



Original Article

Ovarian Cancer (OC) Risk among Millennials in India

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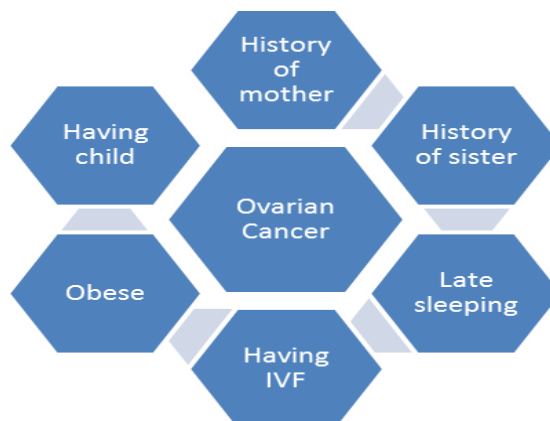
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ABSTRACT

The present study is an initiative to understand various related factors associated with ovarian cancer among millennials in India. The spark behind the present study after visiting various cancer patients in the locality motivated the author to work on the present study. In this regard, 12 hospitals approached and were able to collect some substantial data from the patients. Nine hypotheses were considered which were developed after the literature reviews. The results revealed that having children, having a mother with an ovarian cancer history, having a sister having ovarian cancer history, being obese, having late sleeping habits, and with IVF have a significant association with ovarian cancer and diabetics, smoking habits, and contraceptive pills do not have any direct association with ovarian cancer as opined by the participants

GRAPHICAL ABSTRACT



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Introduction

In many locales of the globe, cancer is the main source of death and presently the greatest hindrance to acquire an adequate life [1]. The third most incessant gynecologic cancer after uterine and cervical cancer is ovarian cancer. The cancer occurrence changes internationally, similar to that of numerous different cancers [2, 3]. The threat factors that cause ovarian cancer can be a fault for the epidemiological fluctuation of the illness in different geographic regions. The Millennial generation, on the note to demographer David Foot, consists of people born between 1980 and 1995. Foot and Stoffman (2000) frequently call the Millennials "Baby Boom Echo" because they are the Baby Boomers kids (1946–65). Some ideas contend that Millennials as a whole are influenced by historical events and experiences from the same era (cf. Gilleard, 2004). This concept of a "generation" is based on Mannheims sociology of generations thesis from 1952, which claims that individuals from the same generation have more characteristics than only their year of birth [4-6]. Even though scientists have used various birth year confines to classify millennials, in practice precise birth year boundaries are far less dominant than shared historical events and experiences accompanying social change. The authors review the historical events shaped their lives, in this chapter, we use the term "millennials" to be consistent with the literature. The factors affected millennials are their Boomer parents, such as rising divorce rates, more women entering the workforce, and quickening technological change, which have had a significant impact on Millennials as a generation. According to a certain study, younger patients enjoy the benefit of survival since they are bound to have the beginning phase, lower-grade sickness, and a generally safe threat. In any case, more young age is certainly not a dependable indicator of the improved survival. As per distributed various ions, age is a vital prognostic component, with more seasoned patients having more awful results than more young women [7, 8].

Objectives of the study

This study aimed to examine various factors that lead to ovarian cancer and contribute to the existing literature relating to ovarian cancer among millennials.

The rationality of the present study

The present study is needed to understand various factors that lead to ovarian cancer. In recent times, it is experienced due to changes in lifestyle, food habits, lack of physical exercise, etc. also play an important role in ovarian cancer. The study was done after a recent visit to various cancer hospitals for charity purposes and after interacting with some patients and medical staff of various hospitals under study, we felt that there is a need for an empirical study in this topic.

Scope of the study

The present study is restricted to 12 leading cancer hospitals in the Eastern part of India. It includes American Oncology Institute and Hemalata Cancer Institute, KIMS Hospital, Acharya Harihar Cancer Research Institute, Sum Ultimate Medicare, Kalinga Hospital, Sparsh Hospital, Care Hospital, Amri Hospital, Panda Cancer Hospital, Utkal Hospital, and Blue Wheel Hospital.

Literature review

Based on family history

Ovarian cancer, which accounts for about 2,25,000 new cases (3.7% of all female cancers) and more than 1,40,000 fatalities (4.2% of all female casualties) per year [9], is the most well-known cause of cancer death in the gynecological field. In India, 14,270 predicted deaths and 21,980 evaluated new cases were expected in 2014 [10, 11]. The high-level stage at the conclusion and awful visualization found in ovarian cancer patients is linked to a lack of productive early discovery apparatuses and a perilous design [12]. The least fortunate of all gynecologic operations malignancies, overall survival (operating system) has a five-year relative survival rate of 44% at all stages. More

than one-fifth of ovarian cancer cases have a hereditary component. Although the system of hereditary ovarian tumorigenesis is currently thought to have no fewer than 16 characteristics, certain alterations are still unknown and cannot be detected by unambiguous testing [13, 14]. Carcinoma and microbial susceptibility are two of the worlds most problematic public health issues [15]. A family history of ovarian cancer is a high-risk factor, with the relative risk predicted to be 2.0 to 4.0 for individuals who have a first-degree relation afflicted by the illness [16].

Based on In vitro fertilization (IVF)

In the US, it is estimated that there will be 14,030 ovarian cancer-related fatalities and around 22,240 episode instances of ovarian cancer in 2013. Most ovarian cancer patients are related to the provincial or far-off cancer when 5-year survival rates are 72% and 27%, individually. Ovarian cancer is a convoluted, multi-layered sickness that, unfortunately, is much of the time found at a late stage. With infertility affecting around 9% of the population, a rising number of couples are seeking reproductive therapy. IVF use has continuously increased, accounting for 1.5% of live births in the United States and adding to pregnancies caused by ovulation inducement and superovulation. As a result, there are worries about the long-term hazards of fertility drugs causing cancer, because, despite their various routes of action, all can promote repeated ovulation and alter steroidogenesis [17, 18]. To foster methodologies that help forestall or potentially lead to the prior discovery of the sickness, it is pressing to recognize women who are at an expanded threat of developing ovarian cancer as well as to distinguish early side effects related to the illness [19]. In industrialized countries, the utilization of fertility drugs has essentially developed and is anticipated to keep on rising in light of expanding and more women are postponing attempting to get pregnant until they are above the age of 35. Since the time that ovulation-initiating prescriptions were initially given, there has been stress over how long-haul utilization of them might have unfavorable outcomes, including an expanded threat of

ovarian cancer [20, 21]. Experiences in the association between the utilization of regenerative medications and the threat of ovarian cancer are featured in this outline of late findings [22, 23].

