

State of cancer in Bangladesh: An introductory review on microbiological perspective

Saurab Kishore Munshi, Ifra Tun Nur and Rashed Noor*

Department of Microbiology, Stamford University Bangladesh, 51 Siddeswari Road, Dhaka 1217, Bangladesh

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Present review emphasized on the current state of cancer in Bangladesh and attempted to focus on the association of microorganisms in the development of cancer. People of Bangladesh have long been suffering from a range of diseases principally due to lack of required resources for disease diagnosis and subsequent treatment facilities. As a developing country with the lack of public awareness on hygiene, emerging infectious diseases conquered the countries. In this circumstance, the onset of cancer is appears as the incurable complications. In the country, cancer is an escalating fatal issue causing a mass scale of mortality each year. Moreover, while etiologic microorganisms are frequently detected during the common disease diagnosis, the pathogenic association with cancer formation remains obscure to the physicians in Bangladesh. While mortality and survival rate of cancer largely depends on the diagnosis and subsequent mitigation, hence it is essential to resolve the possible risk of cancer development in accordance to microbial attack.

Key words: Emerging infectious diseases; Cancer; Microorganisms; Risk of cancer development

Cancer is known as a group of disease complications which are commonly characterized by the uncontrolled growth and proliferation of cells, resulting in death (1). It is indeed a pandemic burden affecting people of all ages, races, incomes, and statuses; principally by commencing a state of sustaining proliferative signaling, avoiding the growth suppressors, protecting the defective cell death, accelerating the replicative immortality, inducing angiogenesis, and finally by activating invasion and metastasis (1-6). However, cancer has been commonly noticed to develop in older people; and interestingly 78% of all cancer diagnoses are being conducted in people 55 years of age or older (1). Cancer is usually triggered by tobacco usage, exposure to infectious microorganisms, consumption of unhealthy diet, and by the inherited genetic alterations, hormones, and immune suppressive conditions (1, 7-10). Cancer initiation has long been known to require only a single exposure to a carcinogen with the concomitant effect on DNA damage. Though a single genetic change may suffice the onset of a malignant tumor, most of the researches pointed to a multistep process of chronological alterations in many oncogenes, tumor-suppressor genes, or microRNA genes (11-16). Cancer promotion involves multiple exposures to the carcinogens that do not trigger the DNA damage directly; rather this stage involves the conversion of benign to malignant tumors which in turn continue to

progress in their degree of malignancy and heterogeneity (11-15). The molecular mechanisms of cancer development have also been well chalked out during the last decade (16-21). Treatment of cancer is till date ambiguous; however, decline of cancer progression may be set by the site specific surgery, irradiation, chemotherapy, hormone therapy, immune therapy, and finally by the drugs that specifically interfere with cancer cell growth (i.e., targeted therapy) (1, 22, 23). The first tumor-suppressor protein discovered was the Retinoblastoma protein (pRb) in human retinoblastoma; however, recent studies has also implicated pRb as a tumor-survival factor (24, 25). Another important tumor suppressor is the p53 tumor-suppressor protein encoded by the TP53 gene (25).

There were an estimated 14.1 million cancer cases around the world in 2012, of which 8.2 million went dead (6). Among these, lung cancer was responsible for 17.8% cases, stomach cancer for 10.4% cases and liver cancer for 8.8% of all cancer originated deaths (1). The highest numbers of cancer induced deaths (72%) are found in low and middle income countries with primitive or limited medical facilities and financial support (6). It is also the second leading cause of death in the United States, where 23% of peoples deaths from cancer (1). The high mortality rates caused by cancer are noticed in Australia/New Zealand, Western Europe, Central and Eastern Europe, Eastern Asia, South-Eastern Asia, Eastern Africa, Southern Africa, North America, Caribbean and South America (6).

Since Bangladesh is a developing country with

*Corresponding Author: Mailing address. Dr. Rashed Noor, Department of Microbiology, Stamford University Bangladesh, 51 Siddeswari Road, Dhaka 1217, Bangladesh, Bangladesh; E-mail: noor.rashed@yahoo.com.

high density population accompanied with limitations in disease diagnosis and subsequent mitigation, the Bangladeshi community suffers from varieties of diseases (26). While documentation covers the ongoing incidence of cancer with the associated mortality and survival rate, cancer symptoms and diagnosis, research and reviews on the possible risk of cancer development in accordance to microbial attack is scarce in Bangladesh. However, cancer is an increasingly imperative cause of morbidity and mortality in Bangladesh with the projected incidence of 12.7 million new cancer cases each year (27). The present review focuses on the current state of cancer in Bangladesh together with the association of microorganisms in the development of cancer.

