be considered by Medical Geneticists or Physicians.

Biopsy Proven Atypical Hyperplasia or Lobular Carcinoma in situ
Women with atypical hyperplasia or lobular carcinoma in situ in previous breast biopsies, have a four-fold increased risk of cancer which persists for at least 25 years (Hartman et al,2005).

Radiation
Women with a history of chest wall radiation as treatment for another cancer have up to a ten-fold increased risk for breast cancer (Terenziani M et al, 2013). The risk of breast cancer due to radiation exposure during mammography is negligible compared with the expected mortality reduction that can be gained through screening (Yaffe, 2011).
Breast Density
Women with extremely dense breasts have about a two-fold increased risk compared to women with breasts of average density (Gierach, 2012).

Hormonal Influences
Women with earlier age of menarche and/or later age of menopause (Collaborative Group, 2012) have an increased risk of breast cancer, mediated in part by the increased number of menstrual cycles and the longer lifetime exposure to estrogen and progesterone.

Reproductive History:
Nulliparity also increases a woman's risk of breast cancer, and every live birth reduces the relative risk by about 7%. Women 30 years or older at the time of their first live birth have a higher risk of breast cancer than women having their first child at a younger age (Nelson, 2012).

Breastfeeding:
Breastfeeding can lower breast cancer risk, especially if a woman breastfeeds for longer than 1 year (Collaborative group, 2002).

Hormone Replacement Therapy:
Prolonged use of combined estrogen-progesterone hormone replacement therapy (HRT) increases the breast cancer by 15% though this returns to baseline within about 2 years of stopping HRT. Estrogen therapy alone increases breast cancer risk as well, but the increased risk is lower than for combined therapy (Beral et al 2011).

Lifestyle risk factors
- **Obesity:** Obesity is associated with an increased risk of postmenopausal breast cancer, as is weight gain throughout adulthood. Obesity also negatively affects prognosis of early stage breast cancer (Ligibel J, 2011).
- **Physical Activity:** Breast cancer risk is reduced by about 25% among physically active women compared to the least active women (Friedenreich CM, 2011).
- **Alcohol Consumption:** Regular consumption of as little as one drink per day elevates the risk of breast cancer by about 4%. (Mandelson et al, 2000). The risk increases steadily with increasing consumption regardless of the type of alcohol consumed (Seitz HK et al, 2012).
- **Tobacco Use:** Studies have demonstrated that there is a causal association between active smoking and second-hand tobacco smoke and breast cancer (Collishaw et al, 2009).
SCREENING INTERVENTIONS AND FREQUENCY OF SCREENING

Mammography

Mammography is the recommended method of screening for women in the average risk population (NCCN 2016).

Mammography is the only screening modality shown to reduce breast cancer mortality. Possible risks of mammography are listed in the table below.

<table>
<thead>
<tr>
<th>Risks of Mammography</th>
</tr>
</thead>
<tbody>
<tr>
<td>False negative results – this gives a false sense of security that may delay diagnosis</td>
</tr>
<tr>
<td>False positive results - associated with anxiety and requiring extra unnecessary tests</td>
</tr>
</tbody>
</table>

Clinical Breast Examination (CBE) & Ultrasound
CBE should be considered as part of a physical examination and used as an opportunity to discuss and educate the woman on breast health. *It should not be considered as a replacement for mammography screening.* Ultrasound, when available and if conducted by a competent clinician, should be considered as an adjunct to CBE in women between 35 and 39 years.

Breast Self-Examination (BSE) and Awareness
BSE is not recommended as a screening method. However, women should be encouraged to be aware and to report changes in their breasts, such as nipple discharge, rash on nipples, inversion, dimpling or new mass in the breast or axilla.

The healthcare provider should discuss and educate the women about their breast health and promote breast awareness.
**Key messages on breast self-examination and breast awareness:**

- Knowledge of what is normal in your breast is important to maintain good breast health
- Discuss breast health and awareness with your healthcare provider
- Report any abnormality noted in your breast
- Self-breast examination and clinical breast examination are complementary but do not substitute mammography as screening tool
- Asymptomatic women above 40 years require a baseline mammography screening
- Breast cancer also occurs in men though rarely. They need to have breast awareness and not routine screening

**Magnetic Resonance Imaging (MRI):**
MRI is not recommended for routine screening the average risk population. MRI may be used for screening in select high-risk populations or in specific circumstances as determined by a clinician such as previous lumpectomy, radiation or trauma to breast.

**Ultrasound:**
Ultrasound is not recommended for routine screening for the average risk population. It may be used to complement mammography in situations where patients have increased breast density.

**Other Methods**
Tomosynthesis, thermography, elastography and PET scans are not recommended for screening of breast cancer. Tomosynthesis may be used in specific circumstances to complement mammography as may be determined by a radiologist.

**BSE, CBE and ultrasound are not screening modalities, but they aid in early detection of breast cancer**
TARGET POPULATION
The target population for screening will depend on the risk of the patient, which could be defined as high risk or average risk as defined below (NCCN 2016).

Risk Assessment
An assessment of risk for breast cancer should be done for all women considering age, medical history, family history, and other associated risks in determining her breast cancer screening needs. This guideline recommends the Tyrer-Cuzik Model for Risk assessment model for assessment of risk of developing breast cancer (see Annex). Assessment of risk stratifies women into two risk categories as follows:

- Average Risk Population - More than 80% of breast cancer occurs in women in the average risk population.
- High Risk Population
  - Women Requiring More Intensive Screening
  - Criteria for Referral to Medical Genetics

AVERAGE RISK POPULATION
The average risk population, is defined as that population of women who do not exhibit any of the risk factors that define the high-risk population. The clinician should discuss the benefits and risks of screening specific to each age group. (NCCN 2016).

Exclusions:
- Women with signs and symptoms suggestive of breast cancer
- Women with a previous diagnosis of ductal carcinoma in situ or invasive breast cancer
- Men

Recommendations per age category for Women with average risk are provided below.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Recommendation</th>
<th>Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>25 - 34 years</td>
<td>CBE every 3 years</td>
<td>1 to 3 years</td>
</tr>
<tr>
<td></td>
<td><em>Mammogram not recommended</em></td>
<td></td>
</tr>
<tr>
<td>35 - 39 years</td>
<td>CBE and Ultrasound OR mammography*</td>
<td>1 to 3 years</td>
</tr>
<tr>
<td>40 - 55 years</td>
<td>CBE + mammography</td>
<td>Annual</td>
</tr>
<tr>
<td>56 - 74 years</td>
<td>CBE + mammography</td>
<td>Every 2 years</td>
</tr>
<tr>
<td>75 years and older</td>
<td>Consider individual health factors and woman’s preference to continue screening</td>
<td>Discuss with patient</td>
</tr>
</tbody>
</table>

Notes:
* The balance of benefits and risks is not great enough to recommend routine screening. Clinical judgment may be used to adjust the frequency of screening considering individual differences.

Women who have had surgery for breast augmentation, breast reduction or sex-reassignment should follow the same recommendations below for mammographic screening as those in the average risk population. The clinician should clearly state presence of breast implants in the mammography requisition form.
HIGH RISK POPULATION

Women in the high-risk population require more intensive screening and/or genetic counseling. Women with the following characteristics are classified as high risk for breast cancer. Women who do not fulfill any of the four criteria should be classified in the average risk group.

- Affected first degree relatives
- Previous abnormal breast biopsy
- Previous chest wall radiation
- Previous breast cancer

The screening recommendations for these are as follows:

1. Women with one or two first degree relatives with invasive breast cancer, but who do not meet the criteria for referral to Medical Genetics (See criteria below).
   - CBE - starting at age 25 years
   - Annual mammography starting 10 years younger than the youngest case in the family, but no earlier than age 25 and no later than age 40;
   - Complementary imaging like ultrasound and MRI in addition to the above where justified.

2. Women with a breast biopsy showing atypical hyperplasia or lobular carcinoma in situ and following surgical management to rule out invasive carcinoma:
   - CBE every 6-12 months
   - Annual mammography

3. Women with a history of chest wall radiation (i.e. mantle radiation for treatment of Hodgkin's lymphoma) at age 30 or younger:
   - Annual mammography and MRI starting 5 years after radiation given, but starting no earlier than age 25 and no later than age 40
   - Annual CBE

4. Women with previous breast cancer require screening of contralateral breast
   - CBE every 6-12 months
   - Annual mammography
Criteria for referring high risk women for genetic counseling.
Some women in the high-risk population will require referral for genetic testing and counseling.
These include the following:

1. An individual with a number of relatives with breast and/or ovarian* cancer (e.g., three or more cases) in two or more generations, at least one case with onset under the age of 50
2. Bilateral primary breast cancer
3. Breast cancer at age 35 or younger
4. Breast cancer that is hormone receptor negative and HER2 negative (triple negative), age 60 or younger
5. Primary breast and primary ovarian cancer in the same individual
6. Male breast cancer, age 65 or younger, or at any age if with close familial history of breast cancer
7. Confirmed BRCA1 or BRCA2 mutation in the family
RISK ASSESSMENT ALGORITHM

Asymptomatic woman

Risk assessment

Average risk:
- No personal history of breast cancer
- No family history of breast cancer
- No genetic mutation known to increase risk of breast cancer
- No history of chest radiation therapy before age 30

High risk:
- Affected first degree relatives
- Previous abnormal breast biopsy
- Previous chest wall radiation
- Previous breast cancer

ALL AGES: Breast awareness & education
- 34 years and below: CBE every 3 years
- 35-39 years: CBE and ultrasound OR mammography every 1 - 3 years
- 40-55 years: Annual CBE + mammography
- 56-74 years: Annual CBE + mammography every 2 years
- 75 years and older: Shared decision-making on

Screening:
- 1 or 2 first degree relatives with invasive breast cancer: CBE from age 25 years, mammography ±MRI/US starting from 10 years earlier than youngest case in the family (not earlier than 25 years, not later than 40 years of age)
- Previous abnormal breast biopsy (Atypia or lobular CIS): CBE every 6-12 months, mammography annually
- Previous chest wall radiation: annual mammography/MRI + CBE 5 years after radiation given (not earlier than 25 years, not later than 40 years of age)
- Previous breast cancer: Annual mammogram for contralateral breast

Genetic counseling:
- Multiple relatives with breast and/or ovarian cancer, 2 or more generations, at least one under age of 50 years
- Bilateral breast cancer
- Breast cancer at age 35 years or younger
- Triple negative breast cancer, age 60 years or younger
- Primary breast and ovarian cancer in the same individual
- Male breast cancer: ≤ 65 years, or first degree relative with breast cancer
- Confirmed BRCA1 or 2 in the family
### SCREENING AT VARIOUS HEALTHCARE LEVELS

The table below shows the breast cancer screening activities to be performed at the various levels of service delivery of Kenya Essential Package for Health (KEPH):

<table>
<thead>
<tr>
<th>LEVEL</th>
<th>TYPE</th>
<th>CADRES</th>
<th>ACTIVITIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Community</td>
<td>Community Health Volunteers (CHVs) &amp; CHEWS</td>
<td>Breast awareness Mobilization</td>
</tr>
<tr>
<td>2</td>
<td>Dispensary</td>
<td>Nurses</td>
<td>Breast awareness Mobilization, CBE</td>
</tr>
<tr>
<td>3</td>
<td>Health Centres</td>
<td>Clinical officers, Nurses</td>
<td>Breast awareness Mobilization, CBE</td>
</tr>
<tr>
<td>4</td>
<td>Sub-county</td>
<td>Nurses, Clinical officers, Medical officers, General surgeon, Radiographers, Sonographers, Radiographers, Radiologist, Pathologist, Oncologist</td>
<td>Breast awareness Mobilization, CBE, Ultrasound, Mammogram</td>
</tr>
<tr>
<td>5</td>
<td>County/Regional</td>
<td>Nurses, Clinical officers, Medical officers, General surgeon, Radiographers, Sonographers, Radiographers, Radiologist, Pathologist, Oncologist, Oncologist</td>
<td>Breast awareness Mobilization, CBE, CBE/ BSE/ Biopsy, Mammogram, Ultrasound, CBE/BSE, Mammography, CBE/Biopsy, Reporting, Treatment</td>
</tr>
<tr>
<td>6</td>
<td>Referral</td>
<td>Nurses, Clinical officers, Medical officers, Breast surgeon, Radiographers, Sonographer, Radiologist, Pathologist, Oncologist</td>
<td>Breast awareness Mobilization/ Breast awareness, CBE, CBE/ Treatment, CBE/Biopsy, Surgery, Mammogram, Ultrasound, CBE/BSE, Mammography, CBE/Biopsy, Reporting, Treatment/ Monitoring</td>
</tr>
</tbody>
</table>
BREAST CANCER SCREENING AND REFERRAL ALGORITHM
REFERENCES


Breast Cancer Screening. Information available at: www.ScreeningforLife.ca/breastcancer


CERVICAL CANCER SCREENING

Introduction
Cervical cancer is a consequence of long-term infection with human papillomavirus (HPV). Globally, it ranks 4th in both incidence and cancer-related mortality amongst women with an estimated 569,847 new cases new cases and 311,365 deaths annually. It accounts for 13.1% of all new female cancers globally. In Eastern Africa, cervical cancer remains the most common cancer in women with estimated age-standardized incidence and mortality rates of 40.1 and 30.0 per 100,000 respectively (GLOBOCAN, 2018).

In Kenya, cervical cancer contributes 5,250 (12.9%) of the new cancer cases annually and 3,286 (11.84%) of all cancer deaths annually. It is the leading cause of cancer related deaths in Kenya and the 2nd most common cancer among females. (GLOBOCAN, 2018).

According to the World Health Organization (WHO), over 1 million women worldwide are currently living with cervical cancer, many of whom have no access to health services for prevention, curative treatment or palliative care. As a result, many present late when treatment is more difficult and expensive and chances of cure are abysmal. Screening allows for treatment in the asymptomatic precancerous stage (WHO, 2014). Unfortunately, according to the Kenya STEPwise survey for NCDs Risk Factors 2015 report (KNBS, 2015), only 16.4% of women aged 30 – 49 years had ever been screened for cervical cancer.