Based on smoking habits

The usage of tobacco in the studies conducted in 2009 shows that they are interlinked with the cause of developing cancer, but previously in many studies this was not understood and this was not the root cause for ovarian cancer, the mucinous ovarian cancer was included in the World Organization for Research on Cancer [24-27]. The research on cancer transmission focuses on acquiring data on womens smoking habits have found that there is a connection between smoking habits and ovarian cancer as ovarian cancer is further caused due to smoking habits in women [28]. Although the information on smoking-related hazards was given in about 33% of the 56 studies, just a few findings from 55 of the 56 investigations were disclosed [29]. The linkage between smoking and the chances of creating ovarian cancer, in general, was not discovered in the majority of the studies that were conducted. Not all, but there are very few, both distributed and unpublished by the Cooperative Gathering on Epidemiological Research on Ovarian tumors was introduced to collect and reconsider the epidemiological data which are based on the connections between the threat causes and also for the development of ovarian cancer [30]. To avoid emphasizing findings from a small number of studies that have widely disseminated their findings, this research sought information from any evaluations larger than a certain size that had amassed significant data regarding the association between ovarian cancer risk and womens smoking history, regardless of whether they had been published or not [31]. This group combined and reanalyzed information from 51 studies on the influence of smoking on ovarian cancer incidence from around 28,000 ovarian cancer patients [32]. These publications include virtually all epidemiological information on the subject. Even though it has previously been documented that

continued smoking is associated with ovarian cancer, we discovered that the increase in ovarian cancer was exclusively in marginally damaging growths rather than in really severe cancers [33-35]. When contrasted with research that utilized elective plans, the discoveries of case control concentrated on utilizing medical clinic controls were subjectively unique [36]. Since the discoveries of the review studies using medical clinic controls and the review concentrates on utilizing populace controls vary essentially, it is suspicious that these inconsistencies are the outcome of a specifically incorrect review detailing smoking [37]. Smoking is connected to various ailments that could require hospitalization. Therefore, it is possible that emergency clinic controls had higher normal smoking rates than women in the overall local area [38]. The connection between smoking and the threat of ovarian cancer would be diminished by this change, and it might try to be switched. Along these lines, we did exclude preliminaries with emergency clinic controls in the essential studies [39]. Subtleties of those reviews are in any case given to ensure that all the epidemiological data is distributed [40]. Smoking is linked to a lower chance of survival in various malignancies. Mucinous ovarian cancer accounts for 10% to 20% of all epithelial ovarian cancers, a histologic form that frequently implies a bad prognosis if the illness is progressed. In the 1980s, the percentage of smokers was considerably greater among women with mucinous ovarian cancer than those who had other histologic forms of ovarian cancer [41, 42].

Based on the genetic disorder

This study set off on a mission to give an exhaustive and current evaluation of the pathology, counteraction, visualization, and therapy of this specific sort of ovarian cancer containing the hereditary transformations [43]. Hereditary ovarian cancer has exceptional pathologic and subatomic scientific qualities [44]. Ovarian cancers with BRCA1 and BRCA2 transformations are most often high-grade serous adenocarcinomas [45]. As far as clinical studies show, innate ovarian cancer happens sooner than

irregular ovarian cancer [46]. Oral contraceptives might be a feasible procedure for chemoprevention. Patients with ovarian cancer that display BRCA1 and BRCA2 changes had expanded movement-free survival and in general survival, as per another study [47]. Certain uncommon genetic disorders, such as Lynch syndrome, are associated with a greatly elevated risk of ovarian cancer. Lynch syndrome is most commonly linked with MLH1, or MSH2 gene abnormalities, and accounts for 10 to 15% of hereditary ovarian malignancies [48, 49].

Based on delay in bearing a child

The combined information from the epidemiological study demonstrates that the number of ovulations a lady has throughout her conceptual life fundamentally corresponded with her possibility of creating ovarian epithelial carcinoma [50]. It has for quite some time been known that ovulation-smothering variables, including pregnancy or the utilization of hormonal contraceptives, bring down a lady's possibility of creating ovarian cancer [51]. Albeit urgent to appreciate the causal pathways promoting ovarian cancer and comprehension of the sub-atomic cycles at play is inadequate [52]. There have generally been two essential speculations on the reasons for ovarian cancer. As per the "relentless ovulation" hypothesis, the month-to-month pattern of epithelial harm and ensuing recuperation make the ideal circumstances for cancer improvement [53]. As per the "gonadotropin" idea, ovarian cancer is essentially welcomed by gonadotropin levels that are sufficiently high to invigorate estrogen creation and the multiplication of ovarian surface epithelial cells [54]. Having notable impacts on growth advancement as well as cancer-hindering exercises, the job of reproductive pills in the improvement of female regenerative cancer today has all the earmarks of being undeniably more muddled than recently envisioned [55]. As indicated by one hypothesis, the elevated degrees of circling progesterone during pregnancy make a small part of modified epithelial cells kick the bucket by apoptosis [56].

A significant number of these explorations incorporate institutional investigation with a restricted patient populace. Furthermore, most of the discoveries were from scholarly foundations and clinical preliminary information, which might have inclinations and results that do not precisely address everybody. As per prior research, just 3-17% of ovarian cancer patients were younger than 40. There have been clashing data on the prognostic significance old enough in female cancer. It has been laid out that breast cancer visualization is more regrettable the younger the patient [57]. The way that young women ordinarily have more resigned grade 1 cancer might be a figure of their initial show stage and generally ideal guess. More young age was an autonomous prescient variable for the expanded survival in our multivariable study, despite the way that lower grade and prior stage may to some extent make sense of the unrivaled survival of more young patients with the ovarian cancer [58-61].

Approximately 16 characteristics are thought to be involved in the genetic ovarian cancer process at this time, and many further alterations are yet unknown and untestable and cannot be located with particular testing. The discovery of a shift in ovarian cancer weakening characteristics is a fundamental step toward understanding the location and the board of these malignancies [62, 63].

According to a certain research, more young women can tolerate more severe chemotherapy regimens, which may represent the patients typical outcomes. If the controlled study from separate scholarly foundations were performed after treating for the specialists mastery, the extent of the activity and adjuvant chemotherapy, more youthful age remained a vital free predictor for improved survival [64].

The two investigations enjoy benefits, for example, a sizable example size and admittance to members far-reaching conceptual and clinical histories. Importantly, the specialists had the option to isolate the threat related to these qualities from the threat related to the utilization of regenerative medications because of the ability to separate and record factors associated with

the ovarian cancer. Like prior studies, subgroup investigation in the expectation preliminary revealed a measurably critical ascent in the threat of ovarian cancer among women who utilized fruitfulness drugs but kept on being nulligravid notwithstanding looking for clinical help for their barrenness [65]. The quantity of IVF cycles was demonstrated to be related to a slight expansion in the threat of ovarian cancer, it was found that IVF therapy was shown to be strongly associated with an increased risk of fringe ovarian malignancies [66].