ASSOCIATION OF MICROORGANISMS IN THE DEVELOPMENT OF CANCER

Viral or bacterial infections have been linked to higher risks of onset of cancer leading to malignancy (28-35). Although viral infections are known to be inherently associated with developing cancers, bacterial associations, like *Helicobacter pylori* (triggering gastric cancer and mucosa associated lymphoid tissue lymphoma), *Salmonella typhi* (inducing gallbladder cancer), *Streptococcus bovis* (causing colon cancer) and *Chlamydia pneumonia* (associated with lung cancer) are also significant (36-43). One of the bacterial pathogens reported to be regularly associated with colorectal carcinoma (CRC) is *Streptococcus bovis/gallolyticus* (32). *C. pneumoniae* infection has been reported to be associated with the increased risk of lung cancer (32). Several bacterial species including *Escherichia coli* and several streptococci have been linked to chronic infections of the colon and increased risk of colon cancer (35).

Chronic infection with concomitant production of toxins disturbing the cell cycle thereby triggering DNA damage, immune system evasion or stimulation contributing to carcinogenic changes through the stimulatory and mutagenic effects of cytokines released by inflammatory cells, chronic stimulation of reactive oxygen species (ROS), interleukin-8 (IL-8), cyclooxygenase-2 (COX-2), , reactive oxygen species (ROS) and nitric oxide (NO) generally account for the carcinogenesis mediated by the bacteria (3-47). The occurrence of bacteria within tumors could be due to the infection via the vasculature and their ability of growth and survival supported by nutrients within the hypoxic region of the tumour at a later stage in tumor growth (32).

Viral infections have long been known to pose high risks of cancer round the globe (24, 25, 48, 49). Approximately one-fifth of all cancers (including

the cancers of cervix, liver, bladder, colon, etc.) worldwide are caused by chronic infections produced principally by viral agents (6, 50-54). Infection with carcinogenetic pathogens including human papillomavirus (HPV), Epstein-Barr virus (EBV), *Helicobacter pylori* and esophageal bacterial biota has been proposed as a risk factor for esophageal cancer (29). However, HPV is most likely to contribute to esophageal squamous cell carcinoma (ESCC) in high-risk populations (29). Epstein-Barr virus (EBV) has a long history of generating tumors, which is specifically known as the causative agent of Burkitt's lymphoma, nasopharyngeal carcinoma, breast carcinomas and hepatocellular carcinomas (6, 55-58). Human papillomavirus (HPV) is a DNA virus containing a large genome of the papilloma virus family, which is known as the etiological agent of cervical cancer (59-65). Several studies reported that smoking and alcohol synergistic effects may lead HPV infections to one of the major risk factors for cancer in oral cavity (6. 66-73). HTLV-1 stimulated) adult T-cell leukaemia/lymphoma (ATL), in which T lymphocyte cells become cancerous (1, 74-76).

CURRENT STATUS OF CANCER IN BANGLADESH

As a developing country, Bangladesh is conquered by an array of diseases including the incurable cancers which mostly remains undiagnosed as well as lacks the general public health awareness. Cancer is indeed the sixth leading cause of death in this country with the incidence of lung cancer in males, and breast and cervical cancers in females (27). Recent studies conducted in Bangladesh revealed that nearly two million cancer patients currently exist in Bangladesh and every year about two hundred thousand patients are affected by cancer with death estimation of about 150,000 per year (77-79). At present, Bangladesh is known as the ninth most populous country in the world regarding cancer sufferings (79). Lung cancer, mouth-oropharynx cancer, esophagus cancer and stomach cancer in male are predominant while in women, cancer of cervix, uterus and breast are most frequent in addition to the onset of mouth and oropharynx cancer, lung cancer, and esophagus cancer (27, 80, 81). According to the estimation by the National Institute of Cancer Research and Hospital in 2014, approximately 29% male fight against lung cancer and 26% female patients battle against breast cancer (77).

CANCER DIAGNOSIS AND TREATMENT IN BANGLADESH

Lots of research on cancer eradication is ongoing in Bangladesh (27, 79-90). However, for the cancer treatment purposes, the country has a very little quantity of qualified clinical oncologists (less than 200) working in about 20

hospitals with the facilities for chemotherapy in the oncology/radiotherapy departments (26, 91). Approximately 60 cancer chemotherapeutic agents are available in Bangladesh with the research facilities available at few tertiary care centers. This is to be mentioned that Bangladesh has a unique National Cancer Control Strategy and Plan of Action 2009–2015 formulated in collaboration with WHO with an objective to improve the cancer situation by raising the preventive measures including reduced tobacco smoking, change of dietary habit and reduced food adulteration, ensuring reproductive hygiene, increased physical activity, and reduced occupational hazard in order to reduce the incidence of cancer. Some other major cancer preventive programs have also been undertaken by the governmental and non-governmental organizations (79, 90, 92-94).

