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Review Article

Impact of Environment and Lifestyle on Gynaecological Cancer Risk

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Abstract: The risk of gynaecological cancer (GC) is nonlinearly correlated with women's exposure to air pollutants ($PM_{2.5}$, CO, O_3, and SO_2). Residents in municipalities with greater levels of $PM_{2.5}$, a proxy measure of polycyclic aromatic hydrocarbons (PAHs) and NO_2, had a higher risk of dying from ovarian cancer. The prevalence of pulp and paper manufacture was found to be highly linked with the incidence rates of ovarian cancer. Ovarian cancer risk factors include physical inactivity, obesity, talc-containing body powder used in the vaginal region, silica dust exposure, dry cleaning, telegraph and telephone work, and graphic and printing work. Women who had previously smoked cigarettes were more likely to develop cervical cancer as well as invasive and borderline mucinous ovarian tumours. Cervical dysplasia was more common in women in the highest residential benzene and diesel particulate matter (DPM) exposure categories and with high levels of home exposure to PAHs. It was thought that exposure to PAHs influenced how cervical cancer developed and would strengthen the impact of high-risk human papillomavirus (HR-HPV) on cervical dysplasia. HPV infection and cervical, vaginal, and vulvar cancer have been linked in numerous studies. These comprised 62.5% of all cancer cases in the current analysis, and 76% of cervical cancer cases were found among low-income populations. Consumption of soybean milk, dietary additives, sweeteners, and preserved foods, as exposure to plastics, cosmetics, and other pollutants like $PM_{2.5}$, could all be risk factors for uterine leiomyoma. Endometrial cancer risk factors include obesity, diabetes, and hypertension. Due to civilization, industry, and urbanization, humans are continually exposed to endocrine-disrupting substances (EDS), which promote the growth of endometrial cancer.
Keywords: Gynaecological cancers, Pollutants, PAH, HPV.

Introduction

One of the most important public health concerns and a global priority is women's health. It has been hypothesised that air pollution exposure is linked to a higher risk of illnesses affecting women's health [1]. However, it is still unclear how much alterations in ambient air pollution have an impact on gynaecological cancer. Gynaecological illnesses are a concern to the health of women all over the world, with more than 100,000 women dying each year [2, 3]. Gynaecological tumours are one of the most common causes of disease worldwide [4, 5], particularly in developing and undeveloped areas [6]. Numerous studies have established a link between ambient air pollution and gynaecological cancers such as ovarian cancer [7], cervical cancer [8], and uterine fibroid [9]. Much research [7, 8] was only carried out in developed nations with good air quality, making it challenging to draw conclusions about the association between the risk of gynaecological cancer and air pollution in developing nations due to severe air pollution. Further analysis of the link and mechanism must be done utilising accurate exposure estimates for various exposure levels and trustworthy data. Two types of data are necessary to ensure the validity and reliability of the study: clinical data comprising specific patient information and tightly controlled government statistics on air pollution.

The National Institutes of Health (NIH) recently confirmed that the Human Papillomavirus (HPV), which is present in more than 90% of cases of cervical cancer, is the main cause of cervical cancer [10]. The majority of vaginal malignancies have an HPV infection as a cause. Age-related changes, multiple sexual partners, early sexual activity, smoking, alcohol use, cervical cancer, and HIV infection are all risk factors for vaginal cancer. Ovarian cancer is also a risk due to occupational exposure [7]. Advanced age, hereditary gene mutations such as BRCA1 and BRCA2, obesity, postmenopausal hormone replacement therapy, endometriosis, lengthier menstrual cycles, and never having been pregnant are all risk factors for ovarian cancer [11].

Changes in the endometrium result from variations in the oestrogen-to-progesterone ratio. Endometrial cancer risk factors include obesity, diabetes, polycystic ovarian syndrome, and any illness or condition that raises body oestrogen levels but not progesterone levels. Menstruation that lasts longer, starts earlier (before age 12), or enters menopause later all increase the risk of endometrial cancer when estrogen-only medications are used beyond menopause. Endometrial cancer risk factors include age, breast cancer hormone therapy, never having children, and inherited factors [12]. The data regarding how lifestyle and environmental factors affect the risk of gynaecological cancer are summarised in this chapter.