Siristatidis *et al.* played out a meta-investigation of 9 partner studies, incorporating a sum of 109,969 women who went through the IVF therapy, of whom 76 were distinguished as having obtrusive ovarian cancer, to produce a bigger number of ovarian cancer cases. All included studies were at that point distributed; none of the fresher explorations referenced above were incorporated. Each study that was incorporated gave just four recorded studies of barren women treated with IVF with those not treated with IVF being made in their unique distribution, contrasted and everybody [67, 68].

Smoking is known to affect the ovaries because it causes menopause to develop earlier in smokers than in nonsmokers. This impact, however, does not suggest that smoking would alter the incidence of ovarian cancer or specific cancer subtypes. The findings support the hypothesis that different forms of ovarian cancer can arise in different cell types. Smokers are less likely to acquire endometrial cancer, and our result that intermittent smokers are less likely to develop endometrial growth supports the theory that endometrioid ovarian cancer originates in endometrial cells. For clear-cell growths, there is no comparable simple [69].

However, even though there is a substantial overabundance of mucinous cancers in smokers, this abundance has all the earmarks of being to some degree offset by a little decline in clear-cell and endometrioid growths. Smoking has a wide assortment of the adverse results that outcome in huge expansions in mortality from various causes. Accordingly, even in this enormous example, the general expansion in ovarian cancer

occurrence among smokers is unobtrusive and barely critical. Taking into account that generally 50% of the mucinous growths among smokers were fringe harmful, this study could not address survival. Smoking is most likely not going to make a big deal about a distinction regarding the ovarian cancer mortality [70, 71].

The "raised gonadotropin levels" hypothesis proposes that ovarian cancer improvement is altogether affected by openness to high groupings of flowing pituitary gonadotropins, which animate the ovarian surface epithelium and subsequently upgrade the threat of the harmful changes. Related to each is a lower chance of ovarian cancer this idea is upheld by the additional pregnancies and oral prophylactic utilization. As per these speculations, infertility raises the threat of ovarian cancer because barren women are more averse to bringing down their lifetime number of ovulations by being pregnant and nursing. Furthermore, they could invest more energy attempting to get pregnant and take oral contraceptives for more limited timeframes [72].

By raising the gonadotropin levels, fertility medications support the development of numerous follicles and, subsequently, a few ovulations. infertility meds that are recommended as often as possible incorporate clomiphene citrate, which has been around since the mid-1960s and is a specific estrogen receptor modulator with synthetic properties like tamoxifen. Different gonadotropins incorporate human menopausal gonadotropin (hMG), follicle animating chemical (FSH), human chorionic gonadotropin (hCG), and gonadotropin-delivering chemical (GnRH). These prescriptions may depend on the infertility explanation and the endorsed treatment plan, either alone or in the blend [73, 74].

The well-being of ovulation-prompting meds was truly addressed by these papers, which prompted a few more explorations. The utilization of regenerative meds was not connected to a raised frequency of ovarian cancer in various later case-control and associate studies. Notwithstanding,

stresses over expected results of fertility prescriptions continued as additional exploration uncovered that women treated with ripeness meds for more than 12 cycles had an expanded threat of developing ovarian cancer. Results from some of these past studies likewise proposed that women who kept on being nulligravid after contraceptive treatment would be at a raised threat for ovarian cancer and that ripeness meds could specifically upgrade the threat of fringe ovarian cancers. There has likewise been conflict in the discoveries of a couple of prior studies that took a gander at the association between the IVF treatment and the threat of the ovarian cancer. Some revealed that there is no association, while others found one. The IVF clients had a higher possibility of creating ovarian cancer [75].

Sample size determination

Taro Yamanes sample size determination formula was applied,

$$\text{Sample size} = N / 1 + \text{SD}^2 N = 593 / 1 + (0.05)^2 \times 593 = 239$$

Here, N= 593 patients in hospitals during the study period.

SD= 0.05.

Source: Patient admission records of different hospitals under study

Research design

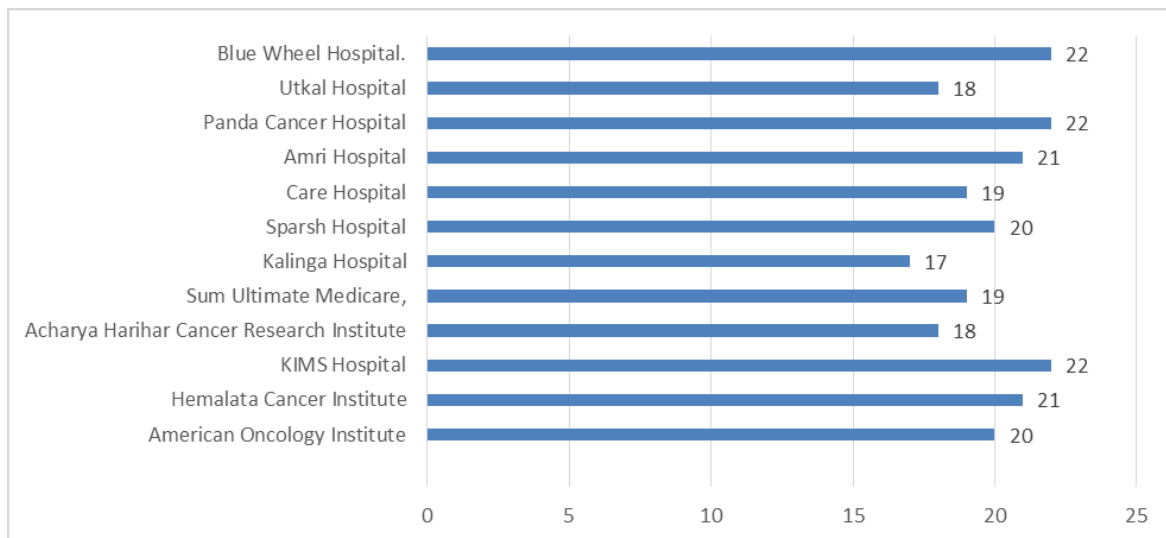
In [Table 1](#), it shows the response rates for various hospitals were 50%, 52.5%, 55%, 45% 45.24%, and 44.74% for Oncology Institute, Hemalata Cancer Institute, KIMS Hospital, Acharya Harihar Cancer Research Institute, Sum Ultimate Medicare, and Kalinga Hospital respectively. Similarly for the others like Sparsh Hospital, Care Hospital, Amri Hospital, Panda Cancer Hospital, Utkal Hospital, and Blue Wheel Hospital the response rate was 48.78%, 51.35%, 52.38%, 43.90%, and 52.38%, respectively. The overall rate of response was 49.48%.