CANCER DIAGNOSIS IN MICROBIAL PERSPECTIVE

As stated earlier, lots of researches on detecting microorganisms associated with varieties of diseases have been conducted so far in Bangladesh while such effort to characterize microbial proliferation in cancer cases is still scarce (88, 95-108). So far in Bangladesh, cancers associated with papilloma virus infection, Hepatitis B and C infection, *Helicobacter pylori* infections have been mostly reported (27). Hence the microbiologists should take care of the microbial diagnosis of cancer cases in order to correlate the microbial infections to the possible cancer onsets. As stated elsewhere, Bangladesh has developed a National Cancer Control Strategy and Action Plan with the aim of delivering a universal, quality-based and timely service by which the cancer prevention through tobacco control, health promotion and vaccination program, cancer early detection program for oral cavity, breast and cervix has initiated (27).

According to the estimation by the International Agency for Research on Cancer (IARC), the cancer-related death rate in Bangladesh will increase to 13% by 2030 (27). The 10 leading causes of deaths due to cancer in males have been identified by IARC as the mouth and oro-pharyngeal, esophageal, pharynx, stomach, larynx, colorectal, lymphoma, liver and bladder cancers in males, while in females such mortality derive from the mouth, cervical, breast, oro-pharyngeal, lung, esophageal, gallbladder, stomach, ovary, liver and colorectal cancers (27). Microorganisms associated with these cancers must be isolated and quantified, and the possible sources of such etiological agents should be chalked out as well. Another important point is to ponder on the usage of

tobacco, which is a prime risk factor for cancer. 20 million people in Bangladesh use tobacco in some form, and including 5 million women and 57 000 people die every year due to tobacco-related diseases (27). Thus besides the microbiological research, health awareness among mass population regarding the stoppage of tobacco usage for cancer prevention, dissemination of information on early signs and symptoms of cancer are required.

RISK FACTORS ON COMMONLY DIAGNOSED CANCERS

For most types of cancer, risk is usually higher with a family history of the disease, as especially is noticed in cases of prostate cancer, thyroid cancer, and skin cancer (1). Women who have one first-degree relative (mother, sister, or daughter) with a history of breast cancer are nearly twice as likely to develop breast cancer and to some extent, the ovarian cancer. Inherited mutations in *BRCA1* and *BRCA2* genes may account for 5–10% of all female breast cancers (1, 4, 7, 23). People who have a habit of smoking are about 25 times more likely to develop lung cancer than nonsmokers (1, 68). The risk of colorectal- and kidney cancer is likely to increase with age along with obesity, high blood pressure, physical inactivity, alcohol consumption, smoking; consumption of red or processed meat; and very low intake of whole-grain fiber, fruit, and vegetables and calcium (1, 28). Exposure to ionizing radiation even used in cancer treatment is known to increase the risk of most types of leukemia (1). The risk factors for liver cancer include obesity, diabetes, alcoholic liver disease, chronic infection with hepatitis B virus (HBV) or hepatitis C virus (HCV), together with the habit of smoking (1, 28). Smoking is indeed the most important risk factor for lung cancer, throat cancer, urinary bladder cancer, myeloid leukemia, and pancreatic cancer too (1, 67-70). Indeed, the tobacco smoking related diseases are most responsible causes for the deaths globally with an estimation of 6 million premature deaths a year (1).

CONCLUSION

The global burden of cancer can be prevented through the implementation of existing knowledge on cancer among mass community including encouraging healthy diet together with an improved life style and physical activities, vaccination programs (especially for liver and cervical cancers), discouraging tobacco usage, early detection and treatment measures both by the relevant professionals, enhancing the microbial research to diagnose the microbial agents associated with cancers, and finally by the public health campaigns promoting the public concerns on cancer. In perspective of Bangladesh, strengthening the cancer management infrastructure together with the microbiology based cancer diagnosis

facilities are emerging issue for the betterment of the mass public health state.