Effects of pollutants

Women's exposure to air pollution ($PM_{2.5}$, CO, O_3, and SO_2) is nonlinearly associated with their likelihood of developing gynaecological cancer (GC). The solitary impact of each air pollutant on gynaecological cancer was calculated with and without accounting for other air pollutants. After controlling for other pollutants, it was discovered that the relationships between each pollutant and gynaecological cancer were very similar to those that existed before controlling for other pollutants, indicating that the influences of individual factors on gynaecological cancer were largely independent [1].

Similar to how smoking habits increase the risk of gynaecological tumours [8], prolonged exposure to hazardous environments dominated by air pollutants can cause cervical cancer cells to undergo an oxidative stress reaction, which damages DNA and causes symptoms that are similar to those of an HPV infection [13]. The relationship between the Polycyclic Aromatic Hydrocarbons (PAHs) and ovarian malignancies [14], cervical epithelial tumours [15], and uterine dysplasia [8] have been supported by significant investigations. Elderly individuals' immune systems were generally poorer and may have various underlying disorders [16, 17]. As a result, even when exposed to the same level of air pollution as young patients, the elderly are likely to be at an increased risk for gynaecological cancers. Different sociodemographic circumstances may significantly affect how patients are exposed to air pollution, which in turn affects how likely they are to develop gynaecological cancer. The elderly (>65 years old) and blue-collar patients were more likely than their counterparts to develop gynaecological cancer when exposed to air pollution [1]. Households with higher incomes are less likely to experience harmful health effects from exposure to hazardous air pollution [18, 19].

GC was strongly linked with SO_2 exposure. As a result, it was determined that long-term exposure to high concentrations of the industrial pollutant SO_2 is linked to the emergence of GC [20]. Exposure to air pollution can have detrimental effects on one's health and may contribute to the development of ovarian cancer. It was determined that long-term exposure to air pollution by conceivable pathways, estrogen-like effects, and genetic alterations may have an impact on the development of ovarian tumours [21]. Independent of socio-demographic and therapeutic determinants, increased exposure to NO_2 and $PM_{2.5}$ may have a negative influence on ovarian cancer-specific survival [22]. When compared to people who lived in areas with the lowest levels of $PM_{2.5}$, a proxy measure of PAH, those who lived in areas with the highest levels of $PM_{2.5}$ had a greater chance of dying from ovarian cancer [14]. Compared to the lowest exposure category, women in the highest household benzene and diesel particulate matter (DPM) exposure categories showed a higher prevalence of cervical

dysplasia. Similar to this, cervical dysplasia was more common in women who had high levels of residential exposure to PAHs. Although not statistically significant, the highest PAH exposure category showed a favourable correlation with the occurrence of cervical dysplasia. Exposure to high levels of several hazardous air pollutants (HAPs) is positively correlated with the prevalence of cervical dysplasia, according to an assessment of the cumulative effect of HAP exposure [8]. There is evidence that hormone action is impacted by air pollution, notably from automobile exhaust. The occurrence of uterine leiomyomata, a uterine tumour that is hormonally sensitive, is not known to be correlated with exposure to air pollution. An increase in the incidence of uterine leiomyomata from chronic exposure to $PM_{2.5}$ may be slight [9]. Due to the industrial revolution and the adoption of modern lifestyles, a significant amount of synthetic chemicals have been introduced into the environment over the previous three to four decades. Some have the potential to be teratogenic, mutagenic, or carcinogenic, making them potential reproductive toxicants [23].

Humans are regularly exposed to endocrine disruptive substances (EDCs) because of civilization, industry, and urbanisation. The most prevalent malignant neoplasms in women are endometrial cancer and breast cancer, both of which can be partially induced by oestrogen. An increased risk of their occurrence may result from prolonged exposure to estrogens or drugs with estrogenic characteristics. In our surroundings, hundreds of chemicals have been found to have the ability to alter hormones. More and more data are emerging that connect EDC exposure to endometrial and mammary cancer development [24]. Heavy metal and known carcinogen, cadmium is a contaminant in both the environment and the workplace. It can imitate the proliferative effects of oestrogen and is categorised as a metal oestrogen because it is a known endocrine disruptor. The effects of cadmium have not been thoroughly investigated, despite the fact that the proliferative effect of oestrogen on the growth of malignant cells is widely known [25].