The [Figure 1](#) data being taken from the [Table 1](#) for various hospitals.

Table 1: Sampling frame

Name of the hospitals	Questionnaire distributed	Questionnaire collected	Percentage of response
American Oncology Institute	40	20	50
Hemalata Cancer Institute	40	21	52.5
KIMS Hospital	40	22	55
Acharya Harihar Cancer Research Institute	40	18	45
Sum Ultimate Medicare,	42	19	45.24
Kalinga Hospital	38	17	44.74
Sparsh Hospital	41	20	48.78
Care Hospital	37	19	51.35
Amri Hospital	40	21	52.5
Panda Cancer Hospital	42	22	52.38
Utkal Hospital	41	18	43.90
Blue Wheel Hospital.	42	22	52.38
Total	483	239	49.48

Source: Primary data



Source: Table 1

Figure 1: Sample size for each hospital collected

In Table 2, it shows the awareness level of ovarian cancer was 23% completely aware, 31.80% moderate, and 45.2% not aware. Concerning the occupation, 64% were homemakers and the rest were working women. In the case of place of origin, 26.77% were rural, 32.64% were semi-urban and the rest were urban. In the case of literacy rate, 78.24% were literate and the rest were illiterate. In the case of marital status, 30.13% were married, 26.36% were unmarried, 23.85% were widows and the balance were divorcees. For the age group, 51.05% were in the group 23-30, and the rest were between 31-38 years old.

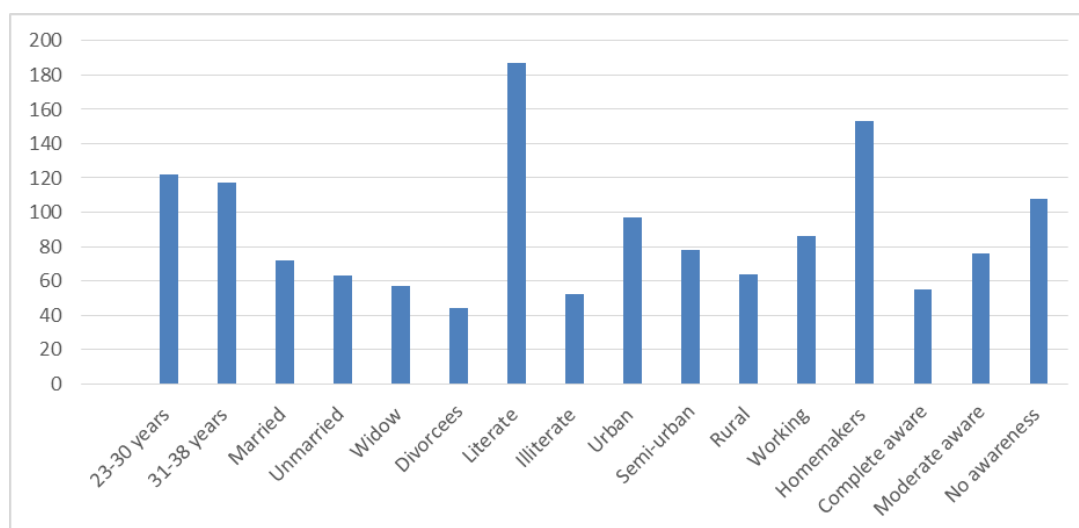
Convenience and snowball sampling is being used. To collect the desired sample size, 483 questionnaires were administered and the rate of response was 49.48%. The various attributes considered from the research gaps like night shifts or sleeping late at night, IVF, contraceptive pills affect the circadian cycle, and the overall health of an individual. Initially, fourteen variables were identified. However, after the pilot study was the same, they were reduced to nine only.

Figure 2 demographic profile data being taken from the Table 2.

Table 2: Demographic profile of the respondents

Age	23-30 years old	122	51.05%
	31-38 years old	117	48.95%
Marital status	Married	72	30.13%
	Unmarried	63	26.36%
	Widow	57	23.85%
	Divorcees	44	18.41%
Literacy status	Literate	187	78.24%
	Illiterate	52	21.76%
Place of origin	Urban	97	40.59%
	Semi urban	78	32.64%
	Rural	64	26.77%
Occupation	Working	86	36%
	Homemakers	153	64%
Level of awareness about ovarian cancer	Complete	55	23%
	Moderate	76	31.80%
	No awareness	108	45.2%

Source: Primary data



Source: Table 2

Figure 2: Demographic Profile

This exploratory study examined the extent to which variables such as night shifts, smoking, IVF, and others contribute to ovarian cancer. Pearsons Chi-square, one sample t-tests, Cramers V, and Phi tests were conducted for the present study to establish whether a significant association exists between the attributes considered and ovarian cancer cases.

Data analysis

For the non-parametric tests conducted with a smaller sample size, Chi-square was applied to test the hypothesis. This test was used to compare the observed frequencies to the

anticipated frequencies. For parametric tests like an independent t-test is applied, when the sample size is small and the population standard deviation is not available, it is essentially testing the significance of the difference between the mean values, to validate factors relating to lifestyle and habits of women which could be associated with ovarian cancer among millennials belonging to Odisha, India. The details about the respondents were received from selected hospitals and with permission from the authorities after consent from the hospital and respondents this study has been undertaken. Out of the total respondents, only 31 respondents had reported ovarian cancer in their mothers.

Table 3: Frequency table for ovarian cancer related to mother

Ovarian_cancer_mother					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	207	86.7	86.7	86.7
	Yes	31	13.3	13.3	100.0
	Total	239	100.0	100.0	

Table 4: Frequency Table for consumption of the contraceptive pill

Contraceptive_pill					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	48	20.0	20.0	20.0
	Yes	191	80.0	80.0	100.0
	Total	239	100.0	100.0	

Table 5: Frequency table of smoking habits

Smoking_habit					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	207	86.7	86.7	86.7
	Yes	32	13.3	13.3	100.0
	Total	239	100.0	100.0	

Table 3, 4 and 5 represents the frequency table related to the ovarian cancer mothers, frequency table for consumption of the contraceptive pills and frequency for smoking habits of the respondents respectively. Eighty-six percent (approx.) of the respondents reported that they have never smoked a cigarette and 32 of the respondents have a habit of smoking cigarettes or bidi (local rolled cigarettes from Coromandel ebony leaves or tendu leaves where tobacco is inserted).

Eighty percent of the respondents which is 191 reported that they consumed contraceptive pills occasionally for a certain period.

Hypothesis tests

H₁: There is no relationship between respondents having children and ovarian cancer.