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REFERENCES

- American Cancer Society.** 2014. Cancer Facts and Figures. *Lancet*. 371 (9625): 1695–1709.
- Torre LA, Bray F, Siegel RL, Ferlay J, Lortet-Tieulent J, Jemal ACA.** 2015. Global cancer statistics, 2012. *Cancer J Clin*. 65 (2): 87–108.
- Bette M, Mandic R.** 2014. Intra-peritoneal oxidative stress as an oncolytic immunomodulator? *Oncoimmunology*. 3 (9): e955347.
- Herbein G, Kumar A.** 2014. The oncogenic potential of human cytomegalovirus and breast cancer. *Front Oncol*. 4: 230.
- Hanahan D, Weinberg RA.** 2011. Hallmarks of cancer: the next generation. *Cell*. 144 (5): 646–74.
- Jemal A, Bray F, Melissa M, Ferlay J, Ward E, Forman D.** 2011. Global Cancer Statistics. *CA Cancer J Clin*. 61: 69–90.
- Bhagirath D, Zhao X, West WW, Qiu F, Band H, Band V.** 2015. Cell type of origin as well as genetic alterations contribute to breast cancer phenotypes. *Oncotarget*. 6 (11): 9018–30.
- Wu H, Gao L, Li F, Song F, Yang X, Kasabov N.** 2015. Identifying overlapping mutated driver pathways by constructing gene networks in cancer. *BMC Bioinformatics*. 16 (Suppl 5): S3.
- Leiserson MD, Blokh D, Sharan R, Raphael BJ.** 2013. Simultaneous identification of multiple driver pathways in cancer. *PLoS Comput Biol*. 9 (5): e1003054.
- Vandin F, Upfal E, Raphael BJ.** 2012. De novo discovery of mutated driver pathways in cancer. *Genome Res*. 22 (2): 375–85.
- Caja F, Vannucci L.** 2015. TGFβ: A player on multiple fronts in the tumor microenvironment. *J Immunotoxicol*. 12 (3): 300–7.
- Miyagi T, Takahashi K, Shiozaki K, Yamaguchi K, Hosono M.** 2015. Plasma membrane-associated sialidase confers cancer initiation, promotion and progression. *Adv Exp Med Biol*. 842: 139–45.
- Zarogoulidis P, Tsakiridis K, Karapantou C, Lampaki S, Kioumis I, Pitsiou G, et al.** 2015. Use of proteins as biomarkers and their role in carcinogenesis. *J Cancer*. 6 (1): 9–18.
- White MK, Pagano JS, Khalili K.** 2014. Viruses and human cancers: a long road of discovery of molecular paradigms. *Clin Microbiol Rev*. 27 (3): 463–81.
- Weinstein IB.** 1988. The origins of human cancer: molecular mechanisms of carcinogenesis and their implications for cancer prevention and treatment –twenty–seventh G. H. A. clowes memorial award lecture. *Cancer Res*. 48: 4135–4143.
- Reddy KB.** 2015. MicroRNA (miRNA) in cancer. *Cancer Cell Int*. 15: 38.
- Torgovnick A, Schumacher B.** 2015. DNA repair mechanisms in cancer development and therapy. *Front Genet*. 6: 157.
- Weissmueller S, Machado E, Saborowski M, Morris JP, Wagenblast E, Davis CA, et al.** 2014. Mutant p53 drives pancreatic cancer metastasis through cell–autonomous PDGF receptor β signaling. *Cell*. 157 (2): 382–94.
- Liu DP, Song H, Xu Y.** 2010. A common gain of function of p53 cancer mutants in inducing genetic instability. *Oncogene*. 29 (7): 949–56.
- Mendoza-Rodríguez CA, Cerbón MA.** 2001. Tumor suppressor gene p53: mechanisms of action in cell proliferation and death. *Rev Invest Clin*. 53 (3): 266–73.
- Bos JL.** 1998. *ras* oncogenes in human cancer: a review. *Cancer Res*. 49 (17): 4682–9.
- Punglia RS, Burstein HJ, Weeks JC.** 2012. Radiation therapy for ductal carcinoma in situ: a decision analysis. *Cancer*. 118: 603–611.
- Farhat GN, Walker R, Buist DS, Onega T, Kerlikowske K.** 2010. Changes in invasive breast cancer and ductal carcinoma in situ rates in relation to the decline in hormone therapy use. *J Clin Oncol*. 28: 5140–5146.
- Cooper G.** 1995. *Oncogenes*. Jones and Bartlett Publishers.
- Sherr CJ.** 2004. Principles of tumor suppression. *Cell*. 116 (2): 235–46.
- Noor R, Munna MS.** 2015. Emerging diseases in Bangladesh: current microbiological research. *Tzu Chi Med J*. 27 (2): 49–53.
- Hussain SA, Sullivan R.** 2013. Cancer control in Bangladesh. *Jpn J Clin Oncol*. 43 (12): 1159–69.
- Arvelo F, Sojo F, Cotte C.** 2015. Biology of colorectal cancer. *E cancer medical science*. 9: 520.
- Xu W, Liu Z, Bao Q, Qian Z.** 2015. Viruses, Other Pathogenic Microorganisms and Esophageal Cancer. *Gastrointest Tumors*. 2: 2–13.
- Perez-Chanona E, Jobin C.** 2014. From promotion to management: the wide impact of bacteria on cancer and its treatment. *Bioessays*. 36 (7): 658–64.