HPV is the primary cause of 99.7% of all cervical cancers and is sexually transmitted. Infection with one or more of the 15 high-risk oncogenic types (types 16, 18, 31, 33, 35, 39, 41, 51, 52, 56, 58, 59, 66, 67 and 68) usually results in invasive cervical cancer after 10-20 years. Globally, about 70% of all cases of cervical cancer are caused by HPV types 16 and 18. With the increasing availability of vaccines against high-risk HPV, there exists great potential to reduce the incidence of cervical and other anogenital cancers (ACCP 2004, WHO 2006).

The lifetime risk for HPV infection among sexually active women is 50-70%. By the age of 50 years, at least 80% of women will have acquired genital HPV infection. Fortunately, over 80% of HPV infections are transient, asymptomatic and resolve spontaneously in 2-3 years due to the natural cell-mediated immunity, hence the majority of women who get infected with high-risk oncogenic HPV types do not develop cervical cancer. Of the 20% of infections that persist, the HPV viral gene is incorporated into the DNA of cervical cells stimulating abnormal cell division. This may cause mild cyto-
logical abnormalities and/or mild cervical intraepithelial neoplasia (CIN 1), which usually clears spontaneously in about 60% of women in 2-3 years. In about 40% of women, the abnormalities progress to high grade squamous intraepithelial lesions (HSIL) or CIN 2/3 and carcinoma-in-situ (CIS) that subsequently progress to invasive cancer. About 40% and 30% of CIN 2 and CIN 3, respectively spontaneously regress to normal.

The presence of additional co-factors is necessary for the HPV infection to progress to invasive cervical cancer is implied by the fact that not all persistent HPV infections progress to cervical cancer (Mati, 1984; Berraho, 2017)

RISK FACTORS FOR CERVICAL CANCER:

- Early initiation of sexual intercourse
- Having multiple sexual partners
- Having a sexual partner with multiple sexual partners
- Co-infection with other sexually transmitted infections, such as Chlamydia trachomatis and herpes simplex virus type 2
- Multiparity
- Immunosuppression due to HIV/AIDS infection
- Tobacco use

RATIONALE FOR SCREENING

Screening programmes continue to have a vital role, allowing for early detection and treatment in order to achieve a maximal impact on cervical cancer prevention. The natural history of cervical cancer is many years to decades, with a long precancerous, preclinical phase, allowing for testing (screening) for precancerous lesions and cancer. When screening detects precancerous lesions, these can easily be treated and cancer avoided. Screening can also detect cancer at an early stage, enabling women to receive treatment when it is highly effective (WHO, 2014).

The increasing availability of HPV vaccination for girls and the potential for reduction of the possibility of developing cervical cancer later in life, however, does not eliminate the need for regular screening when women get older.

The success of a screening programme in reaching its aims is dependent on achieving adequate coverage. While the screening programme will be introduced incrementally depending on health service capacity, the ultimate goal is to screen at least 70% of women, nationally, within the target age group within 10 years of initiating the programme (IAEA, 2016).
WHO SHOULD BE SCREENED AND WHEN?

Any woman who has ever had sexual intercourse is eligible for cervical cancer screening.

The target population for screening is women aged 25 to 49 years.

Women aged 50-65 years are still at risk of cervical cancer and can therefore receive screening every 5 years on individual resources.

Screening interval is 5 years among women who test negative for HIV.

For HIV positive clients and other special groups refer to special group section.

SCREENING METHODS FOR CERVICAL CANCER

1. HPV testing is recommended as the primary screening method for women above 30 years of age.

2. Where HPV testing is not yet available, or loss-to-follow-up is a risk, then Visual Inspection with Acetic acid (VIA) or Visual Inspection with Acetic acid and Visual Inspection with Lugol’s iodine (VIA/VILI) is recommended as the primary screening method.

3. Pap smear is recommended as a primary screening method in the following situations:
   a. For women not eligible for VIA or VIA/VILI because their squamo-columnar junction (SCJ) is not visible, and HPV screening not accessible.
   b. As a primary test in women under 30 years of age.
   c. As a co-test with HPV in HIV positive women where the resources are available.
Molecular HPV testing methods are based on the detection of DNA from high-risk HPV types in vaginal and/or cervical samples. Since persistent HPV infection is the cause of nearly all cases of cervical cancer, a positive test result indicates that she may have an existing lesion or may be at risk for future pre-cancer and cancer.
HPV TEST

HPV testing algorithm

Source: WHO Guideline for Screening and Treatment of Precancerous Lesions for Cervical Cancer Prevention 2013
Who should be tested?

HPV testing should be reserved for women over the age of 30 years. Testing women younger than 30 years old is not advised because many young women are infected with HPV most of which will be spontaneously resolved by age 30 years. This will therefore lead to a high number of false positive results resulting in unnecessary procedures which overwhelm the health system, with little impact on the incidence.

How to screen

HPV sample collection can be done by the client (self-collected) or by a health provider according to the manufacturers’ instructions.

A health-care provider can collect a sample of cells with or without a speculum, by inserting the provided swab or other appropriate device deep into the vagina, and then placing it in a container with a preservative solution.

For self-collected samples, the woman can be given the sample-collection kit with instructions for use. This strategy offers greater convenience to women and can be implemented at substantially lower cost to the healthcare system.

Follow-up after Results:

- For woman who is High-risk HPV (HR-HPV) negative, re-testing should be done after 5 years (after 2 years for HIV positive).
- If the test is HR-HPV positive, colposcopy, VIA or VIA/VILI and further management is advised (See algorithm for HPV Test)
VISUAL INSPECTION METHODS

VIA/VILI algorithm

VIA or VIA/VILI

Negative
Re-screen every 5 years, annually if HIV positive

Positive

Suspicious for cancer
Refer for appropriate diagnosis and

Eligible for Cryotherapy (or thermo-coagulation): treat

Not eligible for Cryotherapy (or thermo-coagulation): treat with LEEP

Post-treatment follow-up screening after 1 year (see algorithm for post-treatment screening)

Source: WHO Guideline for Screening and Treatment of Precancerous Lesions for Cervical Cancer Prevention 2013
Visual Inspection With Acetic Acid (VIA)

Description
Visual inspection with acetic acid (VIA) is a method for detecting early cell changes that are visible when using a speculum to inspect the cervix with the naked eye after applying dilute (3–5%) acetic acid to it. It requires training and supervision of primary care providers, as well as ongoing quality control and quality assurance. It is quite inexpensive, utilizes locally sourced supplies (vinegar and cotton), and does not require laboratory services. It can be performed by trained providers, with adequate visual acuity, at any level of the health system.

VIA is appropriate to use in women whose squamocolumnar junction (SCJ) is visible, typically in those younger than 50. This is because the SCJ gradually recedes into the endocervical canal when menopause occurs, making it possible to miss lesions when relying on visual inspection.

How to screen?
The provider performs a speculum examination, identifying the SCJ and carefully inspecting the cervix for visual signs suspicious for cancer or pre-cancer. A 3–5% acetic acid solution is liberally applied to the cervix with a large cotton swab. After removing the cotton swab, the provider waits for at least one minute. Acetowhite changes on the cervix indicate likely cervical pre-cancer or cancer. If these changes are seen in the transformation zone and have well-defined borders, they are considered a positive result. If no persistent acetowhite changes are noted, a negative result is reported. The one-minute waiting time allows for any areas that became faintly white due to inflammation or physiological cell changes (metaplasia) to recede.

Follow-up after Results:
- For woman who is VIA negative, re-testing should be done 5 years. If HIV positive, test every year.
- If the test is VIA positive, treat as appropriate (See algorithm)

VIA testing can detect both early changes and those representing more advanced pre-cancer. A single visit approach using the ‘screen & treat’ strategy is recommended to avoid loss to follow up. If the cervix shows any unusual signs or the provider suspects cancer, the patient can be referred for further diagnostic tests (colposcopy and/or biopsy).
Visual Inspection With Acetic Acid (VIA) And Visual Inspection With Lugol’s Iodine (VILI) Testing

Description
Visual inspection with Lugol’s iodine (VILI) - also known as Schiller’s test, uses Lugol’s iodine instead of acetic acid. VILI should only be done after VIA in a co-test strategy as it improves the specificity of the testing. Many guidelines now recommend the use of VIA alone as there is little difference in the sensitivity and specificity when combined. However, this guideline recommends the continued use of both VIA and VILI as a co-test as the shift may affect quality of results.

How to screen?
VILI involves looking at the cervix after swabbing it with Lugol’s iodine. Squamous epithelium contains glycogen, whereas precancerous lesions and invasive cancer contain little or no glycogen. Iodine is glycophilic and is taken up by the squamous epithelium, staining it mahogany brown or black. Columnar epithelium does not change color, as it has no glycogen. Immature metaplasia and inflammatory lesions are at most only partially glycogenated and, when stained, appear as scattered, ill-defined uptake areas. Precancerous lesions and invasive cancer do not take up iodine (as they lack glycogen) and appear as well-defined, thick, mustard or saffron yellow areas.

Results interpretations
- VIA/VILI test is interpreted as positive if either of the tests is positive
- If the test is VIA/VILI positive, treat as appropriate (See VIA/VILI algorithm)
- For woman who is VIA and VILI negative, retest after 5 years. If HIV positive, retest every year.

Exclusion Criteria for VIA/VILI
- Women who are very ill
- Women who are in 2nd & 3rd trimester of pregnancy
- Women less than 6 weeks after delivery
- Women with cauliflower-like growth or ulcer; fungating mass
- Women with previous history of treatment of cancerous lesions
- Women with known allergy to acetic acid
- Women with a history of total hysterectomy
Cytology-based screening involves taking a sample of cells from the entire transformation zone. The cells are either fixed on a slide at the facility (Pap smear) or placed in a transport medium (liquid-based cytology- LBC) and then sent to the laboratory for microscopic examination. Well-implemented cytology programmes have successfully prevented cervical cancer in developed countries; however, they require highly skilled personnel and logistics which have been challenging in low-resource settings.

How to screen
Collection of a cytology sample requires a speculum and adequate lighting to visualize the entire surface of the cervix. The provider takes specimens from the face of the cervix and the endocervix using a spatula or brush and transfers the specimen to a slide (Pap smear) or a preservative solution (LBC). The sample must be appropriately labelled and transported to the laboratory, where expert cytotechnologists are needed to process and interpret it. If abnormal cells are seen on microscopic examination, the extent of their abnormality is classified using the Bethesda System.
SCREENERING AT VARIOUS HEALTHCARE LEVELS

<table>
<thead>
<tr>
<th>HEALTHCARE LEVEL &amp; PERSONNEL</th>
<th>VIA</th>
<th>HPV</th>
<th>PAP SMEAR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 1</td>
<td></td>
<td>+ self-collected sample collection</td>
<td></td>
</tr>
<tr>
<td>Level 2</td>
<td>+</td>
<td>+ sample collection</td>
<td></td>
</tr>
<tr>
<td>Level 3</td>
<td>+</td>
<td>+ sample collection</td>
<td></td>
</tr>
<tr>
<td>Level 4</td>
<td>+</td>
<td>+ sample collection</td>
<td>+</td>
</tr>
<tr>
<td>Level 5</td>
<td>+</td>
<td>+ sample collection</td>
<td>+</td>
</tr>
<tr>
<td>Personnel required</td>
<td>Trained nurse, clinical officers, medical officers, gynecologist</td>
<td>Sample collection: Client, Nurse, Clinical officer, Medical officer</td>
<td>Trained nurse, clinical officers, medical officer, gynecologist</td>
</tr>
</tbody>
</table>

KEY POINTS

- Screening all women in the target age group, followed by treatment of detected precancerous lesions can prevent the majority of cervical cancers.
- Decisions on which screening and treatment approach to use in a particular county or health-care facility are based on various factors, including, potential for loss to follow-up, cost, and availability of the necessary equipment and human resources.
- Every woman in the target age group (25-49 years) should have a cervical cancer screening test performed at least once when most benefit can be achieved.
- HPV testing is recommended as the primary screening method
- Where HPV testing is not yet available, or loss-to-follow-up is a risk, then Visual Inspection with Acetic acid (VIA) or Visual Inspection with Acetic acid and Visual Inspection with Lugol’s iodine (VIA/VILI) is recommended as the primary screening method
- A “screen-and-treat” approach is recommended
- Any suspected cancer case after screening should immediately be referred for diagnosis and treatment of cancer.
RECOMMENDATIONS FOR SPECIAL POPULATIONS:

Women who are HIV positive or immunosuppressed for any other reasons.
- Begin cervical cancer screening at the point of diagnosis or at 25 years, whichever comes earlier.
- Screening should continue throughout their lifetime.
- Screening frequency should be yearly if using VIA or VIA/VILI, every 2 years if using HPV testing and yearly if using cytology.

Women who are pregnant
- Screening can be done during the 1st trimester.
- Treatment for precancerous lesions should NOT be performed during pregnancy.
- For suspicious lesions in pregnancy, a biopsy can be done at any trimester by an obstetrician/gynecologist.

Post-partum women
- Cervical cancer screening can commence 6 weeks after delivery.

Women who have had total abdominal hysterectomies (with no history of ≥CIN2)
- Screening should be discontinued in women who have received a total hysterectomy for benign causes with no history of gynecological malignancy.
- Women who have received a subtotal hysterectomy (with an intact cervix) should continue to receive routine screening.

NOTE

Women who have received HPV vaccination
- Women who have been vaccinated should receive routine screening as per the national guidelines above.

Women 50-64 years:
- Screening should be done at 5-year intervals on an individualized basis using HPV and cytology methods.