To measure the strength between the association Cramers V* is applied to test data when a significant Chi-square result has been obtained, according to this test which measures the value that lies between 0 to 0.5 where the relationship grows stronger as the value gets higher from 0 to

0.5. In this study, Cramers V is 0.092 for having a child and ovarian cancer which means there is a very weak relationship between bearing a child and ovarian cancer. One sample t-test statistics section displays descriptive statistics for the sample, including a comparison of mean and test value. The one-sample test section displays the results of t-test. The significance level (alpha) is 0.05. The Sig. or p-value column displays the significance level for the test. The results show that the p-value (0.492) is above 0.05. This indicates that the variables are independent of each other. These multiple-hypothesis tests allow us to accept the null hypothesis that having a child or not having one has no impact on the ovarian cancer.

H₂: There is no relationship between the mothers of the respondents having ovarian cancer and ovarian cancer in respondents. The Chi-square value is 0.423 which indicates that both variables are dependent on each other. The one sample t-test is *0.041 which implies that *p-value is comparatively more than the standard 0.05.

Table 6: Hypotheses were tested through i) Pearsons chi-square test*, ii) Cramers V*, and iii) Phi*

Formulae	$\chi^2 = \sum \frac{(O-E)^2}{E}$	$T = \frac{(\bar{X} - \mu)}{S/\sqrt{n}}$	$\phi = \sqrt{\frac{\chi^2}{N}}$	$\sqrt{(X2/n)/mi}$ n(c-1, r-1)
	Pearson Chi-Square	T-test	Phi	Cramers V
	P-value	p-value	p-value	p-value
Having child	0.057	0.069	0.492	0.492
History of mother	0.423	0.041	0.207	0.207
History of sister	0.36	0.083	0.237	0.237
Contraceptive pill	0.001	0.000	0.001	0.001
Obese	0.068	0.179	0.118	0.118
Late sleeping habits	0.207	0.08	0.318	0.318
Tobacco smoke	0.003	0.000	0.002	0.002
IVF	0.207	0.219	0.123	0.123
Diabetic	0.034	0.000	0.025	0.025

In Table 6, the various analysis related to pearsons chi-square test, t-test and other relevant tests for analysis of data done. Cramers V and Phi results are 0.207 in this study which concludes that the association between both variables is independent of each other and from the above study, we conclude that irrespective of the fact that the mother of the respondent having or does not have ovarian cancer has no impact on ovarian cancer. We accept the null hypothesis.

H₃: There is no relationship between sisters of the respondent having ovarian cancer and ovarian cancer in respondents.

The Chi-square value is 0.36 which indicates that both variables are independent of each other. The one sample t-test is *0.083 which implies that * p-value is more than the standard 0.05. Cramers V and Phi results are 0.237 in this study which concludes that the association between sisters having ovarian cancer is independent of ovarian cancer among the respondent. The null hypothesis is accepted.

H₄: There is no relationship between respondents taking contraceptive pills and ovarian cancer among the respondents.

The Chi-square value is 0.001 which indicates that both variables are independent of each other. The one sample t-test is 0.000 which implies that the p-value is more than the

standard 0.05. Cramers V and Phi results in 0.001 in this study which concludes that the association between the consumption of contraceptive pills is dependent on the variable ovarian cancer. Hence, the null hypothesis is rejected.

H₅: There is no relationship between respondents being obese and having ovarian cancer among the respondents.

The Chi-square value is 0.068 which indicates that both variables are independent of each other. The one sample t-test is 0.179 which implies that the p-value is more than the standard 0.05. Cramers V and Phi results are 0.118 in this study which concludes that there is no association between respondents being obese and ovarian cancer among the respondents. The null hypothesis is accepted.

H₆: There is no relationship between respondents sleeping late and ovarian cancer among the respondents.

The Chi-square value of 0.207 indicates that both variables are independent of each other. The one sample t-test is 0.08 which implies that the p-value is more than the standard 0.05. Cramers V and Phi results in 0.318 in this study which concludes that the relationship between respondents sleeping late and ovarian cancer among the respondents is null. The null hypothesis is accepted.

H₇: There is no relationship among respondents smoking habits and ovarian cancer among the respondents.

The Chi-square value is 0.003 which indicates that both variables are independent of each other. The one sample t-test is 0.000 which implies that the p-value is more than the standard 0.05. Cramers V and Phi results in 0.002 in this study which concludes that there lies a strong relationship among respondents smoking habits and ovarian cancer among the respondents. The null hypothesis is rejected.

H₈: There is no relationship between respondents having children through IVF and ovarian cancer among the respondents.

The Chi-square value of 0.207 indicates that both variables are independent of each other. The one sample t-test is 0.219 which implies that the p-value is more than the standard 0.05. Cramers V and Phi results in 0.123 in this study which concludes that there is no relationship between variables having children through IVF and ovarian cancer among the respondents. The null hypothesis is accepted.

H₉: There is no relationship among respondents being diabetic and ovarian cancer among the respondents.

The Chi-square value is 0.034 which indicates that both variables are independent of each other. The one sample t-test is 0.000 which implies that the p-value is more than the standard 0.05. Cramers V and Phi results are 0.025 in this study which concludes that there is a relationship among respondents being diabetic and ovarian cancer among the respondents. The null hypothesis is rejected.

Conclusion

The study observed that having children, a mother with an ovarian cancer history, a sister having ovarian cancer history, being obese, having late sleeping habits, and having IVF have a significant association with ovarian cancer. However, using contraceptive pills, smoking habits, and diabetic history do not have any significant association from the survey data. The result may differ if we consider the different demographic profiles. This needs to be addressed

to the extent possible. Especially those attributes can be diet, late sleeping, etc. Regular physical exercise should be included in the daily routine and whenever any such symptoms are being felt should be consulted with the physicians without any delay so that appropriate treatment can be taken care.

Scope for future research

The present study concentrated on ovarian cancer only; however, another related study can be undertaken such as breast, colorectal, endometrial, lung, cervical, and skin. The study can be extended to the other segments like generation X and Boomers in addition to millennials for ovarian cancer research in different parts of society and segments.

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Authors contributions

All authors contributed equally in this manuscript regarding selecting the criteria of patients, analyzing the data, writing, and revising the paper.

Contributions of authors

All authors participated in the data analysis, drafting, and revising of the publication, as well as accepting responsibility for all elements of this work.

Conflict of Interest

There is no conflict of interest with any data, organization, community, or individual.