Reproductive health is negatively impacted by an increase in industrialization and the resulting environmental pollution, an increase in the use of synthetic chemicals, and recurrent exposure to harmful substances at work and at home. Pulp and paper mills are linked to exposure to substances that are known to cause ovarian cancer [26]. Incidence rates of ovarian cancer at the state level were previously found to be highly associated with the volume of pulp and paper production. Data from the Environmental Protection Agency's Harmful Release Inventory was used to specifically study the association between spatial patterns of ovarian cancer incidence rates and toxic emissions from pulp and paper operations. South-Central Iowa, Wisconsin, New York, Pennsylvania, Alabama, and Georgia all have groups of counties with high ovarian cancer incidence rates, according to geospatial research. Counties with high ovarian cancer rates were linked to counties with lots of pulp and paper industries, according to a bivariate local indicator of spatial autocorrelation (LISA) analysis. Ovarian cancer incidence and emissions of water pollutants are positively correlated, according to regression analysis of state-level data. The examination of county-level data led to the discovery of a comparable link [27]. The risk of ovarian cancer was shown to be higher in Norwegian pulp and paper workers who may have been exposed to asbestos at work [28]. Asbestos, which is a known carcinogen for the human ovary, is the only substance thought to be sufficiently evidence-based to cause cancer in the female reproductive system in humans [29].

Effects of lifestyle

The aetiology of gynaecological cancers is recognised to be significantly influenced by a number of lifestyle factors; however, only a small number of published research have looked into potential links with occupational factors. Some occupational exposures have been linked to higher risks of these diseases, although the data is very scant, with the exception of what is known about the links between Tetrachloroethylene and cervical cancer and asbestos fibres and ovarian cancer [30]. Using the Canadian Standard Occupational Classification (SOC) and Standard Industrial Classification (SIC), jobs and industries were coded. After taking into account potential confounders, it was discovered that teaching vocations had a heightened epithelial ovarian cancer risk, making it the first to make this observation [31]. Often, occupational exposures are significantly higher than ambient

exposures. The evidence points to the possibility that lifestyle factors (such as diet and sexual practises) and exposure to some organochlorine compounds, ionising radiation, electromagnetic radiation, etc. may have the ability to cause cancer of the reproductive organs, although there is little evidence to support this claim [32].

Dry cleaning, telegraph and telephone work, paper packing, and design and printing work are among the occupations with higher ovarian cancer risks. It was discovered that hairdressers and beauticians did not have an elevated risk of ovarian cancer, in contrast to the findings of some earlier research. It is suggested that specific etiologic agents such as organic dust, aromatic amines, aliphatic hydrocarbons, and aromatic hydrocarbons may be involved [7]. A lower chance of developing ovarian cancer was linked to ever working in the production of cotton. It was discovered that cumulative exposure to silica dust was linked to an elevated risk of ovarian cancer and was associated with ever having worked in textile finishing. Cotton dust and endotoxin may lower the risk while silica dust may raise the risk of ovarian cancer [32].

Although the confidence interval was large and no indication of a trend could be found, women exposed to talc had a relative risk of ovarian cancer below the null. The relative risk for women exposed to polycyclic aromatic hydrocarbons was higher, with a broad confidence interval and no indication of a trend over time [33]. It has been suggested that smoking cigarettes are linked to some subtypes of ovarian cancer [34]. Smokers have an increased risk of developing cervical cancer due to exposure to cigarette smoke, which has been shown to enhance oxidative stress [13].

On the other hand, smoking is strongly linked to cervical cancer in the US population but not among Venezuelans [35]. Smoking contributes to the development of cervical cancer and raises the risk of ovarian cancer, especially mucinous tumours [36]. The progression of cervical intraepithelial neoplasia (CIN) and cervical cancer is mostly attributed to high-risk human papillomavirus (HR-HPV) infection. Exposure to PAHs could worsen the impact of HR-HPV on CIN and be linked to the development of cervical cancer [15].

In Taiwan, young women may be more likely to get cervical cancer as a result of their cooking habits, according to the findings of a study [37]. Consumption of soybean milk, dietary additives, sweeteners, and preserved foods, as well as exposure to plastics, cosmetics, and other pollutants, could all be risk factors for uterine leiomyoma [38]. A woman's risk of developing gynaecological cancer is influenced by a number of lifestyle choices, some of which may be changed to lower risk. In the industrialised world, obesity is becoming more common to the point where it now accounts for as many cancer deaths as smoking. Compared to women of normal weight, women with a body mass index (BMI)>40 had a 60% greater risk of dying from all malignancies. Additionally, they have a higher risk of developing gynaecological cancer. Gynaecological cancer risk is greatly influenced by diet: fruit, vegetables, and antioxidants lower risk, but high animal fat and energy intake raise the risk.