Women 65 years and above
- Screening is not recommended.
MANAGEMENT OF CERVICAL PRE-CANCEROUS LESIONS

Treatment of precancerous lesions is a key consideration for the success of any cervical cancer prevention and screening program. A ‘screen and treat’ strategy is usually composed of two phases; the screening test followed by treatment of cervical intraepithelial lesions. Most screen-and-treat strategies to prevent cervical cancer will usually involve treatment with cryotherapy, or LEEP when a patient is not eligible for cryotherapy. Other treatment methods include cold knife conization and thermo-coagulation.

Points to note:

- Before treatment, ALL women who have screened positive with any test, especially HPV testing, should undergo VIA to determine their eligibility for treatment (i.e. cryotherapy versus LEEP) and to rule out large lesions of cervical cancer. Women who are VIA positive will then be treated while those who are VIA negative will not be treated.
- Women presenting for treatment of pre-cancerous lesions should be offered HIV counseling and testing.
- ALL women with suspected cancer should be referred immediately for colposcopy with biopsy and further management by specialists.
- All women who have had treatment for precancerous lesions should receive post-treatment follow-up screening at one year after the treatment.

A) CRYOTHERAPY

This is an ablative form of treatment for precancerous lesions of the cervix which freezes cells using a cryoprobe with a tip made of highly conductive metal (usually silver and copper), that makes direct surface contact with the ectocervical lesion. Carbon dioxide or Nitrous oxide are usually the coolants of choice. Cells reduced to −20°C for one or more minutes will undergo cryonecrosis.

Cryotherapy is highly effective with cure rates of 85-90% for lesions occupying less than 75% of the cervix; however, for larger lesions the cure rate is reduced. When cryotherapy is indicated, only healthcare providers (including nurses or midwives) trained in cryotherapy should perform the procedure.
Cryotherapy Procedure:

1. Explain why the treatment is recommended and describe the procedure including side effects and reassure her.
2. Check that instruments and supplies are available and arrange on high level disinfected tray. Check that cryotherapy instrument is ready to use, that gas (CO₂) is turned on at the cylinder and the pressure reads at least 40–70 kg/cm². Set timer to 0. Insert high-level disinfected cryotip into protective sleeve. Remove protective cover from end of probe.
3. Check that the woman has emptied her bladder if more than 30 minutes since VIA test. Help her onto examining table and once in lithotomy position, drape her.
4. Wash hands thoroughly with soap and water and dry. Wear a pair of examination or high-level disinfected surgical gloves on both hands.
5. Insert speculum and adjust the speculum so that the entire cervix can be seen. Fix the speculum blades in the open position so that the speculum will remain in place with the cervix in view.
6. Move the light source so that you can see the cervix clearly. Use a clean cotton swab to remove any discharge, blood or mucus from the cervix. Dispose of swab.
7. Identify the cervical os, SCJ and site and size of the lesion. Apply dilute acetic acid with a clean swab so that lesion can be seen clearly.
8. Point probe at ceiling. Press freeze button for 1 second and then defrost button for 1 second.
9. Screw cryotip with sleeve onto end of probe.
10. Apply the cryotip to the cervix ensuring that the nipple is placed squarely onto the os. Check to be sure the cryotip is not touching the vaginal walls.

<table>
<thead>
<tr>
<th>Exclusion Criteria for Cryotherapy</th>
<th>Eligibility Criteria for Cryotherapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Women with a history of prior treatment for precancer</td>
<td>• Women with a positive test and an entirely visible lesion on the ectocervix, not extending to the vaginal wall or into the endocervix</td>
</tr>
<tr>
<td>• Women with suspected cancer</td>
<td>• The lesion can be adequately covered with a 2.5 cm cryotherapy probe</td>
</tr>
<tr>
<td>• Women with known pregnancy and until 6 weeks postpartum</td>
<td>• Women with no evidence of pelvic inflammatory diseases or cervicitis and with no polyps</td>
</tr>
<tr>
<td>• Women with a lesion occupying more than 75% of the surface area of the cervix</td>
<td>• Women who are not pregnant</td>
</tr>
<tr>
<td>• The cryotherapy probe does not cover the lesion or leaves space of more than 2mm</td>
<td>• Women who have given consent for treatment</td>
</tr>
<tr>
<td>• The lesion extends more than 2mm into cervical canal or onto the vaginal wall</td>
<td></td>
</tr>
</tbody>
</table>
11. Set timer for 3 minutes. Press freeze button. Apply pressure to the cervix as the gas begins to flow to the cryoprobe. Watch as the ice ball develops. Use “freeze-clear-freeze technique,” (double-freeze) and freeze cervix for 3 minutes. Adequate freezing has been achieved when the margin of the ice ball extends 4-5 mm past the outer edge of the cryotip. After 3 minutes, allow time for adequate thawing before removing the probe from the cervix.

12. After thawing, repeat the procedure. Inspect the cervix carefully to ensure that a hard, white completely frozen ice ball is present.


14. Inspect cervix for bleeding. If there is bleeding, apply pressure to area using clean cotton swab.

15. Remove the speculum and place it in 0.5% chlorine solution for 10 minutes for decontamination.

**Post-cryotherapy Tasks**

1. Light source, cryogun, regulator and gas cylinder should be wiped with 0.5% chlorine solution or 60-90% ethyl, isopropyl alcohol.

2. Wash hands thoroughly with soap and water and dry with clean, dry cloth or air dry.

3. Women may be provided with a supply of sanitary pads to prevent the secretions staining their clothes.

4. Advise the woman about post-treatment care, warning signs and follow up instructions, as follows:
   - Women may experience some mild cramps and a clear or lightly blood-stained watery discharge for up to 4-6 weeks after treatment.
   - They should be advised not to use a vaginal douche or tampons or to have sexual intercourse for one month after treatment. If it is not possible to abstain, condoms may be used from 2 weeks post treatment.

Appointments should be made for a follow-up visit one year after treatment. Cervical lesions persist or recur in about 5-10% of HIV-negative cryotherapy clients. Repeat VIA one year after cryotherapy to assess the persistence or recurrence of lesions. Retreatment is carried out if lesions persist but VIA negative women will require annual screening for 5 years after which they may be referred back to the screening program.

**B) LOOP ELECTROSURGICAL EXCISION PROCEDURE (LEEP)**

LEEP, also referred to as Large Loop Excision of the Transformation Zone (LLETZ), is an excisional method of treatment of precancerous lesions. It is the treatment of choice for cervical lesions that do not meet the eligibility criteria for cryotherapy, or when a histological specimen is needed.
It involves removal of abnormal areas of the cervix by applying a low voltage high frequency alternating current to a thin wire loop electrode and slowly passing it through the cervix. The loop cuts and coagulates at the same time. LEEP is successful in eradicating pre-cancer in over 90% of cases. However, unlike cryotherapy, LEEP requires more highly skilled personnel, electricity and local anesthesia including colposcopy. Clients requiring LEEP should be referred to appropriately trained personnel.

C) COLD KNIFE CONISATION
Cold knife conisation is the removal of a cone-shaped area from the cervix including the ectocervix and endocervix. It is usually done under general or regional anesthesia by gynaecologists and gynaeoncologists trained in the procedure and able to recognize and manage its complications, in an equipped surgical facility. Because of the possible side effects, cold knife conisation should be reserved for cases that cannot be managed with cryotherapy or LEEP excision. The extent of conisation depends on the size of the lesion; the woman's desire to have more children, and the likelihood of finding invasive cancer. The tissue removed is then subjected to histopathology.

D) THERMOCOAGULATION
The use of thermocoagulation for the treatment of CIN is as effective as other methods, such as cryotherapy and LEEP, with the advantage of being rapid and is also associated to a low occurrence of side effects [Dolman et al, 2017]. It is a low-cost and simple treatment method comparable to cryotherapy. While cryotherapy is a highly effective intervention with a good cure rate, the low availability of refrigerant gas makes its use challenging in LMIC [Elit et al, 2011]. In this regard, thermocoagulation represents an attractive alternative for the treatment of cervical precancerous lesions especially where electricity is available.

This method involves destruction of precancerous lesions in the transformation zone with temperatures between 100 - 120°C. It is a 20 second treatment procedure, followed by 20 seconds of waiting, followed by another 20 second treatment procedure. It requires electricity and overall takes about 1 minute to perform. Thermocoagulation is currently under review by World Health Organization as an alternative to cryotherapy where electricity is available.
POST-TREATMENT SCREENING

Screening for cervical cancer after treatment for pre-cancerous lesions focuses on a distinct sub-set of women and thus requires unique considerations as compared with primary screening.

A study on post treatment testing in HIV+ women done in Western Kenya found that HPV test had the highest sensitivity and specificity. The second-best approach was found to be a co-test of VIA and pap smear (Orang’o, 2017). Where HPV test and pap smear are not available, this guideline recommends performing VIA and referring women for pap smear.
REFERENCES


Colorectal cancer is a leading cause of cancer morbidity and mortality worldwide, with 1,849,518 new cases and 880,792 deaths in 2018. In Kenya it is among the top cancers affecting both men and women, with the number of new cases estimated at 2,316 and deaths at 1,466 (GLOBOCAN, 2018). It is preventable since most colorectal cancers develop from precancerous polyps which when detected early can be removed. It can also be cured if diagnosed early.

Patients normally present with a change in bowel habits (diarrhea or constipation), rectal bleeding, persistent abdominal discomfort (cramps, bloating/flatulence, abdominal pain), tenesmus (feeling of incomplete bowel emptying), weakness or fatigue or unexplained weight loss, iron-deficiency anemia and intestinal obstruction. However, many people with colon cancer experience no symptoms in the early stages of the disease. When symptoms appear, they will likely vary, depending on the size of the cancer and location in the large intestine. The symptoms are relatively non-specific and may mimic other conditions such as gastro-intestinal infections and other inflammatory conditions. Therefore, a high index of suspicion is required.

Majority of colorectal cancers arise from polyps. The two classes of precancerous lesions that predispose to colorectal cancer are conventional adenomas and serrated polyps.

**KEY RECOMMENDATIONS**

- Age to begin screening is 45 years for average-risk persons.
- OBT is the recommended screening test in average-risk persons
- Colonoscopy is recommended for high-risk persons
- More frequent screening in high-risk persons - on average, every 5 years.
- Genetic testing for familial CRC

**Introduction**

Colorectal cancer is a leading cause of cancer morbidity and mortality worldwide, with 1,849,518 new cases and 880,792 deaths in 2018. In Kenya it is among the top cancers affecting both men and women, with the number of new cases estimated at 2,316 and deaths at 1,466 (GLOBOCAN, 2018). It is preventable since most colorectal cancers develop from precancerous polyps which when detected early can be removed. It can also be cured if diagnosed early.
RISK FACTORS

Non-modifiable risk factors
- Older age - greater than 45 years old
- Inflammatory bowel disease such as Crohn’s disease or ulcerative colitis.
- A family history of colorectal cancer or colorectal polyps.
- Presence of genetic syndromes like familial adenomatous polyposis (FAP) or hereditary non-polyposis colorectal cancer (Lynch syndrome)

Modifiable risk factors
- Intake of red and processed meats
- Physical inactivity
- Low fruit and vegetable intake
- A low-fiber and high-fat diet
- Obesity
- Alcohol intake
- Tobacco use

RATIONALE FOR SCREENING

Colorectal cancer is associated with high morbidity and mortality rates in Kenya. Majority of colorectal cancer cases are locally invasive or distantly metastatic at diagnosis. Screening provides an opportunity for detection and removal of pre-cancerous lesions which prevent or delay the occurrence of colorectal cancer. In addition, detection of early stage disease allows early therapeutic intervention/treatment with good clinical outcomes.

The aim of colorectal cancer screening is the detection of precancerous lesions (adenomas and serrated polyps) and early cancer lesions.

RECOMMENDED APPROACH TO SCREENING

There are various approaches that may be used for screening colorectal cancer. This guideline recommends using the risk-stratified approach. This where the patient’s risk for advanced adenoma is assessed and the patient classified as per risk status, either as high risk or low risk. We also recommend starting with FOBT and then guided screening for low risk clients. For high risk clients (refer to Table 1 for risk stratification), colonoscopy is the recommended modality for screening.
Other approaches to screening

In settings of opportunistic screening, various approaches may be used to offer screening to patients as described below:

i. **Multiple screening options**
This is whereby the client is informed about all the available screening modalities (FOBT and colonoscopy) and they choose their preferred method. It is usually helpful in well-resourced settings.

ii. **Sequential approach**
This involves a step-wise approach where the most effective screening modality is first recommended to the patient then a less preferred option is offered subsequently if the patient declines the initial method. This guideline recommends that for opportunistic screening, colonoscopy should be offered first and FOBT next. However, for the programmatic screening, FOBT should be offered first then colonoscopy.

Guideline recommendations

- Risk stratified approach is most recommended
- In low risk patients, begin with FOBT then guided screening.
- For high risk patients, risk stratification should be done and colonoscopy
SCREENING INTERVENTIONS

CRC screening tests are based on performance features, costs, and practical considerations. Colonoscopy and FOBT are recommended as the cornerstones of screening regardless of the screening approach taken.

Colonoscopy
Colonoscopy involves using an endoscope inserted via the anus to visualize the entire colon and rectum directly.
The advantages of colonoscopy include:
- High sensitivity for cancer and all classes of precancerous lesions
- Diagnosis and treatment can be done in a single session
- It allows for long intervals between examinations (10 years) in subjects with normal findings. One or 2 negative examinations may signal lifetime protection against CRC.
It is ideal for patients who value the highest level of sensitivity in detection of precancerous lesions and are willing to undergo invasive screening.

Disadvantages of colonoscopy include the need for thorough bowel cleansing and procedure-related complications like perforation and sedation risks. However, these are rare when the procedure is done by skilled personnel.

Fecal occult blood testing (FOBT)
Fecal occult blood testing can be either guaic fecal occult blood (gFOBT) testing or Fecal immunochemical Test (FIT). In Kenya, gFOBT is the most widely available test.
Advantages of FOBT include its noninvasive nature and low cost. FIT is more sensitive than gFOBT.
FOBT is recommended annually and is commonly the test of choice in programmatic screening. It is also one of the tests that can be used in sequential or multiple-options approach.
Disadvantages of FOBT include the need for repeated testing and poor sensitivity for some precursor lesions.