- Alibek K, Kakpenova A, Mussabekova A, Sypabekova M, Karatayeva N.** 2013. Role of viruses in the development of breast cancer. *Infect Agent Cancer*. 8: 32.
- Cummins J, Tangney M.** 2013. Bacteria and tumours: causative agents or opportunistic inhabitants? *Cummins and Tangney Infectious Agents and Cancer*. 8: 11.
- Wang ZK, Yang YS.** 2013. Upper gastrointestinal microbiota and digestive diseases. *World J Gastroenterol*. 19: 1541–1550.
- de Martel C, Ferlay J, Franceschi S, Vignat J, Bray F, Forman D, et al.** 2012. Global burden of cancers attributable to infections in 2008: a review and synthetic analysis. *Lancet Oncol*. 13: 607–615.
- Mager DL.** 2006. Bacteria and cancer: cause, coincidence or cure? *A review. J Translational Med*. 4: 14.
- Cheng HC, Wang JD, Chen WY, Chen CW, Chang SC, Sheu BS.** 2015. Helicobacter pylori test–and–treat program can be cost–effective to prevent gastric cancer in Taiwanese adults: referred to the nationwide reimbursement database. *Helicobacter*. 20: 114–124.
- de Korwin JD.** 2014. Epidemiology of Helicobacter pylori infection and gastric cancer (in French). *Rev Prat*. 64: 189–193.
- Biarc J, Nguyen IS, Pini A, Gosse F, Richert S, Thierse D, et al.** 2004. Carcinogenic properties of proteins with pro–inflammatory activity from *Streptococcus infantarius* (formerly *S. bovis*). *Carcinogenesis*. 25: 1477–1484.
- Gold JS, Bayar S, Salem RR.** 2004. Association of *Streptococcus bovis* bacteremia with colonic neoplasia and extracolonic malignancy. *Arch Surg*. 139: 760–765.
- Montalban C, Santon A, Boixeda D, Bellas C.** 2001. Regression of gastric high grade mucosa associated lymphoid tissue (MALT) lymphoma after *Helicobacter pylori* eradication. *Gut*. 49: 584–587.
- Dutta U, Garg PK, Kumar R, Tandon RK.** 2000. Typhoid carriers among patients with gallstones are at increased risk for carcinoma of the gallbladder. *Am J Gastroenterol*. 95: 784–787.
- Ellmerich S, Scholler M, Duranton B, Gosse F, Galluser M, Klein JP, et al.** 2000. Promotion of intestinal carcinogenesis by *Streptococcus bovis*. *Carcinogenesis*. 21: 753–756.
- Kuper H, Adami HO, Trichopoulos D.** 2000. Infections as a major preventable cause of human cancer. *J Intern Med*. 248: 171–83.
- Schoppmann SF, Birner P, Stockl J, Kalt R, Ullrich R, Caucig C, et al.** 2002. Tumor–associated macrophages express lymphatic endothelial growth factors and are related to peritumoral lymphangiogenesis. *Am J Pathol*. 161: 947–956.
- Lara–Tejero M, Galán JE.** 2000. A bacterial toxin that controls cell cycle progression as a deoxyribonuclease I–like protein. *Science*. 290: 354–357.
- Sheng H, Shao J, Washington MK, DuBois RN.** 2001. Prostaglandin E2 increases growth and motility of colorectal carcinoma cells. *J Biol Chem*. 276: 18075–18081.
- Baik SC, Youn HS, Chung MH, Lee WK, Cho MJ, Ko GH, et al.** 1996. Increased oxidative DNA damage in Helicobacter pylori infected human gastric mucosa. *Cancer Res*. 56: 1279–1282.
- Carlo M, Croce MD.** 2008. Oncogenes and cancer. *N Engl J Med*. 358: 502–511.
- Carrillo–Infante C, Abbadessa G, Bagella L, Giordano A.** 2007. Viral infections as a cause of cancer (review). *Int J Oncol*. 30 (6): 1521–8.
- Chen H, Chen XZ, Waterboer T, Castro FA, Brenner H.** 2015. Viral infections and colorectal cancer: A systematic review of epidemiological studies. *Int J Cancer*. 137 (1): 12–24.
- Ruiz AJ, Russell SJ.** 2015. MicroRNAs and oncolytic viruses. *Curr Opin Virol*. 13: 40–48.
- Mesri EA, Feitelson MA, Munger K.** 2014. Human viral oncogenesis: a cancer hallmarks analysis. *Cell Host Microbe*. 15 (3): 266–82.
- Morales–Sánchez A, Fuentes–Pananá EM.** 2014. Human viruses and cancer. *Viruses*. 6 (10): 4047–79.
- World Health Organization.** 2009. Vaccine–preventable diseases: monitoring system 2009 global summary. WHO/UNICEF coverage estimates for 1980–2008, as of August 2009. Available at: http://www.who.int/immunization_monitoring/routine/immunization_coverage/en/index4.html. Accessed May 01, 2010.
- Burke AP, Yen TSB, Shekitka KM, Sobin LH.** 1990. Lymphoepithelial carcinoma of the stomach with Epstein–Barr virus demonstrated by polymerase chain reaction. *Mod Pathol*. 3: 377–80.
- Bonnet M, Guinebretiere JM, Kremmer E, Grunewald V, Benhamou E, Contesso G, et al.** 1990. Detection of Epstein–Barr virus in invasive breast cancers. *J Natl Cancer Inst*. 91: 1376–81.