References

- [1]. Sánchez-Lorenzo L., Salas-Benito D., Villamayor J., Patiño-García A., González-Martín A., The BRCA Gene in Epithelial Ovarian Cancer. *Cancers* 2022, **14**:1235 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [2]. Huang J., Chan W.C., Ngai C.H., Lok V., Zhang L., Lucero-Prisno D.E., Xu W., Zheng Z.J., Elcarte E., Withers M., Wong M.C.S., Worldwide Burden, Risk Factors, and Temporal Trends of Ovarian Cancer: A Global Study, *Cancers*, 2022, **14**:2230 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [3]. Oktem O., In response to: why double ovarian stimulation in an in vitro fertilization cycle is potentially unsafe? *Human Reproduction*, 2022, **37**:1945 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [4]. Gkamprana A.M., Adamopoulou K., Kavvadias A., Grammatikakis K., Halkia E., Patsouras K., P-785 Assisted reproduction techniques and the risk of ovarian tumors, *Human Reproduction*, 2022, **37**:deac105-084 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [5]. Tocci A., Why double ovarian stimulation in an in vitro fertilization cycle is potentially unsafe, *Human Reproduction*, 2022, **37**:199 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [6]. Inamdar A., Loo A., Rare case of metastatic ovarian clear cell carcinoma with an in vitro fertilization pregnancy, *American Journal of Clinical Pathology*, 2019, **152**: [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [7]. Gersekowski K., Delahunty R., Alsop K., Goode E.L., Cunningham J.M., Winham S.J., Pharoah P., Song H., Jordan S., Fereday S., DeFazio A., Friedlander M., Obermair A., Webb P.M., Germline BRCA variants, lifestyle and ovarian cancer survival, *Gynecologic Oncology*, 2022, **165**:437 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [8]. Beaudoin C.E., Hong T., Emotions in the time of coronavirus: Antecedents of digital and social media use among millennials, *Computers in Human Behavior*, 2021, **123**:106876 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [9]. Minlikeeva A.N., Cannioto R., Jensen A., Kjaer S.K., Jordan S.J., Diergaard B., Szender J.B., Odunsi K., Almohanna H., Mayor P., Starbuck K., Zsiros E., Bandera E.v., Cramer D.W., Doherty J.A., DeFazio A., Edwards R., Goode E.L., Goodman M.T., ... Moysich K.B., Joint exposure to smoking, excessive weight, and physical inactivity and survival of ovarian cancer patients, evidence from the Ovarian Cancer Association Consortium, *Cancer Causes & Control*, 2019, **30**:537 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [10]. Santucci C., Bosetti C., Peveri G., Liu X., Bagnardi V., Specchia C., Gallus S., Lugo A., Dose-risk relationships between cigarette smoking and ovarian cancer histotypes: a comprehensive meta-analysis, *Cancer Causes & Control*, 2019, **30**:1023 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [11]. Scherübl H., Tobacco smoking and gastrointestinal cancer risk, *Visceral Medicine*, 2022, **38**:217 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [12]. Adani G., Filippini T., Wise L.A., Halldorsson T.I., Blaha L., Vinceti M., Dietary intake of acrylamide and risk of breast, endometrial, and ovarian cancers: a systematic review and dose-response meta-analysis, *Cancer Epidemiology Biomarkers and Prevention*, 2020, **29**:1095 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [13]. Nguyen H.N., Averette H.E., Janicek M., Ovarian carcinoma: a review of the significance of familial risk factors and the role of prophylactic oophorectomy in cancer prevention, *Cancer*, 1994, **74**:545 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [14]. Angeli D., Salvi S., Tedaldi G., Genetic predisposition to breast and ovarian cancers: how many and which genes to test?, *International journal of molecular sciences*, 2020, **21**:1128 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [15]. Al Abdeena S.H.Z., Mustafa Y.F., Mutlagc S.H., Synthesis of disubstituted anisolodipyronederived ester compounds: The search for new bioactive candidates, *Eurasian Chemical Communications*, 2022, **4**:1171 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [16]. Yaseen M.M., Alkubaisy S.A., Mohammad W.T., Jalil A.T., Dilfy S.H., Cancer and Complications of Peptic Ulcer in Type 2 Diabetes Mellitus patients at Wasit province, Iraq, *Journal of Medicinal and Chemical Sciences*, 2023, **6**:335 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [17]. Abdullahi S.H., Uzairu A., Ibrahim M.T., Umar A.B., Chemo-informatics activity prediction,

- ligand based drug design, Molecular docking and pharmacokinetics studies of some series of 4, 6-diaryl-2-pyrimidinamine derivatives as anti-cancer agents, *Bulletin of the National Research Centre*, 2021, **45**:167 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [18]. Lheureux S., Braunstein M., Oza A.M., Epithelial ovarian cancer: Evolution of Management in the era of Precision Medicine, *CA: A Cancer Journal for Clinicians*, 2019, **69**:280 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [19]. Tripathy S., Gangwar R., Supraja P., Rao A.V.S.S.N., Vanjari S.R., Singh S.G., Graphene doped mn2o3 nanofibers as a facile electroanalytical DNA point mutation detection platform for early diagnosis of breast/ovarian cancer, *Electroanalysis*, 2018, **30**:2110 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [20]. Sahoo A.P., Patnaik B.C.M., Satpathy I., Quality of Life (QoL) of Breast Cancer (BC) survivors of the rural investor community in India, *Journal of Medicinal and Chemical Sciences*, 2023, **6**:946 [[Crossref](#)], [[Publisher](#)]
- [21] Llorca G., Chirivella I., Morales R., Serrano R., Sanchez A.B., Teulé A., Lastra E., Brunet J., Balmaña J., Graña B., SEOM clinical guidelines in Hereditary Breast and ovarian cancer, *Clinical and Translational Oncology*, 2015, **17**:956 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [22]. Jacobson M., Bernardini M., Sobel M.L., Kim R.H., McCuaig J., Allen L., No. 366-gynaecologic management of Hereditary Breast and Ovarian Cancer, *Journal of Obstetrics and Gynaecology Canada*, 2018, **40**:1497 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [23]. von Gruenigen V.E., Huang H.Q., Beumer J.H., Lankes H.A., Tew W., Herzog T., Hurria A., Mannel R.S., Rizack T., Landrum L.M., Rose P.G., Salani R., Bradley W.H., Rutherford T.J., Higgins R.V., Secord A.A., Fleming G., Chemotherapy completion in elderly women with ovarian, primary peritoneal or fallopian tube cancer - an NRG oncology/gynecologic oncology group study, *Gynecologic Oncology*, 2017, **144**:459 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [24]. Chen W., Zheng R., Baade P.D., Zhang S., Zeng H., Bray F., Jemal A., Yu X.Q., He J., Cancer statistics in China, 2015, *CA: a cancer journal for clinicians*, 2016, **66**:115 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [25]. Majidi A., Na R., Jordan S.J., De Fazio A., Webb P.M., Statin use and survival following a diagnosis of ovarian cancer: A prospective observational study, *International Journal of Cancer*, 2020, **148**:1608 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [26]. Reid F., Jones A., The World Ovarian Cancer Coalition Every Woman Study: Identifying Global and local challenges, and the opportunities to improve survival and quality of life for women no matter where they live, *Gynecologic Oncology*, 2019, **154**:39 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [27]. Menon U., Griffin M., Gentry-Maharaj A., Ovarian cancer screening—current status, *Future Directions, Gynecologic Oncology*, 2014, **132**:490 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [28]. Chen V.W., Ruiz B., Killeen J.L., Coté T.R., Wu X.C., Correa C.N., Howe H.L., Pathology and classification of ovarian tumors, *Cancer: Interdisciplinary International Journal of the American Cancer Society*, 2003, **97**:2631 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [29]. Kossai M., Leary A., Scoazec J.Y., Genestie C., Ovarian cancer: A heterogeneous disease, *Pathobiology*, 2017, **85**:41 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [30]. Prat J., Ovarian carcinomas: Five distinct diseases with different origins, genetic alterations, and clinicopathological features, *Virchows Archiv*, 2012, **460**:237 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [31]. Tomczak K., Czerwińska P., Wiznerowicz M., Review
The Cancer Genome Atlas (TCGA): an immeasurable source of knowledge, *Contemporary Oncology/Współczesna Onkologia*, 2015, **2015**:68 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [32]. Marquez R.T., Baggerly K.A., Patterson A.P., Liu J., Broaddus R., Frumovitz M., Atkinson E.N., Smith D.I., Hartmann L., Fishman D., Berchuck A., Whitaker R., Gershenson D.M., Mills G.B., Bast R.C., Lu K.H., Patterns of Gene Expression in Different Histotypes of Epithelial Ovarian Cancer Correlate with Those in Normal Fallopian Tube, Endometrium, and Colon, *Clinical Cancer*