Flexible sigmoidoscopy
This can be an option to colonoscopy in settings lacking adequate infrastructure to support a full colonoscopy.
Advantages of flexible sigmoidoscopy include:
- Lower cost and risk compared with colonoscopy
- Limited bowel preparation required
- No need for sedation
Disadvantages of flexible sigmoidoscopy include:
- Lower benefit in protection against right-sided colon cancer compared to colonoscopy.
- Relative patient discomfort due to lack of sedation

Flexible sigmoidoscopy, when used, is often recommended at 5-year intervals. Patients with a positive FOBT and negative flexible sigmoidoscopy should have a full colonoscopy done since it is possible that they may be having a right-sided adenoma or cancer.

**Double Contrast Barium Enema (DCBE)**
The DCBE is a relatively low risk procedure, less invasive and more affordable than colonoscopy. It can be easily done in any facility with an X-ray machine. However, a negative result does not rule out abnormalities or colorectal cancer. Therefore, the patient still requires a follow-up colonoscopy. The disadvantages of the procedure include allergic reactions and procedure-related risks.

**Virtual (CT) colonoscopy**
This is a reconstruction of the colon from CT scan images. It may be used as an alternative to DCBE and in settings where contraindications to colonoscopy may exist.

**Advantages:**
- Minimally invasive screening procedure therefore lower complication rate
- Takes less time
- Less vigorous bowel preparation due to tagging of residual stool and fluid
- Can visualize colon beyond an obstruction or narrowing
- Detects both colonic and extra-colonic pathology

**Disadvantages:**
- Small risk of perforation
- High cost as compared to DCBE and FOBT
- Any positive findings will require conventional colonoscopy for confirmation
- Exposure to low-level ionizing radiation
- Any residual/untagged faecal matter can give rise to wrong interpretation
- There is currently no evidence to test and demonstrate survival benefit
WHO SHOULD BE SCREENED & WHEN?

Screening is conducted in asymptomatic patients without prior colonoscopy unlike surveillance where colonoscopy is performed in patients with previous precancerous or dysplastic lesions, inflammatory bowel disease or after another positive screening test.

Persons at average risk (asymptomatic healthy individuals without family history) should begin screening for colorectal cancer at age 45 years in Kenya (Rex et al, 2009). The recommended screening modality is annual FOBT followed by colonoscopy. If the initial colonoscopy findings are normal, repeat the colonoscopy after 10 years. If the screening is done using FOBT, screen the patient annually. Other screening options include a flexible sigmoidoscopy, Virtual colonoscopy or a double-contrast barium enema every 5 years. If any of the alternative tests are positive, the patient should have a colonoscopy done.

Persons with a family history of CRC or a documented advanced adenoma in a first-degree relative age <50 years or 2 first-degree relatives with these findings at any age are recommended to undergo screening by colonoscopy every 5 years, beginning at age 40 years or 10 years before the age at diagnosis of the youngest affected relative, whichever comes earlier.

Persons with a single first-degree relative diagnosed at ≥50 years with CRC or an advanced adenoma can have 10-yearly colonoscopy from 40 years of age, or 10 years earlier than the youngest relative diagnosed with CRC, whichever comes earlier.
### Summary of Risk Stratification

<table>
<thead>
<tr>
<th>CATEGORY</th>
<th>CHARACTERISTICS</th>
<th>RECOMMENDATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Average risk</td>
<td>• Age ≥45 years&lt;br&gt;• Asymptomatic&lt;br&gt;• No family history of CRC</td>
<td>• Screening should be initiated from 45 years of age&lt;br&gt;• The patient should be offered FOBT using FIT and then colonoscopy.&lt;br&gt;• Colonoscopy should be offered every 10 years if the initial one is normal&lt;br&gt;• FOBT using FIT can be offered annually if the initial one is negative Positive tests should be followed by colonoscopy.&lt;br&gt;• Patients can be offered 5-yearly sigmoidoscopy if the colonoscopy is not available</td>
</tr>
<tr>
<td>2. Increased risk</td>
<td>• CRC in one first-degree relative &lt;50 years or&lt;br&gt;• an advanced adenoma in one first-degree relative diagnosed &lt;50 years&lt;br&gt;• Persons with multiple first-degree relatives or other relatives with CRC diagnosed at &gt;50 years</td>
<td>• 10-yearly colonoscopy from 40 years of age, or 10 years earlier than the youngest relative diagnosed with CRC, whichever comes earlier&lt;br&gt;• 5-yearly colonoscopy from 50 years of age, or 10 years earlier than the youngest relative diagnosed with CRC whichever comes earlier</td>
</tr>
<tr>
<td>3. High risk</td>
<td>• Hereditary or genetic predisposition, e.g. FAP, polyposis syndrome,&lt;br&gt;• Non-hereditary polyposis&lt;br&gt;• Inflammatory bowel disease</td>
<td>• Genetic counselling and testing&lt;br&gt;• Colonoscopy should be done from age of 10 years with FAP, 18 years for HNPCC, 10 years earlier than the affected relative</td>
</tr>
</tbody>
</table>
AGE TO STOP SCREENING
Discontinuation of screening should be considered when persons up to date with screening, who have prior negative screening (particularly colonoscopy), reach age 75 or have less than 10 years life expectancy. Persons without prior screening before the age of 75 years should be considered for screening up to age 85, depending on comorbidities.

WHO DOES THE SCREENING?
FOBT can be performed in an appropriately equipped laboratory. A level III facility and above should be able to do this test. If positive, the patient should have a colonoscopy. Colonoscopy: A trained endoscopist should perform the colonoscopy. Genetic testing: This should also be done in an appropriately equipped laboratory.

SCREENING AT VARIOUS HEALTHCARE LEVELS
Colonoscopy and sigmoidoscopy should be offered from a Level 5 facility or higher. It should be well equipped with facilities for management of procedure complications such as complications of sedation or gut perforation. The recommendation for colonoscopy at Level 5 or higher is informed by staffing and safety considerations as well as the need to create high volume centers.

FIT and DCBE should be done in facilities with the requisite expertise (laboratory and radiology respectively). This guideline recommends for Level 3 facilities and higher to be able to conduct FOBT.
ALGORITHM: SCREENING FOR COLORECTAL CANCER

Symptoms of colorectal cancer

- Yes → Diagnostic studies
- No → Risk

Risk

Average

- Age

<45yr

- Do not screen

≥45yr

- Adenomatous polyps
- Colorectal cancer
- Inflammatory bowel

Personal history

Increased

History

Family history

Options

- Annual FOBT
- Colonoscopy every 10yr
- Flexible sigmoidoscopy every 5 yr
- FOBT and Flexible sigmoidoscopy
- DCBE every 5-10yr

Positive

Evaluation of entire colon

- Colonoscopy
- Alternative for positive FOBT: DCBE, preferably with flexible sigmoidoscopy

Colonoscopy

- Alternative: DCBE, preferably with flexible sigmoidoscopy

Consider surveillance colonoscopy

Genetic syndromes (FAP, HNPCC)

- Colorectal cancer in 1 or 2 1st-degree relatives
- Adenoma polyposis in 1st-degree relative <60y

Screening, genetic counselling, genetic testing

Same screening options as for average risk but starting at 40 yr

Negative

Adapted from Center for Colon and Rectal Surgery 2009 (original source: Winswer SJ, Fletcher RH, Miller L, et.al, Colorectal cancer screening; clinical guidelines and rationale, Gastroenterology)
REFERENCES:


Colorectal (Colon) Cancer; Division of Cancer Prevention and Control, Centers for Disease Control and Prevention, Available from: https://www.cdc.gov/cancer/colorectal/basic_info/risk_factors.html Accessed 25/09/2018

Dr Michael P. Hartung, Prof Frank Gaillard, et.al. Colorectal Carcinoma. Radiopia [Internet]. 2018; Available from: https://radiopaedia.org/articles/colorectal-carcinoma


OESOPHAGEAL CANCER EARLY DETECTION

Introduction
Oesophageal cancer is the 7th most common cancer and 6th most frequent cause of cancer mortality in the world, with estimates of 572,034 new cases and 508,585 deaths in 2018 (GLOBOCAN, 2018). In Kenya it is one of the top cancers in both men and women. With an incidence of 4,380 cases, it is the second most common in males and third in females. Worryingly, the mortality rate and the incidence rates are almost equal at 18.4% (GLOBOCAN, 2018). In addition, the cancer has been reported to be on the rise and in particular hot spots in the country. The main variant of oesophageal cancer seen in Kenya is oesophageal squamous cell carcinoma unlike in the Europe and A

Currently, international screening guidelines for oesophageal cancer do not exist. However, due to the high disease burden in Kenya, there is a need provide guidance on how to improve early detection of the cancer. This can lead to better treatment outcomes, better prognosis and will be a more cost-effective approach as compared to the current status whereby late diagnosis is most common.

RISK FACTORS

Non-modifiable risk factors
• Increasing age
• Family history of oesophageal cancer

Modifiable risk factors
• Overweight and obesity
• Alcohol Consumption
• Tobacco use
• Carbon exposure from firewood and other sources
RATIONALE FOR EARLY DETECTION
The most recent data from GLOBOCAN shows an increase in mortality from oesophageal cancer in Kenya (GLOBOCAN, 2018). Many patients present late, at a point when curative treatment cannot be offered. Early detection provides a chance to catch precancerous lesions or dysplasia and early disease which can lead to less morbidity, better chances of successful treatment and lower treatment costs. Current international recommendations do not support population wide screening. However, in high-risk populations in Asia and in Africa, it seems feasible to do targeted screening in these populations as has been done in China (Codipilly, 2018; Chen 2016, Roshandel, 2014). A study done in Kenya showed the feasibility of this and also provided initial estimates of dysplasia rates (Mwachiro, 2016).

GOALS OF SCREENING
Early detection makes it possible to detect precancerous lesions and early cancer lesions.

APPROACHES TO SCREENING
1. Identifying individuals in areas of high-risk/ incidence for oesophageal cancer
2. Recommending screening tests that can be done for detection
3. Providing facilities that can carry out the screening tests

SCREENING TESTS
1. **White Light Endoscopy:**
   a. Advantages: Can be done in any endoscopy unit.
   b. Disadvantage: Misses out on precursor lesions. Has a cost attached to it and needs sedation as well as trained endoscopists.

2. **Lugol’s chromoendoscopy**
   a. Advantages: Shows precursor lesions. Lugol’s iodine is cheap.
   b. Disadvantages: Endoscopists need additional training. Allergic reaction to iodine can occur. Needs sedation and endoscopy.

3. **Narrow band Imaging endoscopy**
   a. Advantages: Shows precursor lesions
   b. Disadvantages: Needs special endoscopy equipment. Endoscopists need additional training and also sedation risks.

Possible future screening test:
1. Breath test- still in research phase
2. Cytosponge screening test
3. Blood markers- to check for autoantibodies and methylated DNA markers

SCREENING RECOMMENDATIONS:
1. Individuals with first degree relatives with biopsy proven oesophageal cancer should have screening endoscopy 10 years prior to index age of diagnosis of the first degree relative or at age 40 whichever is earlier
2. Asymptomatic individuals living in high risk areas should get a one-time screening endoscopy at age 40 years.
3. Patients treated for head and neck cancer squamous cell carcinoma should be screened annually for ten years.
4. Patients who have had caustic acid ingestion should be screened 10 years from the injury via endoscopy.
WHO SHOULD BE SCREENED & WHEN?
Screening is conducted in asymptomatic patients without prior colonoscopy unlike surveillance where colonoscopy is performed in patients with previous precancerous or dysplastic lesions, inflammatory bowel disease or after another positive screening test.

Persons at average risk (asymptomatic healthy individuals without family history) should begin screening for colorectal cancer at age 45 years in Kenya (Rex et al, 2009). The recommended screening modality is annual FOBT followed by colonoscopy. If the initial colonoscopy findings are normal, repeat the colonoscopy after 10 years. If the screening is done using FOBT, screen the patient annually. Other screening options include a flexible sigmoidoscopy, Virtual colonoscopy or a double-contrast barium enema every 5 years. If any of the alternative tests are positive, the patient should have a colonoscopy done.

Persons with a family history of CRC or a documented advanced adenoma in a first-degree relative age <50 years or 2 first-degree relatives with these findings at any age are recommended to undergo screening by colonoscopy every 5 years, beginning at age 40 years or 10 years before the age at diagnosis of the youngest affected relative, whichever comes earlier.

Persons with a single first-degree relative diagnosed at ≥50 years with CRC or an advanced adenoma can have 10-yearly colonoscopy from 40 years of age, or 10 years earlier than the youngest relative diagnosed with CRC, whichever comes earlier.

REFERENCES:
GLOBOCAN 2018 data


ORAL CANCER SCREENING

KEY POINTS

- Incidence of oral cancer is increasing globally particularly in Sub-Saharan Africa
- Early diagnosis of oral cancer has a statistically significant impact on prognosis
- Risk factors of oral cancer include habits such as consumption of tobacco, alcohol and betel nut
- Opportunistic screening targeting all individuals at risk of developing the disease is recommended
- A combination of visual and analytical screening gives a high positive predictive value for oral cancer justifying the screening of tobacco users
- Training of personnel at different levels of the health system in visual and analytical screening of oral cancer is recommended
- Incorporation of monitoring and evaluation in the referral chain ensures responsiveness of the health system to oral cancer

Introduction

Oral cancer is by definition any malignancy arising from oral tissues; pharynx and salivary glands. The global incidence of oral cancer is 354,864 cases where mortality attributed to this condition is 177,384 deaths per annum (GLOBOCAN, 2018). Over 90% of oral cancers are squamous cell carcinomas (OSCC), with the remaining fraction consisting of varying percentages of lymphomas, sarcomas and adenocarcinomas (Ibukunle et al, 2018; Idris et al 2016; Monteiro et al, 2016). It predominantly occurs in male patients in the fifth and sixth decades of life. Modifiable risk factors include habits such as consumption of tobacco, alcohol and betel nut. Incidence varies regionally with the prevalence of these high-risk habits, with statistics as high as 40% in parts of Asia (Chuang et al, 2017).