57. **Labrecque LG, Barnes DM, Fentiman IS, Griffin BE.** 1995. Epstein Barr virus in epithelial cell tumors: a breast cancer study. *Cancer Res.* 55: 39–45.
58. **Sugawara Y, Mizugaki Y, Uchida T, Torii T, Imai S, Makuuchi M, et al.** 1999. Detection of Epstein–Barr virus (EBV) in hepatocellular carcinoma tissue: a novel EBV latency characterized by the absence of EBV-associated small RNA expression. *Virology.* 256: 196–202.
59. **Petrick JL, Wyss AB, Butler AM, Cummings C, Sun X, Poole C, et al.** 2014. Prevalence of human papilloma virus among oesophageal squamous cell carcinoma cases: systematic review and meta-analysis. *Br J Cancer.* 110: 2369–2377.
60. **Liyanage SS, Rahman B, Ridha I, Newall AT, Tabrizi SN, Garland SM, et al.** 2013. The aetiological role of human papilloma virus in oesophageal squamous cell carcinoma: a meta-analysis. *PLoS One.* 8: e69238.
61. **Yong F, Xudong N, Lijie T.** 2013. Human papillomavirus types 16 and 18 in esophagus squamous cell carcinoma: a meta-analysis. *Ann Epidemiol.* 23: 726–734.
62. **Carbone A, Gloghini A, Dotti G.** 2008. EBV-associated lymph proliferative disorders: classification and treatment. *Oncologist.* 13: 577e85.
63. **Gillison ML, Koch WM, Capone RB.** 2000. Evidence for a causal association between human papillomavirus and a subset of head and neck cancers. *Journal of the National Cancer Institute.* 92 (9): 709–720.
64. **Boshart M, Gissmann L, Ikenberg H, Kleinheinz A, Scheurlen W, zur Hausen H.** 1984. A new type of papillomavirus DNA, its presence in genital cancer biopsies and in cell lines derived from cervical cancer. *The EMBO J.* 3 (5): 1151–1157.
65. **Dürst M, Gissmann L, Ikenberg H, zur Hausen H.** 1983. A papillomavirus DNA from a cervical carcinoma and its prevalence in cancer biopsy samples from different geographic regions. *PNAS USA.* 80 (12 I): 3812–3815.
66. **Guillaud M, Buys TP, Carraro A, Korbek J, Follen M, Scheurer M et al.** 2014. Evaluation of HPV infection and smoking status impacts on cell proliferation in epithelial layers of cervical neoplasia. *PLoS One* 9(9):e107088.
67. **Garavello W, Bertuccio P, Levi F, Lucchini F, Bosetti C, Malvezzi M, et al.** 2010. The oral cancer epidemic in central and eastern Europe. *Int J Cancer.* 127: 160–171.
68. **Wen CP, Tsai MK, Chung WS, Hsu HL, Chang YC, Chan HT.** 2010. Cancer risks from betel quid chewing beyond oral cancer: a multiple-site carcinogen when acting with smoking. *Cancer Causes Control.* 21: 1427–1435.
69. **D'Souza G, Agrawal Y, Halpern J, Bodison S, Gillison ML.** 2009. Oral sexual behaviors associated with prevalent oral human papillomavirus infection. *J Infect Dis.* 199: 1263–1269.
70. **Jayalekshmi PA, Gangadharan P, Akiba S, Nair RR, Tsuji M, Rajan B.** 2009. Tobacco chewing and female oral cavity cancer risk in Karunagappally cohort, India. *Br J Cancer.* 100: 848–852.
71. **Sherris J, Wittet S, Kleine A, Sellors J, Luciani S, Sankaranarayanan R, et al.** 2009. Evidence-based, alternative cervical cancer screening approaches in low-resource settings. *Int Perspect Sex Reprod Health.* 35: 147–154.
72. **DeLancey JO, Thun MJ, Jemal A, Ward EM.** 2008. Recent trends in Black–White disparities in cancer mortality. *Cancer Epidemiol Biomarkers Prev.* 17: 2908–2912.