Results

The influence of environmental and lifestyle factors on gynaecological malignancies is difficult to distinguish. These two things go hand in hand, and exposure to them is also influenced by a variety of other factors, including socioeconomic position and line of work. It was discovered by looking at the socioeconomic status of all the cases that low-income people were more likely to develop cervical and vaginal cancer. In the middle-class and upper-middle-class categories, endometrial cancer was more prevalent in the present study. Comparing the parity with different gynaecological cancers, the nulliparous state is shown most commonly in ovarian cancer cases and least frequently in vulvar cancer cases. Diabetes mellitus, hypertension, and hypothyroidism were more common among endometrial cancer patients than those with other gynaecological cancers: diabetes mellitus in 22.64% of cases, hypertension in 38.99% of cases, and hypothyroidism in 10.98% of cases in the present study. Out of the total 5524 instances, 139 had one or more family members having cancer at

various sites. Since there were only 53 patients having addictions to things like smoking, chewing tobacco, alcohol, gutka, and zarda, they are not statistically significant.

Conclusions and Recommendations

Given the multifactorial nature of cancers of the female reproductive system, occupational studies that collect comprehensive data on potential individual confounding factors, particularly reproductive history and other factors that affect the body's hormonal environment, along with information on socioeconomic status and lifestyle factors, including physical activity from multiple sources, are crucial [39].

This study supports the idea that, in order to reduce the harmful consequences of air pollution, enhanced public health preventive measures and policymaking should take into account the gynaecologic risks connected with it. Positive results, however, encourage avoiding such exposure and quitting such lifestyle practices. Protecting people from exposure to environmental and industrial chemicals requires promoting their avoidance, substitution with less dangerous/non-toxic substances, and adoption of healthy lifestyles [23].

For women who have survived gynaecological cancer, the advantages of lifestyle changes are still unknown. The purpose of this systematic review was to ascertain the impact of lifestyle changes on cancer recurrence, overall survival, and quality of life (QoL) in GC-affected women. We included randomised controlled trials in which lifestyle interventions (diet, weight loss, physical activity, and/or behavioural interventions) were compared with a control condition (usual care, a placebo, or other lifestyle interventions) in women who had successfully battled endometrial or ovarian cancer [39].

Over the past few decades, it has been clear how important a healthy lifestyle is for GC prevention [40, 41]. Maintaining a healthy weight range through dietary and exercise changes may help lower the chance of recurrence. High body mass index (BMI) is linked to a poor prognosis for GC [42, 43]. Independent of BMI, physical activity can prevent ovarian, endometrial, and postmenopausal breast cancer. The oral contraceptive pill is one of the best instances of widespread chemoprevention in the industrialised world and has a significant and long-lasting impact on the prevention of ovarian and endometrial cancer. While it raises the risk of cervical cancer, pregnancy protects against ovarian, endometrial, and breast cancer [38]. Pre-diagnosis, a healthy diet (focused on fruits and vegetables or a low-fat diet), and routine exercise may increase the survival rates of ovarian cancer survivors, according to observational studies [44, 45, 46].

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References

1. Yu Q, Zhang L, Hou K, Li J, Liu S, Huang K, Cheng Y. Relationship between air pollutant exposure and gynecologic cancer risk. Int J Environ Res Pub Heal. 2021;18(10):5353.
2. International Agency for Research on Cancer GLOBOCAN 2008: Cancer Incidence and Mortality Worldwide in 2008. http://globocan.iarc.fr/
3. Adami HO, Hunter D, Trichopoulos D. Textbook of Cancer Epidemiology. Oxford University Press; New York City, NY, USA: 2009.
4. Parkin DM, Bray F, Ferlay J, Pisani P. Estimating the world cancer burden: Globocan 2000. Int J Cancer. 2001;94(2):153-6.