Modifiable risk factors
- Tobacco use
- Alcohol use
- Betel nut chewing
- Excessive sunlight exposure
- HPV infection
- Vitamin A, C and E deficiencies
- Immunosuppression

Non-modifiable risk factors
- Genetics
Oncogenic strains of the human papilloma virus (HPV) are aetiologically associated with oral cancer (Argiris, 2008). As an HPV-associated malignancy, the incidence of oral cancer increases exponentially in patients infected with the human immunodeficiency virus (HIV) together with cancers of the urogenital systems (Khot et al, 2016). OSCC is ranked as the third most common head and neck malignancy for patients with HIV-infection worldwide, after Kaposi’s sarcoma and non-Hodgkin’s lymphoma (Heigenz, 2005). Mucosal diseases such as leukoplakia, erythroplakia, lichen planus and oral submucous fibrosis have the potential to progress into invasive OSCC.

The incidence of oral cancer varies regionally with only about 4% of all cancers in developed countries but as high as 40% in parts of India and Sri Lanka (Ferlay et al, 2015). A comparison of sub-Saharan countries demonstrates relatively high incidences and mortality due to oral cancer in Kenya. According to the GLOBOCAN data (Bray et al, 2018), Kenyan patients with oral cancer record high mortality rates as compared to survival rates globally (Figure 1). Data from the Nairobi Cancer Registry (Korir et al, 2008) showed that 4.9% of the disease burden was attributable to oral cancer (ASR 8.4 per 100,000). In males, oral cancer accounted for 10.24% of malignancies while in females it contributed to 4.3%. Butt and co-authors (2008), reported OSCC as the second most common malignancy affecting HIV-infected patients in Kenya after Kaposi’s sarcoma. Majority of the patients present in Tumor- Node- Metastasis (TNM) stages 3 and 4, resulting in poor prognosis after treatment (Butt, 2008).

![Estimated age-standardized incidence and Mortality rates (world) in 2008, lip, oral cavity, both sexes, all ages](image)

*Figure 1: Incidence and mortality estimates for lip and oral cavity cancer in sub-Saharan Africa (IARC / Globocan 2018)*
RATIONALE FOR SCREENING
Screening can be defined as the process of examining or applying a rapid test to a population to identify a group at risk from a disease. Evidence of the utility of screening may be derived from Asian countries where the relatively high incidences of oral cancer demanded innovative interventions. In India, a seminal randomized trial of visual screening for oral cancer was conducted for a cohort of 95,000 patients (Sankaranarayanan et al, 2005). A similar number of people in the control group did not undergo screening and were monitored for up to 12 years for mortality by community health workers. Despite the fact that only 63% of people found with lesions had the recommended further assessment, death from oral cancer in users of tobacco or alcohol was reduced by 34% which was statistically significant.

Taiwan scaled up to implement a nationwide program for tobacco and betel nut users incorporating over 2 million participants since 2004 (Chuang et al, 2017). This program resulted in a reduction in the number of patients presenting with TNM stages III and IV oral cancers and 24% reduction (95% CI, 3%-40%) in oral cancer mortality. Increased diagnostic acumen resulted in treatment seeking in TNM stages I and II, with better prognoses. The WHO guidelines recommend screening for early detection of oral cancers.

WHO SHOULD BE SCREENED AND WHEN?
The most effective approach to ensure early diagnosis of oral cancers in a resource-limited setting is to offer opportunistic mass screening, targeting all individuals at risk of developing the disease (NSC, UK, 2018). The criteria for annual screening should simply be a history of consumption of tobacco and/or exposure to any other risk factor for oral cancer.

SCREENING AT VARIOUS HEALTHCARE LEVELS
Oral cancer screening is fairly straightforward because apart from the tumors extending into the pharynx, most lesions are easily accessible and those at risk can be easily identified (Chuang et al, 2017). Visual screening can be integrated into primary health care for a few dollars, while analytical screening remains the preserve of either a cytologist, an anatomical pathologist or an oral pathologist. There is a need to develop a multi sectoral approach that integrates tobacco and alcohol control as part of health education.

Oral cancer screening is fairly straightforward because apart from the tumors extending into the pharynx, most lesions are easily accessible and those at risk can be easily identified (Chuang et al, 2017). Visual screening can be integrated into primary health care for a few dollars, while analytical screening remains the preserve of either a cytologist, an anatomical pathologist or an oral pathologist. There is a need to develop a multi sectoral approach that integrates tobacco and alcohol control as part of health education.
Table 1: Health facilities and staff cadres for oral cancer screening

<table>
<thead>
<tr>
<th>LEVEL</th>
<th>TYPE</th>
<th>CADRE</th>
<th>ACTIVITIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Community</td>
<td>Community Health care worker</td>
<td>Oral Health awareness</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mobilization</td>
</tr>
<tr>
<td>2</td>
<td>Dispensary</td>
<td>Nurses</td>
<td>Oral Health awareness</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mobilization</td>
</tr>
<tr>
<td>3</td>
<td>Health Centres</td>
<td>Nurses</td>
<td>Oral Health awareness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Clinical Officers</td>
<td>Visual screening</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Community Oral Health Officer</td>
<td>Sample collection</td>
</tr>
<tr>
<td>4</td>
<td>Sub-county</td>
<td>Nurses</td>
<td>Oral Health awareness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Clinical officers</td>
<td>Mobilization</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Medical officers</td>
<td>Sample collection and analysis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Community Oral Health Officer</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pathologist</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dentists</td>
<td></td>
</tr>
</tbody>
</table>

SCREENING TESTS TO BE DONE
The clinician should conduct visual and analytical screening in the following sequence (Huber et al, 2014):

1. VISUAL EXAMINATION

   a) Extraoral examination - inspect the head and neck region for asymmetry or swelling. Palpate the submandibular, neck and supraclavicular lymph nodes paying attention to size, number, tenderness and mobility. Inspect and palpate the lips and perioral tissues for abnormalities.

   b) Intraoral examination - systematically inspect and palpate all oral soft and bony tissues paying attention to the high-risk sites for the development of oral cancer including the lateral and ventral aspects of the tongue; floor of the mouth; labial mucosa; cheek; soft palate; pharynx, tonsils, maxilla and mandible.

   c) Lesion inspection – Lesions which persist for more than 14 days should be referred for further investigation. Evaluate the specific characteristics of each lesion with particular attention to the size, colour, texture and contour. Any
white, red, ulcerated and/or indurated lesions are documented.

d) Documentation - at the time of initial assessment and each re-evaluation appointment, it is recommended that a clear record should be maintained.

2. OPTIONAL SCREENING ADJUNCTS

Adjunctive visual tools can enhance contrast between the clinical lesion and the adjacent normal oral tissue. Techniques include toluidine blue staining and direct fluorescence visualization. Mucosal changes stain positively with the application of toluidine blue dye or show loss of fluorescence in premalignant and malignant lesions. These methods, however, do not give a definitive diagnosis (Zhang et al, 2005). Toluidine blue dye is itself mutagenic and, therefore, its use is restricted.

Direct fluorescence involves intraoral application of a hand-held device that emits a cone of blue light which excites various molecules within mucosal cells, causing them to absorb the light energy and re-emit it as visible fluorescence (Lane et al, 2006). Healthy oral tissue emits a pale green fluorescence while altered tissues which attenuate the passage of light appear dark brown or black (loss of fluorescence).

3. IMAGING

Imaging is used as an adjunct for diagnosis for patients with clinical lesions that have a high index of suspicion. Plain X-rays, CT Scans and MRI are used to investigate the extent of the tumor, nodal involvement and metastasis.

4. DIAGNOSTIC BIOPSY

Brush biopsy uses a round stiff bristle brush to collect cells from the surface layers of a lesion by vigorous abrasion. The cells collected are transferred to a microscope slide to identify abnormal cells. Cytological studies tend to be technique sensitive and require trained personnel for accurate interpretation.

Table 2: Diagnostic sequence for early detection of oral cancer

<table>
<thead>
<tr>
<th>TYPE OF TEST</th>
<th>PERSONNEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual screening</td>
<td>Nurse, Clinical officer, Community oral health officer</td>
</tr>
<tr>
<td>Imaging</td>
<td>Radiographer, Radiologist,</td>
</tr>
<tr>
<td>Exfoliative cytology</td>
<td>Sample collection: Nurse, clinical/ medical/ dental officers</td>
</tr>
<tr>
<td></td>
<td>Analysis: Cytologist, pathologist</td>
</tr>
<tr>
<td>Incision biopsy</td>
<td>Sample collection: Dental officer, Maxillofacial surgeon</td>
</tr>
<tr>
<td></td>
<td>Analysis: Pathologist</td>
</tr>
</tbody>
</table>
Figure 3: Screening algorithm for OSCC
REFERENCES:


Lane PM, Ilhuly T, Whitehead PD, Zeng H, Poh CF, Ng S. Simple device for the direct visualization oral cavity tissue fluorescence. Journal of Biomedical Optics 2006; 11 (2)


WHO (2007) Cancer control; Knowledge into action WHO Guide for effective programs – Module 3pg 8

PROSTATE CANCER SCREENING

Introduction
Prostate Cancer burden
Prostate cancer is the fourth leading cancer in incidence globally, with higher mortality reported in less developed regions than in more developed regions. Prostate cancer is a disease of the aging male, the majority presenting after 65 years (average of 68 years). This is the most common cancer among elderly male population all over the world, with a slight preponderance in blacks. Incidentally autopsy prevalence rates of prostate cancer are the same across all races suggesting environmental factors play a crucial role in aetiology. This compares well with.

In Kenya, prostate cancer is the commonest cancer in males with 2,864 new cases (14.9%) (GLOBOCAN, 2018). Currently, the proportion of men diagnosed with prostate cancer by age is as tabulated below:

<table>
<thead>
<tr>
<th>Age of Men in Years</th>
<th>Percentage proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 55</td>
<td>10.1%</td>
</tr>
<tr>
<td>55-64</td>
<td>30.7%</td>
</tr>
<tr>
<td>65-74</td>
<td>35.3%</td>
</tr>
<tr>
<td>75-84</td>
<td>19.9%</td>
</tr>
<tr>
<td>&gt;84</td>
<td>4.4%</td>
</tr>
</tbody>
</table>

*Source: KNH Cancer Registry, 2018: Prostate cancer new cases % by age*

Studies have shown that compared with other countries, patients with prostate cancer in Kenya present at a similar mean age at diagnosis, but with more advanced disease and more aggressive tumours compared(Dickstein et al, 2009; Jonnson et al; Hemed et al, 2014).
**Rationale for Screening**

- There is no role for MASS screening for prostate cancer
- Screening for prostate cancer is a highly individualized decision between a client and his caregiver.

**Effects of Prostate Specific Antigen (PSA) screening**

PSA screening has induced a significant downward migration in age and stage (both clinical and pathologic) at diagnosis. It may also have a beneficial effect on prostate cancer mortality. However, the absolute effect is small relative to the number needed to screen and treat to cure a single individual. For every 1,000 men tested, approximately 100 to 120 will have an elevated PSA value (Eastham et al, 2003).

**Therefore, mass screening is not recommended.**

These guidelines are meant to equip healthcare providers with information on early detection of prostate cancer for the purpose of reducing prostate cancer mortality. They should have a high index of suspicion and should be keen to screen patients presenting with the following lower urinary tract symptoms for prostate cancer:

- Frequency and nocturia
- Difficulty in starting or stopping the urine flow
- Inability to urinate
- Weak, decreased or interrupted urine stream
- A sense of incompletely emptying the bladder.
- Burning or pain during urination (dysuria)
- Post-micturition dribbling
- Urgency in urinating
- Blood in the urine or semen
PSA-Based Screening Guidelines
Various international organizations (listed in the references section) have issued guidelines on prostate cancer screening. These guidelines differ in their recommendations regarding:

- Whether or not to provide routine PSA-based prostate cancer screening
- In what age groups and life expectancies

However, the guidelines agree that:

- PSA-based prostate cancer screening requires an informed, shared decision-making process
- The decision should reflect the patient’s understanding of the possible benefits and risks
- The decision should respect the patient’s preferences and values.

SHARED DECISION MAKING

A well-informed patient understands the ratio of benefit to harm of prostate cancer screening

PSA-based screening should not be performed in the absence of shared-decision making between clinicians and clients. This effectively discourages organized mass screening in settings where shared-decision making is not part of routine practice (e.g. in health camps, health system promotions, community organizations and religious or political meetings).

What a man needs to know before making a decision about PSA screening:

- There is no perfect screening test
- Screening may have associated harms
- Prostate biopsy & treatment of prostate cancer have associated risks
Men considering a screening test for prostate cancer should be aware of several facts:

1. **Prostate gland anatomy and function**

2. **The ratio of benefit to harm of screening:**
   Evidence shows that the absolute benefits of prostate cancer screening are modest for men age 55 to 69 years, while the harms are substantial (Eastham et al, 2003).
   
   a) **Putative (evident) mortality benefit of screening**
   The lifetime risk of prostate cancer is 1 in 6 men. Diagnosis is made in 1 in 9 men and out of these, only 3% of these will die of prostate cancer (ACS, 2018). Men should consider the threat posed by prostate cancer and weigh this against other potential life-threatening conditions that they may have. The ratio of benefit to harm can be improved by considering the age and health status of the individual (Welch et al, 2009), and a man’s personal preferences.

   b) **Harms of screening**
   PSA screening can lead to psychological harm and biopsy related complications. The greatest harm associated with prostate cancer screening is the detection of cancers that would otherwise have remained undetected without screening (over-diagnosis), subsequent treatment of these cancers (over treatment) and the associated side effects from a treatment that does not improve survival. In addition, prostate biopsies and treatments targeting localized prostate cancer have adverse effects, including pain, fever, bleeding, infection or problems urinating, with possible need for hospitalization (Carlsson et al, 2005 and Loeb et al, 2012).