73. **Hashibe M, Brennan P, Chuang SC, Boccia S, Castellsague X, Chen C, et al.** 2009. Interaction between tobacco and alcohol use and the risk of head and neck cancer: pooled analysis in the International Head and Neck Cancer Epidemiology Consortium. *Cancer Epidemiol Biomarkers Prev.* 18: 541–550.
74. **Demontis MA, Sadiq MT, Goltz S, Taylor GP.** 2015. HTLV-1 viral RNA is detected rarely in plasma of HTLV-1 infected subjects. *J Med Virol.* DOI: 10.1002/jmv.24264
75. **Gessain A, Cassar O.** 2012. Epidemiological aspects and world distribution of HTLV-1 infection. *Front Microbiol.* 3: 388.
76. **Lymphoma Research Foundation.** 2008. Adult T-Cell leukemia/lymphoma (HTLV-1). Lymphoma Research Foundation, New York.
77. **National Institute of Cancer Research and Hospital Bangladesh.** 2014. National Institute of Cancer Research and Hospital Bangladesh Report 2008–2010. NICRH, Dhaka, Bangladesh.
78. **Soliven K, Wang X, Small CT, Feeroz MM, Lee EG, Craig KL, et al.** 2013. Simian foamy virus infection of rhesus macaques in Bangladesh: relationship of latent proviruses and transcriptionally active viruses. *J Virol.* 87 (24): 13628–39.
79. **Hossain MA, Raton KA, Noor R.** 2013. Bacteriological study of stool samples collected from children suffering from diarrhoea. *J Global Biosci.* 2: 160e5.
80. **Sestak I, Cuzick J.** 2015. Update on breast cancer risk prediction and prevention. *Curr Opin Obstet Gynecol.* 27 (1): 92–7.
81. **Yang H, Zhan L, Yang T, Wang L, Li C, Zhao J, et al.** 2015. Ski prevents TGF- β -induced EMT and cell invasion by repressing SMAD-dependent signaling in non-small cell lung cancer. *Oncol Rep.* DOI: 10.3892/or.2015.3961
82. **Hussain, SMA.** 2013. Comprehensive Update on Cancer Scenario of Bangladesh. *South Asian J Cancer.* 2 (4): 279–284.
83. **Kundu SS, Noor N, Jalali MA, Ramiz MS, Haque S, Parveen F.** 2014. Synovial sarcoma of neck mimicking thyroid carcinoma. *Mymensingh Med J.* 23 (1): 170–2.
84. **Mostafa MG, Dalquen P, Kunze D, Terracciano L.** 2014. Telecytological diagnosis of space-occupying lesions of the liver. *Acta Cytol.* 58 (2): 174–81.
85. **Iqbal MS, Hossain MS, Niessen LW.** 2014. Breast cancer in low-resource settings: prioritizing the awareness and access programmes to overcome socio-cultural and economic barriers for early detection and improved outcomes. *Eur J Cancer Prev.* 23 (4): 286–7.
86. **Rahman MM, Bhuiyan MH, Mahmud R, Nuton MA.** 2014. A giant hepatic cyst. *Mymensingh Med J.* 23 (1): 160–2.
87. **Nessa A, Nahar KN, Begum SA, Anwary SA, Hossain F, Nahar K.** 2013. Comparison between visual inspection of cervix and cytology based screening procedures in Bangladesh. *Asian Pac J Cancer Prev.* 14 (12): 7607–11.
88. **Rahman T, Tabassum S, Jahan M, Nessa A, Ashrafunnessa.** 2013. Detection and estimation of human papillomavirus viral load in patients with cervical lesions. *Bangladesh Med Res Counc Bull.* 39 (2): 86–90.
89. **Showkat MS, Nabi S, Khondker L, Bhowmik B, Tushar SN, Jahan MU.** 2013. Role of transvaginal sonography in the detection of endometrial carcinoma. *Bangladesh Med Res Counc Bull.* 39 (2): 80–5.
90. **Islam A, Eden T.** 2013. Brief report on pediatric oncology in Bangladesh. *South Asian J Cancer.* 2: 105–6.
91. **Murthy NS, Chaudhry K, Rath GK.** 2008. Burden of cancer and projections for 2016, Indian scenario: gaps in the availability of radiotherapy treatment facilities. *Asian Pac J Cancer Prev.* 9 (4): 671–7.