- Research, 2005, **11**:6116 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [33]. Sun H., Chen X., Zhu T., Liu N., Yu A., Wang S., Age-dependent difference in impact of fertility preserving surgery on disease-specific survival in women with stage I borderline ovarian tumors, *Journal of Ovarian Research*, 2018, **11**:54 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [34]. Ribatti D., Tamma R., Annese T., Epithelial-mesenchymal transition in cancer: A historical overview, *Translational Oncology*, 2020, **13**:100773 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [35]. Kuhn E., Kurman R.J., Vang R., Sehdev A.S., Han G., Soslow R., Wang T.L., Shih I.M., TP53 mutations in serous tubal intraepithelial carcinoma and concurrent pelvic high-grade serous carcinoma-evidence supporting the clonal relationship of the two lesions, *The Journal of Pathology*, 2011, **226**:421 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [36]. Li J., Abushahin N., Pang S., Xiang L., Chambers S.K., Fadare O., Kong B., Zheng W., Tubal origin of ovarian low-grade serous carcinoma, *Modern Pathology*, 2011, **24**:1488 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [37]. Shih I.M., Kurman R.J., Ovarian tumorigenesis, *The American Journal of Pathology*, 2004, **164**:1511 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [38]. Marchbanks P.A., Wilson H., Bastos E., Cramer D.W., Schildkraut J.M., Peterson H.B., Cigarette smoking and epithelial ovarian cancer by histologic type, *Obstetrics & Gynecology*, 2000, **95**:255 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [39]. Farhud, D. D., Zokawi, S., Keykhaei, M., & Zarif Yeganeh, M., Strong evidences of the ovarian carcinoma risk in women after IVF treatment: A review article, *Iranian Journal of Public Health*, 2020, [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [40]. Tung K.H., Reproductive factors and epithelial ovarian cancer risk by histologic type: A multiethnic case-control study, *American Journal of Epidemiology*, 2003, **158**:629 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [41]. Zain Al Abdeen S., Mustafa Y., Mutlag S., Synthesis and biomedical activities of novel multifunctional benzodipyronone-based derivatives, *Eurasian Chemical Communications*, 2022, **4**:938 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [42]. Kondori B.J., Mohammad S., Hemadi H., Gouvarchin Ghaleh H.E., Mohammad A., Fard M., Dorostkar R., Al-Taie A.H.J., Pharmacological Study of the Antitumor Effect of Newcastle Oncolytic Virus in Combination with Copper Nanoparticles, Hyperthermia and Radiation on Malignant Colorectal Cancer Cell Line, *Journal of Medicinal and Chemical Sciences Journal Homepage: Journal of Medicinal and Chemical Sciences*, 2022, **5**:457 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [43]. Bray F., Loos A.H., Tognazzo S., La Vecchia C., Ovarian cancer in Europe: Cross-sectional trends in incidence and mortality in 28 countries, 1953-2000, *International Journal of Cancer*, 2004, **113**:977 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [44]. Buskwofie A., David-West G., Clare C.A., A review of cervical cancer: Incidence and disparities, *Journal of the National Medical Association*, 2020, **112**:229 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [45]. Bjørge T., Lie A.K., Hovig E., Gislefoss R.E., Hansen S., Jellum E., Langseth H., Nustad K., Tropé C.G., Dørum A., BRCA1 mutations in ovarian cancer and borderline tumours in Norway: A nested case-control study, *British Journal of Cancer*, 2004, **91**:1829 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [46]. Gates M.A., Rosner B.A., Hecht J.L., Tworoger S.S., Risk factors for epithelial ovarian cancer by histologic subtype, *American Journal of Epidemiology*, 2009, **171**:45 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [47]. Poole E.M., Wentzensen N.A., Trabert B., Tworoger S.S., Abstract 1761: Serous ovarian cancer risk factors by grade: Evidence for etiologic heterogeneity, *Cancer Research*, 2016, **76**:1761 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [48] Abdulaziz N.T., Mustafa Y.F., The Effect of Heat Variable on the Chemical Composition and Bioactivities of a Citrullus lanatus Seed Aqueous Extracts, *Journal of Medicinal and Chemical Sciences*, 2022, **5**:1166 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]