5. Sankaranarayanan R, Ferlay J. Worldwide burden of gynaecological cancer: the size of the problem. Best Pract Res Clin Obstet Gynaecol. 2006;20(2):207-25.
6. Kamangar F, Dores GM, Anderson WF. Patterns of cancer incidence, mortality, and prevalence across five continents: defining priorities to reduce cancer disparities in different geographic regions of the world. J Clin Oncol. 2006;24(14):2137-50.
7. Shields T, Gridley G, Moradi T, Adami J, Plato N, Dosemeci M. Occupational exposures and the risk of ovarian cancer in Sweden. Am J Ind Med. 2002;42(3):200-13.
8. Scheurer ME, Danysh HE, Follen M, Lupo PJ. Association of traffic-related hazardous air pollutants and cervical dysplasia in an urban multiethnic population: a cross-sectional study. Environ Health. 2014;13(1):1-8.
9. Mahalingaiah S, Hart JE, Laden F, Terry KL, Boynton-Jarrett R, Aschengrau A, Missmer SA. Adult air pollution exposure and risk of uterine leiomyoma in the nurses' health study II. Epidemiol. 2014;25(5):682.
10. Averette HE, Wrennick A, Angioli R. History of gynecologic oncology subspecialty. Surg Clin North Am. 2001;81(4):747-51.
11. Ovarian Cancer: Overview. https://www.mayoclinic.org/diseases-conditions/ovarian-cancer/symptoms-causes/syc-20375941.
12. Endometrial Cancer: Overview. https://www.mayoclinic.org/diseases-conditions/endometrial-cancer/symptoms-causes/syc-20352461.
13. Moktar A, Singh R, Vadhanam MV, Ravoori S, Lillard JW, Gairola CG, Gupta RC. Cigarette smoke condensate-induced oxidative DNA damage and its removal in human cervical cancer cells. Int J Oncol. 2011;39(4):941-7.
14. Hung LJ, Tsai SS, Chen PS, Yang YH, Liou SH, Wu TN, Yang CY. Traffic air pollution and risk of death from breast cancer in Taiwan: fine particulate matter (PM2. 5) as a proxy marker. Aerosol Air Qual Res. 2012;12(2):275-82.
15. Li X, Ding L, Song L, Gao W, Wang L, Wang J. Effects of exposure to polycyclic aromatic hydrocarbons combined with high- risk human papillomavirus infection on cervical intraepithelial neoplasia: A population study in Shanxi Province, China. Int J Cancer. 2020;146(9):2406-12.
16. Derhovanessian E, Solana R, Larbi A, Pawelec G. Immunity, ageing and cancer. Immun Age. 2008;5:1-6.
17. Kiecolt-Glaser JK, Glaser R. Stress and immunity: Age enhances the risks. Curr Direct Psychol Sci. 2001;10(1):18-21.
18. Forastiere F, Stafoggia M, Tasco C, Picciotto S, Agabiti N, Cesaroni G, Perucci CA. Socioeconomic status, particulate air pollution, and daily mortality: differential exposure or differential susceptibility. Am J Ind Med. 2007;50(3):208-16.
19. Peterson CE, Rauscher GH, Johnson TP, Kirschner CV, Barrett RE, Kim S, Fitzgibbon ML, Joslin CE, Davis FG. The association between neighborhood socioeconomic status and ovarian cancer tumor characteristics. Cancer Causes Cont. 2014;25:633-7.
20. Xu Q, Lu C, Murithi RG, Cao L. Increase associated risk of gynaecological cancer due to long-term exposure to high concentration of atmospheric SO_2 industrial pollutant. Indoor Built Environ. 2022;31(8):2183-92.
21. Dehghani S, Moshfeghinia R, Ramezani M, Vali M, Oskoei V, Amiri-Ardekani E, Hopke P. Exposure to air pollution and risk of ovarian cancer: a review. Rev Environ Health. 2022 May 17.

22. Villanueva C, Chang J, Ziogas A, Bristow RE, Vieira VM. Ambient air pollution and ovarian cancer survival in California. Gynecol Oncol. 2021;163(1):155-61.

23. Shankar H, Saluja S, Rawat M, Singh G, Tarique M, Mohammad N, Naz H. The causal association between occupational, environmental, and lifestyle factors and reproductive cancer risk. Curr Mol Biol Rep. 2020;6:149-60.

24. Rutkowska AZ, Szybiak A, Serkies K, Rachon D. Endocrine disrupting chemicals as potential risk factor for estrogen-dependent cancers. Pol Arch Med Wewn. 2016;126(7-8):562-70.

25. Todd H. *The effect of cadmium on ovarian adenocarcinoma cell lines: an investigation of the possible mechanism of action*;2021. Undergraduate Theses 62, https://scholarworks.bellarmine.edu/ugrad_theses/62

26. Schwartz GG, Sahmoun AE. Ovarian cancer incidence in the United States in relation to manufacturing industry. Int J Gynecol Cancer. 2014;24(2):247-51.

27. Hanchette C, Zhang CH, Schwartz GG. Ovarian cancer incidence in the US and toxic emissions from pulp and paper plants: a geospatial analysis. Int J Environ Res Pub Health. 2018;15(8):1619.