92. **Faruk T, Islam MK, Arefin S, Haq MZ.** 2015. The Journey of Elastography: Background, Current Status, and Future Possibilities in Breast Cancer Diagnosis. *Clin Breast Cancer.* DOI: 10.1016/j.clbc.2015.01.002
93. **Uddin AK, Khan ZJ, Islam J, Mahmud AM.** 2013. Cancer care scenario in Bangladesh. *South Asian J Cancer.* 2: 102–4.
94. **Noronha V, Tsomo U, Jamshed A, Hai MA, Wategama S, Baral RP, et al.** 2012. A fresh look at oncology facts on south central Asia and SAARC countries. *South Asian J Cancer.* 1: 1–4.
95. **Alam SMS, Kalam MA, Munna MS, Munshi SK, Noor R.** 2014. Isolation of pathogenic microorganisms from burn patients admitted in Dhaka Medical College and Hospital and demonstration of their drug-resistance traits. *Asian Pac J Trop Dis.* 4: 402e7.
96. **Aurin TH, Munshi SK, Kamal SM, Rahman MM, Hossain MS, Marma T, et al.** 2014. Molecular approaches for detection of the multi-drug resistant tuberculosis (MDR-TB) in Bangladesh. *PLoS One.* 9: e99810.
97. **Noor R, Morsalin M, Chakraborty B.** 2014. Reduction of CD4 count induces opportunistic infections in people living with HIV (PLHIV). *Bangladesh J Med Sci.* 13: 285e91.
98. **Noor R, Akhter S, Rahman F, Munshi SK, Kamal SM, Feroz F.** 2013. Frequency of extensively drug resistant tuberculosis (XDR-TB) among re-treatment cases in NIDCH, Dhaka, Bangladesh. *J Infect Chemother.* 19: 243e8.
99. **Noor R, Hossain A, Munshi SK, Rahman F, Kamal SM.** 2013. Slide drug susceptibility test for the detection of multi-drug resistant tuberculosis in Bangladesh. *J Infect Chemother.* 19: 818e24.
100. **Uddin AI, Pervin M, Munna MS, Noor R.** 2014. Study of risk factors related to HBsAg reactivity among outdoor patients in Dhaka Medical College and Hospital, Bangladesh. *Am J Biomed Life Sci.* 2: 18e21.
101. **Hasan M, Munshi SK, Momi MSB, Rahman F, Noor R.** 2013. Evaluation of the effectiveness of BACTEC MGIT 960 for detection of mycobacteria in Bangladesh. *Int J Mycobacteriol.* 2: 214e9.
102. **Hossain MS, Iqbal MS, Khan MA, Rabbani MG, Khatun H, Munira S, et al.** 2014. Diagnosed hematological malignancies in Bangladesh – a retrospective analysis of over 5000 cases from 10 specialized hospitals. *BMC Cancer.* 14: 438.
103. **Khan SA, Feroz F, Noor R.** 2013. Study of extended spectrum β -lactamase producing bacteria from urinary tract infection in Dhaka city. *Bangladesh. Tzu Chi Med J.* 25: 39e42.
104. **Raton KA, Hossain MA, Noor R.** 2013. Multiplex real-time PCR assay for detection of respiratory pathogens among pneumonia affected children. *Am J Biomed Life Sci.* 1: 53e7.
105. **Munshi SK, Rahman F, Kamal SMM, Noor R.** 2012. Comparison among different diagnostic methods used for the detection of extra-pulmonary tuberculosis in Bangladesh. *Int J Mycobacteriol.* 1: 190e5.
106. **Noor R, Shaha SR, Rahman F, Munshi SK, Rahman MM, Uddin MA.** 2012. Frequency of opportunistic and other intestinal parasitic infections

- among the HIV infected patients in Bangladesh. *Tzu Chi Med J.* 24: 191e5.
107. **Chakraborty B, Bashar T, Roy K, Noor R, Rahman MM.** 2011. Persistence of anti-HBs antibody and immunological memory in healthy individuals vaccinated with hepatitis B vaccine. *Stamford J Microbiol.* 1: 37e41.
108. **Jahan F, Elahi R, Mohiuddin MK, Khan MGM, Alam MS, Noor R.** 2011. Evaluation of two new rapid diagnostic tests (RDTs) for the diagnosis of malaria. *Bangladesh. J Med Microbiol.* 5: 11e5.