3. **The likelihood of false-positive and false-negative results**
   - No screening test is perfect. DRE is not very sensitive and will miss many early prostate cancers. PSA test can generate a significant number of false positive results due to low specificity. A high PSA often leads to a biopsy being performed with many ending up negative, exposing the patient to the side effects of biopsy procedures.
   - PSA cut-off determines the rate of false positives. Cut-off points of <4.0 ng/mL can lead to higher rates of false positives (Volk et al, 2007; Schroder et al, 2009).

4. **Description of options & follow-up tests after abnormal PSA is detected.** These are articulated below under the discussion on biopsy trigger
5. **Treatment options & outcomes for early and late prostate cancer and their possible complications:** Possible treatment outcomes in men diagnosed with prostate cancer include:

- Recurrent cancer that will progress despite their treatment
- No evidence of disease recurrence, but no benefit from treatment either because their cancer was never destined to progress
- No evidence of disease recurrence because their cancer was cured.

Complications of biopsy and treatment in patients screened include, but are not limited to the following: Serious cardiovascular events, deep venous thrombosis or pulmonary embolus, erectile dysfunction, incontinence, and even death (Eastham et al, 2003).

### WHO SHOULD BE SCREENED

**Men to be screened include:**

- ≥40 years, of African descent
- 55-69 years, of Caucasian or Asian origin
- 40-55 years, with a family history of prostate cancer

Decisions regarding prostate cancer screening should be individualized, based on personal preferences and an informed decision regarding the uncertainty of benefit and the associated harms of screening. The likelihood of prostate cancer in an individual with a family history of the disease increases directly with the number of affected first degree relatives, and is higher if the disease occurred in multiple generations and/or was diagnosed at an early age (below age 55 years) as compared to a diagnosis in a single generation at an older age (Mohan et al, 2011).

**These guidelines DO NOT recommend routine screening for the following groups:**

- Men aged ≤40 years - due to the low prevalence of disease in this age group
- Men aged 40 - 54 years at average risk - Caucasians and Asians
- Men age 70 years and above – Though they have a high prevalence of prostate cancer, they also have a greater risk of life-threatening co-morbidities and over-diagnosis compared to younger men.
- Any man with a life expectancy less than 10-15 years
FREQUENCY OF SCREENING BY AGE

<table>
<thead>
<tr>
<th>Symptoms &amp; Age</th>
<th>PSA level</th>
<th>Frequency of testing</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Asymptomatic &amp; 55-69 years</td>
<td>&lt; 1 ng/ml</td>
<td>Every two years*</td>
</tr>
<tr>
<td>• 40-54 years with a family history of prostate cancer</td>
<td>1- 4ng/ml</td>
<td>Every year</td>
</tr>
<tr>
<td>• Age ≥ 40 years with a family history of prostate cancer</td>
<td>2ng/ml</td>
<td>Every 2 years</td>
</tr>
</tbody>
</table>

Source: Mottet et al, 2017 and Conford et al, 2017
*Evidence suggests that annual screening is not likely to produce significant incremental benefits when compared with a two-year screening interval (Kerkhof et al, 2010).

BIOPSY TRIGGER

• PSA > 10ng/mL - generally should lead to a biopsy.
• PSA 4 - 10ng/mL - require further interrogation with adjunctive studies like multi-parametric MRI of the prostate. In addition, DRE, PSA derivatives (PSA density and age specific reference ranges) and PSA kinetics (velocity and doubling time), PSA molecular forms (percent free PSA and proPSA), newer urinary markers (PCA3), and prostate imaging should be considered secondary tests (not primary screening tests) that can help to guide on the justification for a prostate biopsy.

Interpretation of PSA results
• There is no PSA level below which a man can be informed that he does not have prostate cancer. Rather, the risk of prostate cancer, and that of high-grade disease, is continuous as PSA increases (Carter et al, 1997). The urologist should consider factors that lead to an increased PSA including prostate volume, patient’s age, inflammation, ratio of total to free PSA, PSA velocity and PSA doubling time, rather than using an absolute level to determine the need for a prostate biopsy.
• The use of antibiotics to reduce PSA levels in otherwise asymptomatic men is strongly discouraged, and this practice could lead to an increased risk of post-biopsy sepsis. A patient with urinary symptoms should be treated for the infection and the PSA re-confirmed.
• Use of Finasteride or 5 alpha reductase inhibitors can reduce the PSA level by 50%, the PSA result needs to be interpreted using the PSA ratio.
• Ideally the PSA should be done in the same laboratory to avoid inter-laboratory variations.

Age-Specific Reference Range For Serum PSA

<table>
<thead>
<tr>
<th>Age Range in Years</th>
<th>Reference Range</th>
<th>African-Americans</th>
<th>Asians</th>
<th>Whites/Caucasians</th>
</tr>
</thead>
<tbody>
<tr>
<td>40 – 49</td>
<td></td>
<td>0 – 2.0 ng/ml</td>
<td>0 – 2.0 ng/ml</td>
<td>0 – 2.5 ng/ml</td>
</tr>
<tr>
<td>50 – 59</td>
<td></td>
<td>0 – 4.0 ng/ml</td>
<td>0 – 3.0 ng/ml</td>
<td>0 – 3.5 ng/ml</td>
</tr>
<tr>
<td>60 - 69</td>
<td></td>
<td>0 – 4.5 ng/ml</td>
<td>0 – 4.0 ng/ml</td>
<td>0 – 4.5 ng/ml</td>
</tr>
<tr>
<td>70 - 79</td>
<td></td>
<td>0 – 5.5 ng/ml</td>
<td>0 – 5.0 ng/ml</td>
<td>0 – 6.5 ng/ml</td>
</tr>
</tbody>
</table>

No local data hence this is adopted from international data: (Oesterling et al, 1993 and Dalkin et al, 1993)

**WHO SHOULD DO THE SCREENING?**

A general practitioner can prescribe a PSA test in a well-informed patient. Patients with a PSA >4ng/ml regardless of other parameters, should be referred to a urologist for further management. The final diagnosis of prostate cancer must be histological after a biopsy is done.
The standard method of early detection for prostate cancer relies on these 3 tests:
1. Serum PSA test
2. Digital rectal examination (DRE)
3. Transrectal ultrasonography (TRUS) guided biopsy

SCREENING AT VARIOUS HEALTHCARE LEVELS
A PSA test should be available from Level 4 hospitals and above, and at accredited private facilities. Follow up of PSA testing should ideally be in the same laboratory. A high PSA should be re-confirmed in the same laboratory.

Most prostate cancers are located in the peripheral zone of the prostate. DRE may not pick small, central or anteriorly-located cancers. DREs are also subjective. Performing a DRE does not significantly increase PSA levels.

Definitive diagnosis of prostate cancer depends on the histopathologic verification of adenocarcinoma in prostate biopsy cores or operative specimens. Transrectal approach is used for most prostate biopsies but a trans-perineal approach can be used for patients without a rectum (due to a previous resection of the rectum). The minimum number of core samples required to make a proper diagnosis is 10 (Extended prostate biopsy protocol).
Algorithm for Prostate Cancer Screening and Early Detection

Asymptomatic male client

Are you well informed on Prostate Cancer?

Yes

- <40 years
- >70 years
- <15 years of life expectancy

No screening

No

- >40 years, of African descent
- 40-54 years with family history of prostate cancer/
- All 55-69 years

Screen with PSA & DRE

PSA >4ng/ml (regardless of other parameters)

Refer to a Urologist

PSA <4ng/ml

Re-screen after 2 years
REFERENCES


Carter HB, Epstein JI, Chan DW et al: Recommended prostate-specific antigen testing intervals for the detection of curable prostate cancer. JAMA 1997; 277: 1456.


Organizations that have issued guidelines on prostate cancer screening:

- American Cancer Society (ACS)
- American Urological Association (AUA)
- European Association of Urology/European Society for Radiotherapy and Oncology/International Society of Geriatric Oncology (EAU/ESSTRO/SIOG)
- European Society for Medical Oncology (ESMO)
- National Comprehensive Cancer Network (NCCN)
- U.S. Preventive Services Task Force (USPSTF)
CHILDHOOD CANCERS: SCREENING AND EARLY DETECTION

Introduction

BURDEN OF CHILDHOOD CANCERS

There are about 3000 new cases of childhood cancer diagnosed in Kenya every year

According to World Health Organization (WHO), a child is defined to be 19 years or younger. Globally, the annual incidence of cancer in children is estimated at 300,000 cases (GLOBOCAN, 2018). Low- and middle-income countries (LMICs) account for 84% of childhood cancers (Ian Magrath, 2013). The cure rate of childhood cancer in high-income countries is >80%, compared to <10% in LMICs (IARC, 2016).

In Kenya, GLOBOCAN estimated that 3,272 new cancer cases were diagnosed in 2018 (Figure 1)

![FIGURE 1: Estimated number of all childhood cancer cases in Kenya](image-url)
RISK FACTORS FOR CHILDHOOD CANCERS

The causes for most childhood cancers are unknown; however, a few environmental and infectious agents have been implicated.

Genetic factors, environmental factors such as exposure to ionising radiation and chemicals have been implicated in the causation of childhood cancers, but there is insufficient data to make conclusive associations (PAHO, 2015). Infectious agents have also been linked to certain cancers; for example, Epstein Barr Virus (EBV) with Burkitt’s lymphoma, Hodgkin’s lymphoma and nasopharyngeal carcinoma, Human Immunodeficiency Virus (HIV) with Kaposi’s sarcoma and malaria with Burkitt’s lymphoma, Human Papilloma Virus (HPV) with head & neck cancers and Hepatitis B Virus (HBV) with hepatocellular carcinoma (PAHO, 2015).

Diagram 2: Risk factors associated with childhood cancer
RATIONALE FOR SCREENING

Screening for childhood cancers is recommended mainly for hereditary retinoblastoma, certain genetic syndromes and in childhood cancer survivors

The majority of childhood cancers are not amenable to screening, apart from retinoblastoma (RB) and other rarer heritable conditions. Furthermore, unlike some adult cancers, childhood cancers are not associated with lifestyle. The emphasis therefore, in childhood cancers is early detection as there is high potential for cure.

WHO SHOULD BE SCREENED?

Screening is recommended for the following groups:

- Children with a family history of genetic cancers like RB, which may be heritable
- Children with a history of RB – should be screened for RB in the other eye and for other related cancers
- Childhood cancer survivors - should be regularly screened for secondary cancers due to their exposure to chemotherapy and radiation
- Children with some familial genetic syndromes associated with cancers, such as shown below:

<table>
<thead>
<tr>
<th>Genetic Syndrome</th>
<th>Characteristic features</th>
<th>Associated Cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Downs Syndrome</td>
<td>Flat facial features, bulging tongue, eyes that slant upwards</td>
<td>Leukemias</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Testicular germ cell tumors</td>
</tr>
<tr>
<td>Beckwith-Weidemann Syndrome</td>
<td>Large size of newborn, large tongue, enlarged body organs, abdominal wall defects</td>
<td>Wilms tumor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hepatoblastoma</td>
</tr>
<tr>
<td>Li Fraumeni Syndrome</td>
<td>Strong family history of cancer at an early age</td>
<td>Brain tumors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Leukemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rhabdomyosarcoma</td>
</tr>
<tr>
<td>Fanconi anemia</td>
<td>Short stature, microcephaly, dangling thumbs, bone marrow failure</td>
<td>Leukemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Wilms tumor</td>
</tr>
<tr>
<td>Familial adenomatous polyposis</td>
<td>Prolonged change in normal bowel habits, blood or mucus in stools, polyps in the colon</td>
<td>Hepatoblastoma</td>
</tr>
</tbody>
</table>
RETINOBLASTOMA SCREENING AND GENETIC COUNSELLING

Introduction
Retinoblastoma (RB) is a hereditary disease and is the most common cancer of the eye affecting children less than 5 years of age. In Kenya, it accounts for 4.8% of all childhood cancers and is estimated to have an incidence of 1 in 17,000 live births (Nyamori & Kimani, 2011).

Bilateral RB, found in 40% of cases, is caused by an inherited or a new germline mutation in the RB1 gene (OMIM 180200). Positive family history of RB is reported in 10% to 15% of all RB patients.

RATIONALE FOR SCREENING FOR RB
It has been noted that in countries with comprehensive screening and early detection programs, cure rates of over 90% have been achieved.

Children with a family history of retinoblastoma have a higher risk for retinoblastoma and require surveillance. Early diagnosis, when tumours are small, maximizes survival and vision outcomes and reduces the need for chemotherapy, enucleation, and radiotherapy. Because retinoblastoma tumours may develop over time during early childhood, serial evaluations are beneficial in detecting tumours early and thus preserving vision.

A child who has RB should have genetic testing to determine their susceptibility of developing RB in the other eye or developing related cancers. It is recommended that for children who test positive for RB gene, family members such as siblings and offspring should also be tested.

Genetic testing has been shown to improve risk prediction for patients and family members as well as prevent overutilization of clinical screening tests.

WHO SHOULD BE SCREENED?
A patient "at risk" is defined as a person with a family history of retinoblastoma in a parent, sibling, or first- or second-degree relative.

At-risk family members found to carry the same RB1 mutation as the proband (patient with RB who is the starting point for genetic testing) benefit from early and intensive screening for RB.
WHEN & HOW OFTEN TO SCREEN

**Genetic testing should be done for at-risk family members.**
- Children with a known RB1 mutation should have an eye examination every 3 to 6 weeks until age 1 year, and then every 3 months until age 3 years, and then every 6 months until age 6 years. This examination should be carried out by an ophthalmologist and requires examination under anaesthesia (EUA).
- Those who test negative for a familial RB1 mutation do not require ophthalmologic screening.

**Screening where genetic testing is not available:**
- Positive family history where the parent is proband – the child should be screened at birth (or soonest afterwards) then every month for 3 months, then 3-4 monthly up to 3 years.
- Siblings of children with RB should be screened at birth then after one month then 3-monthly for one year. If they are still clear of disease then screening can be stopped.