- [49] Waheed S.A., Mustafa Y.F., Maleki A., Synthesis and Evaluation of New Coumarins as Antitumor and Antioxidant Applicants, *Journal of Medicinal and Chemical Sciences*, 2022, **5**:808 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [50]. Ogino S., Nishihara R., VanderWeele T.J., Wang M., Nishi A., Lochhead P., Qian Z.R., Zhang X., Wu K., Nan H., Yoshida K., Milner D.A., Chan A.T., Field A.E., Camargo C.A., Williams M.A., Giovannucci E.L., The role of molecular pathological epidemiology in the study of neoplastic and non-neoplastic diseases in the era of precision medicine, *Epidemiology*, 2016, **27**:602 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [51]. Didkowska J., Wojciechowska U., Michalek I.M., Caetano dos Santos F.L., Cancer incidence and mortality in Poland in 2019, *Scientific Reports*, 2022, **12**:10875 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [52]. Herrinton L.J., Stanford J.L., Schwartz S.M., Weiss N.S., Ovarian cancer incidence among Asian migrants to the United States and their descendants, *JNCI Journal of the National Cancer Institute*, 1994, **86**:1336 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [53]. Kliewer E.V., Smith K.R., Breast cancer mortality among immigrants in Australia and Canada, *JNCI Journal of the National Cancer Institute*, 1995, **87**:1154 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [54]. Wei K.R., Zheng R.S., Zhang S.W., Liang Z.H., Li Z.M., Chen W.Q., Nasopharyngeal carcinoma incidence and mortality in China, 2013, *Chinese Journal of Cancer*, 2017, **36**:90 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [55]. Van der Looij, M., Szabo, C., Besznyak, I., Liszka, G., Csokay, B., Pulay, T., Toth, J., Devilee, P., King M.C., Olah E., Prevalence of FOUNDERBRCA1 ANDBRCA2 mutations among breast and ovarian cancer patients in Hungary, *International Journal of Cancer*, 2000, **86**:737 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [56] Ford D., Risks of cancer in BRCA1-mutation carriers, *The Lancet*, 1994, **343**:692 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [57] Antoniou A., Pharoah P.D., Narod S., Risch H.A., Eyfjord J.E., Hopper J.L., Loman N., Olsson H., Johannsson O., Borg Å., Pasini B., Average risks of breast and ovarian cancer associated with BRCA1 or BRCA2 mutations detected in case series unselected for Family history: A combined analysis of 22 studies, *The American Journal of Human Genetics*, 2003, **72**:1117 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [58]. Lynch H.T., Smyrk T., Hereditary nonpolyposis colorectal cancer (Lynch Syndrome): An updated review, *Cancer*, 1996, **78**:1149 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [59]. Pal T., Permuth-Wey J., Betts J.A., Krischer J.P., Fiorica J., Arango H., LaPolla J., Hoffman M., Martino M.A., Wakeley K., Wilbanks G., brca1andbrca2mutations account for a large proportion of ovarian carcinoma cases, *Cancer*, 2005, **104**:2807 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [60]. Bonadona V., Cancer risks associated with germline mutations in mlh1, msh2, and msh6 genes in Lynch syndrome, *JAMA*, 2011, **305**:2304 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [61]. Song H., Dicks E., Ramus S.J., Tyrer J.P., Intermaggio M.P., Hayward J., Edlund C.K., Conti D., Harrington P., Fraser L., Philpott S., Contribution of germline mutations in the rad51b, rad51c, and rad51d genes to ovarian cancer in the population, *Journal of Clinical Oncology*, 2015, **33**:2901 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [62]. Ramus S.J., Song H., Dicks E., Tyrer J.P., Rosenthal A.N., Intermaggio M.P., Fraser L., Gentry-Maharaj A., Hayward J., Philpott S., Anderson C., Germline mutations in the BRIP1, Bard1, PALB2, and NBN genes in women with ovarian cancer, *JNCI: Journal of the National Cancer Institute*, 2015, **107** [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [63]. Lakhani S.R., Manek S., Penault-Llorca F., Flanagan A., Arnout L., Merrett S., McGuffog L., Steele D., Devilee P., Klijn J.G., Meijers-Heijboer H., Pathology of ovarian cancers in brca1 and brca2 carriers, *Clinical Cancer Research*, 2004, **10**:2473 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [64]. Ford D., Risks of cancer in BRCA1-mutation carriers, *The Lancet*, 1994, **343**:692 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [65]. Pal T., Akbari M.R., Sun P., Lee J.H., Fulp J., Thompson Z., Coppola D., Nicosia S., Sellers T.A.,

- McLaughlin J., Risch H.A., Rosen B., Shaw P., Schildkraut J., Narod S.A., Frequency of mutations in mismatch repair genes in a population-based study of women with ovarian cancer, *British Journal of Cancer*, 2012, **107**:1783 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [66]. Pal T., Akbari M.R., Sun P., Lee J.H., Fulp J., Thompson Z., Coppola D., Nicosia S., Sellers T.A., McLaughlin J., Risch H.A., Rosen B., Shaw P., Schildkraut J., Narod S.A., Frequency of mutations in mismatch repair genes in a population-based study of women with ovarian cancer, *British Journal of Cancer*, 2012, **107**:1783 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [67]. Bahcall O.G., ICOGS collection provides a collaborative model, *Nature Genetics*, 2013, **45**:343 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [68]. Shen W., Jiang W., Ye S., Sun M., Yang H., Shan B., Construction and validation of a robust epigenetic gene-set based signature in ovarian cancer, 2020 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [69]. Permuth-Wey J., Sellers T.A., Epidemiology of Ovarian Cancer, *Methods in Molecular Biology*, 2009, 413 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [70]. uchenbaecker K.B., Ramus S.J., Tyrer J., Lee A., Shen H.C., Beesley J., Lawrenson K., McGuffog L., Healey S., Lee J.M., Spindler T.J., Identification of six new susceptibility loci for invasive epithelial ovarian cancer, *Nature genetics*, 2015, **47**:164 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [71]. Bolton K.L., Tyrer J., Song H., Ramus S., Jones C., Notaridou M., Chanock S.J., Garcia-Closas M., Chenevix-Trench G., Gayther S., Pharoah P.D., Abstract 4729: A genome-wide association study of Ovarian Cancer Prognosis identifies a novel locus for aggressive serous cancer on 19p13, *Cancer Research*, 2010, **70**:4729 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [72]. Kelemen L.E., Tyrer J., Phelan C.M., Ramus S.J., Berchuck A., Gayther S.A., Goode E.L., Pearce C.L., Schildkraut J.M., Chenevix-Trench G., Monteiro A.N., Goodman M.T., Sellers T.A., Pharoah P.P., Abstract 3283: GWAS identifies risk variants for mucinous ovarian carcinoma, *Cancer Research*, 2014, **74**:3283 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [73]. Song H., Ramus S.J., Tyrer J., Bolton K.L., Gentry-Maharaj A., Wozniak E., Anton-Culver H., Chang-Claude J., Cramer D.W., DiCioccio R., Dörk T., A genome-wide association study identifies a new ovarian cancer susceptibility locus on 9p22.2, *Nature Genetics*, 2009, **41**:996 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [74]. Goode E.L., Chenevix-Trench G., Song H., Ramus S.J., Bolton K., Vierkant R.A., Berchuck A., Lawrenson K., Palmieri R., Tsai Y.Y., Sellers T.A., Abstract 3860: New ovarian cancer susceptibility loci identified, *Cancer Research*, 2010, **70**:3860 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [75]. Kim J., Park E., Kim O., Schilder J., Coffey D., Cho C.H., Bast R., Cell origins of high-grade serous ovarian cancer, *Cancers*, 2018, **10**:433 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]

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