28. Langseth H, Johansen BV, Nesland JM, Kjaerheim K. Asbestos fibers in ovarian tissue from Norwegian pulp and paper workers. Int J Gynecol Cancer. 2007;17(1).

29. Weiderpass E, Hashim D, Labrèche F. Malignant tumors of the female reproductive system. Occupat Cancers. 2020:439-53.

30. Weiderpass E, Labrèche F. Malignant tumors of the female reproductive system. Saf Health Work. 2012;3(3):166-80.

31. Le ND, Leung A, Brooks-Wilson A, Gallagher RP, Swenerton KD, Demers PA, Cook LS. Occupational exposure and ovarian cancer risk. Cancer Causes Cont. 2014;25:829-41.

32. Wernli KJ, Ray RM, Gao DL, Fitzgibbons ED, Camp JE, Astrakianakis G, Seixas N, Wong EY, Li W, De Roos AJ, Feng Z. Occupational exposures and ovarian cancer in textile workers. Epidemiol. 2008:244-50.

33. Hartge P, Stewart P. Occupation and ovarian cancer: a case-control study in the Washington, DC, metropolitan area, 1978-1981. J Occupat Med. 1994:924-7.

34. Sumanasekera W, Beckmann T, Fuller L, Castle M, Huff M. Epidemiology of ovarian cancer: Risk factors and prevention. Biomed J. 2018;1:13.

35. Au WW. Life style, environmental and genetic susceptibility to cervical cancer. Toxicol. 2004;198(1-3):117-20.

36. Rieck G, Fiander A. The effect of lifestyle factors on gynaecological cancer. Best Pract Res Clin Obstet Gynaecol. 2006;20(2):227-51.

37. Chen TC, Lee JY, Wang SY, Chang CL, Yang YC. Relevant factors for cervical cancer among young women in Taiwan. Taiwan J Obstet Gynecol. 2005;44(2):143-7.

38. Shen Y, Xu Q, Xu J, Ren ML, Cai YL. Environmental exposure and risk of uterine leiomyoma: an epidemiologic survey. Eur Rev Med Pharmacol Sci. 2013;17(23):3249-56.

39. Yeganeh L, Harrison C, Vincent AJ, Teede H, Boyle JA. Effects of lifestyle modification on cancer recurrence, overall survival and quality of life in gynaecological cancer survivors: A systematic review and meta-analysis. Maturitas. 2018;111:82-9.

40. Raj MH, Abd Elmageed ZY, Zhou J, Gaur RL, Nguyen L, Azam GA, Braley P, Rao PN, Fathi IM, Ouhtit A. Synergistic action of dietary phyto-antioxidants on survival and proliferation of ovarian cancer cells. Gynecol Oncol. 2008;110(3):432-8.

41. Paxton RJ, Garcia-Prieto C, Berglund M, Hernandez M, Hajek RA, Handy B, Brown J, Jones LA. A randomized parallel-group dietary study for stages II–IV ovarian cancer survivors. Gynecol Oncol. 2012;124(3):410-6.

42. McCarroll ML, Armbruster S, Frasure HE, Gothard MD, Gil KM, Kavanagh MB, Waggoner S, Von Gruenigen VE. Self-efficacy, quality of life, and weight loss in overweight/obese endometrial cancer survivors (SUCCEED): a randomized controlled trial. Gynecol Oncol. 2014;132(2):397-402.

43. Lutgendorf SK, Shinn E, Carter J, Leighton S, Baggerly K, Guindani M, Fellman B, Matzo M, Slavich GM, Goodman MT, Tew W. Quality of life among long-term survivors of advanced stage ovarian cancer: A cross-sectional approach. Gynecol Oncol. 2017;146(1):101-8.

44. Zhou Y, Chlebowski R, LaMonte MJ, Bea JW, Qi L, Wallace R, Lavasani S, Walsh BW, Anderson G, Vitolins M, Sarto G. Body mass index, physical activity, and mortality in women diagnosed with ovarian cancer: results from the Women's Health Initiative. Gynecol Oncol. 2014;133(1):4-10.

45. Secord AA, Hasselblad V, Von Gruenigen VE, Gehrig PA, Modesitt SC, Bae-Jump V, Havrilesky LJ. Body mass index and mortality in endometrial cancer: a systematic review and meta-analysis. Gynecol Oncol. 2016;140(1):184-90.

46. Siegel RL, Miller KD, Jemal A. Cancer Statistics, 2017. CA Cancer J Clin. 2017;67(1):7-30.

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