![Diagram 3: Flow diagram on RB screening in the absence of genetic testing](image)

**SCREENING TESTS TO BE DONE**
- Molecular genetic testing of the RB1 gene: Comprehensive genetic counselling must be done prior.
- At community level, the healthcare provider should enquire about family history of RB; if positive then refer for examination for children less than 2 years old.
Early detection/early diagnosis of childhood cancer results in better treatment outcomes.

EARLY DETECTION/ DIAGNOSIS OF CHILDHOOD CANCERS

Early detection of childhood cancers through early diagnosis results in better treatment outcomes. This depends on recognition, mainly at the community level where enhanced awareness regarding childhood cancers by parents and community health workers is important (Njuguna, 2016).

Every contact of a healthcare provider (HCP) with a child should provide an opportunity for comprehensive evaluation for possible signs and symptoms of cancer. It is important for the HCP to understand that cancer in children may present with non-specific signs and symptoms. When a child is examined and these signs and symptoms are found, cancer must be suspected and action taken accordingly to prevent late diagnosis.

COMMON SYMPTOMS AND SIGNS OF CHILDHOOD CANCER

Most of the signs and symptoms of childhood cancer are non-specific and require health care providers to have a high index of suspicion.
The following are common symptoms and signs of childhood cancer:

- Continued, unexplained weight loss
- Recurrent or persistent fevers of unknown origin
- Constant tiredness or noticeable paleness
- Development of excessive bruising, bleeding, or rash
- Increased swelling or persistent pain in bones, joints, back, or legs
- Lump or mass, especially in the abdomen, neck, chest, pelvis, or armpits
- Rapidly growing mass on the jaw
- A mass in the abdomen with or without bloody urine
- Headaches, often with early morning vomiting

Diagram 4: Plausible signs and symptoms of childhood cancers
EARLY DIAGNOSIS AT VARIOUS HEALTHCARE LEVELS

Early detection of childhood cancers requires a high index of suspicion and appropriate referral systems to avoid late presentation and worse outcomes. Figure 4 represents the referral system for such patients but early referral to the comprehensive cancer centres is also recommended when the cancer diagnosis is highly likely in order to avoid delays and loss to follow up of patients.

The following are the roles of healthcare providers at each level of care:

1. **Community level:**
   CHWs to mobilize parents or guardians of children with above symptoms to visit the nearest health facility.

2. **Level 2 & 3 (Primary healthcare facility) – Dispensary & health centre**
   - Evaluate the child using the Childhood Cancer Assessment Tool for early detection of cancer (see Figure 6)
   - Generate comprehensive referral document and refer appropriately to higher levels of care

3. **Level 4 - Sub-county/County Hospital**
   - Comprehensive physical examination and identification of plausible cancer cases
   - Basic lab investigations such as TBC and PBF, UEC, LFTs
   - Imaging tests such as ultrasound and x-ray
   - Supportive care such as blood transfusions; treatment of infections, palliative care and pain management
   - Generate comprehensive referral documentation to a childhood comprehensive cancer care center.

4. **Comprehensive Cancer Care Centre (Level 5 and 6)**
   - All the activities done at lower levels of care above
   - Management of cancer cases

THE IMCI (INTEGRATED MANAGEMENT OF CHILDHOOD ILLNESSES) APPROACH FOR ASSESSING A CHILD FOR POSSIBLE CANCER: Childhood Cancer Assessment tool

The diagram below is suitable to guide HCPs at all levels to diagnose childhood cancers early. They should classify the child’s health status through the given colour coding system, and take the corresponding required actions:

- **RED** - Urgent treatment and referral
- **YELLOW** - Outpatient treatment and advice
- **GREEN** - Advice on treatment and home care
Classification Table for Cancer Probability in Children

<table>
<thead>
<tr>
<th>ASSESS</th>
<th>CLASSIFY</th>
<th>TREAT</th>
</tr>
</thead>
<tbody>
<tr>
<td>One of the following signs:</td>
<td>Refer urgently to a high complexity hospital with a pediatric hematology/oncology service; if not possible, refer to a pediatric hospital</td>
<td></td>
</tr>
<tr>
<td>- Fever for over 7 days with no apparent cause</td>
<td>- Stabilize the patient, and if necessary, begin intravenous fluids, oxygen, and pain management</td>
<td></td>
</tr>
<tr>
<td>- Headache: persistent and progressive, and primarily nocturnal, that awakens the child or appears when rising in the morning and may be accompanied by vomiting</td>
<td>- If a brain tumor is suspected and there is neurological deterioration, begin management of intracranial hypertension</td>
<td></td>
</tr>
<tr>
<td>- Bone pain that has increased progressively in the last month and disrupts child’s activities</td>
<td>- Speak with the parents; explain the need and importance of referral and its urgency</td>
<td></td>
</tr>
<tr>
<td>- Petechiae bruises, and/or bleeding</td>
<td>- Resolve all administrative problems that occur</td>
<td></td>
</tr>
<tr>
<td>- Severe palmar or conjunctival pallor</td>
<td>- Communicate with the referral facility</td>
<td></td>
</tr>
<tr>
<td>- Leukokoria (white eye)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Strabismus that has newly appeared</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Aniridia (lack of iris)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Heterochromia (different colored eyes)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Hyphema (blood in the eye)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Proptosis (bulging eye)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Nodes &gt;2.5cm in diameter, hard, painless, lasting ≥4 weeks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Acute and/or progressive focal neurological signs and symptoms:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Convulsion without fever or underlying neurological disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Unilateral weakness (of one limb or one side of the body)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Physical asymmetry (facial)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Changes in consciousness or mental status (behavior change, confusion)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Loss of balance when walking</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Limping from pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Difficulty speaking</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Visual disturbances (blurred, double, sudden blindness)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Palpable abdominal mass</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Hepatomegaly and/or splenomegaly</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Mass in some region of the body with no signs of inflammation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Ensure immunization and growth and development monitoring</td>
<td>Do a complete physical examination to look for a cause for the signs found</td>
<td></td>
</tr>
<tr>
<td>• Ensure a tobacco-free environment</td>
<td>Review the child’s diet and correct any problems found</td>
<td></td>
</tr>
<tr>
<td>• Recommend a healthy diet and regular physical activity</td>
<td>If there is weight loss, loss of appetite, or fatigue or tiredness, refer for pediatric consultation to begin studies and to investigate possible TB, HIV</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• If there is mild palmar pallor, begin iron and follow-up every 14 days. If it worsens, refer urgently, if there is no improvement at one-month follow-up visit, request hemogram and blood smear to look for cause of anemia and to treat or refer as appropriate</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Treat the cause of lymphadenopathy with antibiotics if necessary and follow up in 14 days; if there is no improvement, refer</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Treat with antibiotics any inflammatory process that produces enlargement in a region of the body and follow up in 14 days; if there is no improvement, refer</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Teach danger signs requiring child to return immediately</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Ensure immunization and growth and development monitoring</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Ensure a tobacco-free environment</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Recommend a healthy diet and regular physical activity</td>
<td></td>
</tr>
</tbody>
</table>

Diagram 7: Childhood cancer assessment tool

HCPs need to have a high index of suspicion for early detection of childhood cancers through an integrated approach.
REFERENCES


TUMOR MARKERS & CANCER SCREENING

Introduction
Tumor markers are chemical substances in the body that are either produced by a cancer or by the body in response to a cancer. They are found mainly in blood, but also in urine and other body fluids of some people with certain cancers. They are also known as biomarkers.

Measuring the tumor markers can allow for determination of whether a particular type of cancer is actively growing (and thus producing the corresponding tumor marker). Most tumor markers are proteins or carbohydrate-proteins. More recently, there are also genetic molecules (DNA, RNA) serving as tumor markers. They are based on genetic changes associated with certain cancers.

There are many molecules that have been discovered to be associated with various cancers, but not all are used in the clinical setting because they fail to meet the criteria for such use. For molecules to be used reliably as tumor markers, they have to be both highly sensitive and specific:

- Sensitive: Able to detect presence of tumor even at low volume/low count, hence minimizing false negative results.
- Specific: Only found in the presence of the tumor and not in people who don’t have the tumor. This means there it will not result in false positives, which can cause undue stress for healthy patients.

USE OF TUMOR MARKERS IN CANCER CARE

Tumor marker tests may be useful for a number of purposes in cancer care. These include early detection, diagnosis, and management, including guiding appropriate treatment, staging, determining prognosis, monitoring response to treatment and monitoring disease recurrence. They are usually not used in isolation but in combination with other tests, since they are not definitive but provide additional information which can be helpful in various ways.

Regarding recurrence, once a particular tumor marker has been found to be elevated in a patient with a particular cancer before treatment, it can be used to monitor for recurrence of the cancer after treatment is completed. Some tumor markers may help to detect a recurrence sooner than other tests.
Different tumor markers may be used for different cancers, such as tabulated below:

<table>
<thead>
<tr>
<th>TUMOR MARKER</th>
<th>ASSOCIATED CANCER</th>
<th>USES</th>
</tr>
</thead>
<tbody>
<tr>
<td>AFP (Alpha-feto protein)</td>
<td>Liver</td>
<td>Diagnosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Monitoring disease progression &amp; response to treatment</td>
</tr>
<tr>
<td></td>
<td>Testicular cancer</td>
<td>Diagnosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Monitoring response to treatment</td>
</tr>
<tr>
<td>CA 15-3 (Cancer antigen 15-3)</td>
<td>Breast</td>
<td>Monitoring disease progression &amp; response to treatment</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Assessing recurrence</td>
</tr>
<tr>
<td>CA 19-9 (Cancer antigen 19-9)</td>
<td>Pancreatic cancer</td>
<td>Diagnosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Staging</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Monitoring disease progression &amp; response to treatment</td>
</tr>
<tr>
<td>CA-125 (Cancer antigen 125)</td>
<td>Ovarian</td>
<td>Early detection*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Monitoring disease progression &amp; response to treatment</td>
</tr>
<tr>
<td>Calcitonin</td>
<td>Medullary thyroid cancer</td>
<td>Diagnosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Monitoring disease progression &amp; response to treatment</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Assessing recurrence</td>
</tr>
<tr>
<td>CEA (Carcinoembryonic antigen)</td>
<td>Colorectal</td>
<td>Staging</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Monitoring disease progression &amp; response to treatment</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Assessing recurrence</td>
</tr>
<tr>
<td>HCG (Human chorionic gonadotropin)</td>
<td>Testicular</td>
<td>Diagnosis</td>
</tr>
<tr>
<td></td>
<td>Trophoblastic disease</td>
<td>Staging</td>
</tr>
<tr>
<td></td>
<td>Choriocarcinoma</td>
<td>Monitoring disease progression &amp; response to treatment</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Assessing recurrence</td>
</tr>
</tbody>
</table>
**PSA (prostate-specific antigen)**
Prostate
Early detection* Diagnosis
Monitoring disease progression & response to treatment

**Molecular Tests**

<table>
<thead>
<tr>
<th>Test</th>
<th>Organs</th>
<th>Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>EFGR (Epidermal Growth Factor Receptors)</td>
<td>Lung</td>
<td>To guide treatment &amp; determine prognosis</td>
</tr>
<tr>
<td>KRAS</td>
<td>Colon, lung</td>
<td>To guide treatment</td>
</tr>
<tr>
<td>BCR-ABL</td>
<td>CML (Chronic Myeloid Leukaemia)</td>
<td>Diagnosis, monitoring treatment &amp; detecting recurrence</td>
</tr>
</tbody>
</table>

*in very restricted settings as explained below

There is a lot of ongoing research on many other potential tumor markers.

**LIQUID BIOPSY**
This refers to a new approach that involves the use of blood or other body fluids as a specimen to detect genes produced by a tumor. It is being shown to have a high sensitivity in detecting the presence of a tumor and therefore allowing early diagnosis and hence improving the success of treatment. Just like the other tumor markers, their absence does not exclude the presence of the corresponding cancer. Examples include EGFR for non-small cell lung cancer, KRAS for colon cancer and BRCA1 for breast cancer. Research is still underway to determine the usefulness of liquid biopsies in cancer screening in the general population.

**LIMITATIONS FOR USE OF TUMOR MARKERS**
The use of tumor markers is limited by a number of factors. First, not all cancers of a particular type produce the associated tumor marker - some patients with a particular cancer type may not have elevated levels of the associated tumor marker even if the type of cancer they have usually make the particular tumor marker. Secondly, a tumor marker may not be exclusive to a particular type of cancer - different types of cancers may produce the same tumor marker. For example, CEA is associated with colon cancer, but may also be produced by other cancers such as lung, breast, thyroid, pancreatic, liver, cervix, and bladder cancers. Also, some noncancerous conditions can also be associated with high levels of tumor markers. E.g. Benign ovarian cysts – CA-125; Colon obstruction- CEA and bile duct obstruction – CA 19-9. Further, not all cancers produce tumor markers that have been identified with them.
TUMOR MARKERS IN CANCER SCREENING

Most tumor markers have NO ROLE IN CANCER SCREENING in the general population

Presently, most tumor markers have NO ROLE in cancer screening in the general population. Screening involves the use of medical evaluation (in this case a laboratory test) to detect a cancer before it has developed. Since tumor markers are produced by cancers or by the body in response to cancer, there is no tumor marker that can be used for screening. In addition, most tumor markers are neither sensitive nor specific enough to be used on their own to screen for cancer. This means that their use would result in too many false positives resulting in harmful effects such as anxiety, need for expensive and unnecessary follow-up tests.

However, there is a role for tumor markers in detecting cancers early before they become symptomatic. Since some patients with a particular cancer may not express the corresponding tumor marker, and some patients who do not have a particular cancer may still have elevated levels of the tumor marker due to other conditions that are not cancer, the use of tumor markers in early detection of cancer is VERY RESTRICTED. Very few tumor markers are thus useful for this, and in limited settings. They can be used for screening high risk individuals with either a strong family history or with specific risk factors for a particular cancer, including:

1. **PSA**
   Refer to Prostate Cancer Screening guidelines, Pages 84 - 94

2. **CA 125**
   For early detection of ovarian cancer in females with a positive family history or with abdominal masses.
   How often to test: The frequency of follow up has not yet been determined and there are no established guidelines yet.
   A raised CA 125 does not equate to having cancer. Biopsy is needed for definitive diagnosis.
   No evidence supports the use of CA 125 to screen the general population for ovarian cancer.
WHEN TO USE TUMOR MARKERS

Unfortunately, tumor markers are often misused in the guise of screening by unscrupulous persons claiming that they can detect cancer early, whereas that is but a false notion. Some of the tumor markers that are commonly misused are AFP, CA 15-3, CA 19-9, Calcitonin, CEA and hCG. Their appropriate uses are as indicated earlier.

Tumor markers can be used to promote prompt diagnosis during clinical evaluation in cases where there is a high index of suspicion at the point of contact with the patient. They can be used as an aid to clinical diagnosis because they are relatively inexpensive and accessible (compared to radiology and biopsy). They can also be used for early detection of recurrence.

It must be noted though that the presence or elevated levels of a tumor marker alone is not enough to diagnose cancer. Furthermore, tumor markers are only useful in patients with cancers expressing the tumor markers, therefore a negative result for a tumor markers test does not exclude the presence or recurrence of the cancer.

REFERENCES


American Society of Clinical Oncology
ANNEX

DATA DOCUMENTATION TOOLS

Cervical Cancer Screening Tools
- Cervical Cancer Screening Card
- Cervical Cancer Screening and Treatment Form
- Cervical Cancer Referral Form
- Daily Activity Register
- Cervical Cancer Program Monthly Summary Form
- ANC Register
- PNC Register
- MOH 711 Integrated summary

Current Indicators for Cervical Cancer Available in DHIS
- Cervical cancer clients receiving VIA /VILI /HPV VILI / HPV
- Cervical cancer clients with Positive VIA/VILI result
- Cervical cancer clients screened using Pap smear
- Cervical cancer clients with suspicious cancer lesions
- Cervical cancer clients with Positive Cytology result
- Cervical cancer clients treated using Cryotherapy
- Cervical cancer treated using LEEP
- Cervical cancer clients with Positive HPV result
- Cervical cancer clients screened using HPV test
SECTION A: SOCIO-DEMOGRAPHIC DATA

Inpatient/Outpatient number_______________ National ID no_______________

Name_________________________________________ Sex_________ Age (years) ______

Marital status_____________ No. of children __________

Patient phone no___________________ Address____________________

Next of kin (nok) name____________________ relationship to n.o.k______________ N.O.K. phone

No__________________

Current residence: county______________Sub-county_________________ Ward/Estate___________

Length of time lived in current residence (years) ___________

Highest educational level___________________ Occupation________________________

Ethnicity/Race________________________________________________

Where did you learn about this screening program?

Word of mouth ☐ From media ☐

Healthcare worker ☐ other ☐ (specify) ___________

Screening service point: MCH/FP ☐ CCC ☐ GOPC ☐ OUTREACH ☐

other ☐ (specify) ______________

Referred to this facility? Yes ☐ No ☐ if yes, from______________________________

REASON FOR

REFERRAL____________________________________________

VITAL SIGNS: BP____________________ PULSE RATE____________________

WEIGHT_________________ HEIGHT_________________ BMI_______________

BLOOD SUGAR LEVEL__________________________
SECTION B: FAMILY HISTORY

Any history of cancer in the family?

If yes, which cancer? __________________________________________

Who was affected? Parent ☐ Sibling ☐ 1st or 2nd degree relative ☐

Other ☐ (Specify) __________________

What was the age at diagnosis? (Years)____________________________

What was the sex of the person affected? Male ☐ Female ☐

SECTION C: CLINICAL/RISK FACTOR HISTORY

Tick as appropriate

RISK FACTORS

Risk factors history T

Smoking

Alcohol intake

Previous chemotherapy or radiation treatment

Any other (specify)

COMMON SYMPTOMS

Symptom history

Tick

Recurrent indigestion (dyspepsia)

Blood in stool

Yellow eyes

Blood in urine

Epistaxis (nose bleeding)

Difficulty in swallowing

General weight loss

Easy fatigability, palpitations

Abnormal vaginal bleeding

Enlarging/changing skin moles

Chronic skin ulcers

Any lumps or swellings

Chronic cough

Persistent headaches

Changing bowel habits

Others (specify)
### SECTION D: TYPE OF CANCER SCREENING

<table>
<thead>
<tr>
<th>Cancer Type</th>
<th>Screening Type</th>
<th>Screening Modality</th>
<th>Last Screening Modality Done</th>
<th>Date of Last Screening</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cervical</strong></td>
<td>Initial screening</td>
<td>HPV testing</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Repeat screening</td>
<td>Pap smear</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post-treatment screening</td>
<td>VIA/VILI</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Breast</strong></td>
<td>Initial screening</td>
<td>Clinical breast examination</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Repeat screening</td>
<td>Ultrasound</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post-treatment screening</td>
<td>Mammogram</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Prostate</strong></td>
<td>Initial screening</td>
<td>DRE in combination with PSA testing</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Repeat screening</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post-treatment screening</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Colorectal</strong></td>
<td>Initial screening</td>
<td>Fecal occult blood test</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Repeat screening</td>
<td>Colonoscopy</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post-treatment screening</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Retinoblastoma (known) / Retinoblastoma 1 mutation or positive family history</strong></td>
<td>At birth</td>
<td>Eye exam under anaesthesia</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vaccination clinic</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Retinoblastoma screening frequency**

1. Known RB 1 mutation on genetic testing:
   - Every 6 weeks until 1 year, then every 3 months until 3 years, then every 6 months until 6 years
2. No genetic testing available
   - Option 1 – positive family history for parent
     - At birth, then every month for 3 months, then every 3 months for 3 years
   - Option 2 – positive family history for sibling
     - At birth, then every month for 3 months, then every 3 months for 1 year
## SECTION E: SCREENING RESULTS

<table>
<thead>
<tr>
<th>Cancer Site</th>
<th>Screening modality</th>
<th>Results/findings</th>
<th>Recommended action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervical</td>
<td>HPV</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pap smear</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>VIA/VILI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breast</td>
<td>Clinical breast examination</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ultrasound (&lt;40 years)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mammogram ≥ 40 years</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prostate</td>
<td>DRE in combination with PSA testing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Colorectal</td>
<td>Fecal occult blood test</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Colonoscopy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Retinoblastoma</td>
<td>Eye exam</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

## SECTION F: FOLLOW UP

Return date

Referred to

Referred for further screening (give reasons)

Health service provider:
Name Cadre Signature
THE TYRER-CUZIK MODEL FOR RISK ASSESSMENT MODEL

<table>
<thead>
<tr>
<th>Personal History</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
</tr>
<tr>
<td>Weight</td>
<td></td>
</tr>
<tr>
<td>Height</td>
<td></td>
</tr>
<tr>
<td>Menarche</td>
<td></td>
</tr>
<tr>
<td>Has the woman given birth to more than one child</td>
<td>Unknown</td>
</tr>
<tr>
<td>If yes, at what age was the first live birth?</td>
<td></td>
</tr>
<tr>
<td>Has the woman gone through menopause</td>
<td>Don’t know</td>
</tr>
<tr>
<td>If yes, at what age?</td>
<td></td>
</tr>
<tr>
<td>Hormonal replacement use</td>
<td>Never</td>
</tr>
<tr>
<td>Does the woman have BRCA 1/2</td>
<td>Unknown</td>
</tr>
<tr>
<td>Has the woman had ovarian cancer</td>
<td>No</td>
</tr>
<tr>
<td>Has the woman had breast biopsy</td>
<td>No prior biopsy</td>
</tr>
<tr>
<td>Family history of breast or ovarian cancer</td>
<td>No</td>
</tr>
</tbody>
</table>

**NOTE:**
2. Not intended to assess the risk of women who have already been diagnosed with breast cancer
<table>
<thead>
<tr>
<th>NAME</th>
<th>ORGANIZATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Christine Were</td>
<td>PS-Kenya</td>
</tr>
<tr>
<td>2. David Makumi</td>
<td>KENCO</td>
</tr>
<tr>
<td>3. Dorcas J. Kiptui</td>
<td>MOH –DNCD</td>
</tr>
<tr>
<td>4. Dr Ahmed Kalebi</td>
<td>Pathologists Lance Kenya</td>
</tr>
<tr>
<td>5. Dr Alfred Karagu</td>
<td>National Cancer Institute (NCI) - Kenya</td>
</tr>
<tr>
<td>6. Dr Andrew Odhiambo</td>
<td>UoN</td>
</tr>
<tr>
<td>7. Dr Anne Ng’ang’a</td>
<td>MOH – Head, NCCP</td>
</tr>
<tr>
<td>8. Dr Catherine Murithi</td>
<td>Roche Diagnostics</td>
</tr>
<tr>
<td>9. Dr Charles Muturi</td>
<td>Mama Lucy Kibaki Hospital, Nairobi County</td>
</tr>
<tr>
<td>10. Dr Daniel Ojuka</td>
<td>KNH</td>
</tr>
<tr>
<td>11. Dr David Kimani</td>
<td>KNH</td>
</tr>
<tr>
<td>12. Dr Doreen Mutua</td>
<td>Gertrude’s Children’s Hospital</td>
</tr>
<tr>
<td>13. Dr Edna Kamau</td>
<td>KNH</td>
</tr>
<tr>
<td>14. Dr Elizabeth Dimba</td>
<td>UoN</td>
</tr>
<tr>
<td>15. Dr Eric Hungu</td>
<td>KNH</td>
</tr>
<tr>
<td>16. Dr Eunice W. Gathitu</td>
<td>MOH - NCCP</td>
</tr>
<tr>
<td>17. Dr Gladwell Kiarie</td>
<td>Nairobi Hospital</td>
</tr>
<tr>
<td>18. Dr Grace Kariuki</td>
<td>MOH – DNCD/FELTP</td>
</tr>
<tr>
<td>19. Dr Gregory Ganda</td>
<td>JOOTRH</td>
</tr>
<tr>
<td>20. Dr Jamilla Rajab</td>
<td>UoN</td>
</tr>
<tr>
<td>21. Dr Joan-Paula Bor</td>
<td>MOH - NCCP</td>
</tr>
<tr>
<td>22. Dr Joseph Kibachio</td>
<td>MOH – Head, DNCD</td>
</tr>
<tr>
<td>23. Dr Khadija Warfa</td>
<td>AKUH</td>
</tr>
<tr>
<td>24. Dr Lilian Mbau</td>
<td>Amref Health Africa</td>
</tr>
<tr>
<td>25. Dr Linus Ndegwa</td>
<td>KEMRI</td>
</tr>
<tr>
<td>26. Dr Mary Nyangasi</td>
<td>MOH – NCCP</td>
</tr>
<tr>
<td>27. Dr Michael Mwachiro</td>
<td>Tenwek Hospital</td>
</tr>
<tr>
<td>28. Dr Miriam Mutebi</td>
<td>AKUH</td>
</tr>
<tr>
<td>29. Dr Njoki Njiraini</td>
<td>MOH/KNH</td>
</tr>
<tr>
<td>30. Dr Richard Njoroge</td>
<td>MOH – NCCP/NPHLS</td>
</tr>
<tr>
<td>31. Dr Sarah Muma</td>
<td>Kijabe Hospital</td>
</tr>
<tr>
<td>32. Dr Teresa Kinyari Mwendwa</td>
<td>UoN/PHSK</td>
</tr>
<tr>
<td>33. Dr Valerian Mwenda</td>
<td>MOH – NCCP/FELTP</td>
</tr>
<tr>
<td>34. Dr Vera Manduku</td>
<td>KEMRI</td>
</tr>
<tr>
<td>35. Dr Wycliffe Kaisha</td>
<td>KNH</td>
</tr>
<tr>
<td>36. Evans Obaga</td>
<td>MOH – NCCP</td>
</tr>
<tr>
<td>37. Hannah N. Gitungo</td>
<td>MOH – NCCP</td>
</tr>
<tr>
<td>38. Linda Ogol</td>
<td>MOH-NCCP</td>
</tr>
<tr>
<td>39. Lydia W. Kirika</td>
<td>MOH – NCCP</td>
</tr>
<tr>
<td>40. Pamela Kirika</td>
<td>MTRH/AMPATH Oncology Institute</td>
</tr>
<tr>
<td>41. Patricia Njiri</td>
<td>CHAI</td>
</tr>
<tr>
<td>42. Prof Jessie Githang’a</td>
<td>UoN</td>
</tr>
<tr>
<td>43. Prof Lucy Muchiri</td>
<td>UON</td>
</tr>
<tr>
<td>44. Roselyn Okumu</td>
<td>KNH</td>
</tr>
</tbody>
</table>
LIST OF REVIEWERS

• Dental Services Unit - MOH
• Ophthalmic Services Unit - MOH
• Reproductive and Maternal Health Services Unit (RMHSU)
• Gastroenterology Society of Kenya (GSK)
• Kenya Association of Radiologists (KAR)
• Kenya Association of Urological Surgeons (KAUS)
• Kenya Dental Association (KDA)
• Kenya Obstetrical and Gynaecological Society (KOGS) - Gynaecologic Oncology Committee
• Kenya Society of Hematology & Oncology (KESHO)
• Surgical Society of Kenya (SSK)
• Prof Constance Tenge – MTRH/Moi University
• Prof Elly Ogutu – UoN
• Dr Festus Njuguna – MTRH/Moi University
• Dr Gladwell Gathecha – MOH DNCD
• Dr Johnson Wambugu – Kenya Dental Association
• Dr Kahaki Kimani – UoN
• Prof Mark Chindia – UoN
• Dr Russell E. White – Tenwek Hospital
• Dr Wilson Miriti - KNH