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Towards a Multifactorial Model of Cancer Risk: Findings from a Pilot Survey of 134 Cancer Patients

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Abstract: Background: Cancer is increasingly recognised as a multifactorial disease influenced by the interaction of genetic, environmental, behavioural, and biological factors. While considerable progress has been made in identifying individual risk factors, understanding how these factors coexist and interact within affected individuals remains a major challenge for cancer epidemiology and prevention. Objective: To examine the prevalence and interrelationships of multiple cancer-related risk factors among individuals with medically diagnosed cancer and to explore their relevance to contemporary multifactorial models of cancer risk. Method: This pilot survey study analysed responses from 134 individuals reporting a medically diagnosed cancer. Information was collected on age at diagnosis, cancer type, family history of cancer, tobacco and alcohol use, dietary habits, physical activity, occupational and environmental exposures, and chronic infections or inflammatory conditions associated with increased cancer risk. Descriptive statistics, chi-square analyses, multivariable logistic regression, ordinal logistic regression, and exploratory factor analysis were conducted. Results: The cohort demonstrated a substantial cumulative burden of risk factors. 75% of respondent reported an immediate family history of cancer. 84.2% reported tobacco exposure. 85.1% reported at least one occupational or environmental exposure. And 68.3% reported chronic infections or inflammatory conditions associated with increased cancer risk. More than 70% of the respondents were classified within the high risk burden category. Significant associations were identified between family history and breast cancer status, environmental exposure and lung cancer, radiation exposure and leukaemia lymphoma, and cumulative risk burden, and early age of diagnosis. Exploratory clock factor analysis identified distinct clusters of inherited occupational, and lifestyle related risk factors supporting a multifactorial interpretation of cancer risk.

Keywords: Cancer epidemiology, multicultural risk, cancer prevention, environmental exposure, lifestyle factors, chronic information, early onset cancer, cancer risk.

INTRODUCTION

Cancer remains one of the leading causes of morbidity and mortality worldwide. Unlike many infectious diseases for which a single causal agent has been identified and effectively controlled, cancer represents a highly heterogeneous group of disorders characterised by complex interactions among genetic susceptibility, environmental exposures, behavioural influences, and biological processes.

Historically, epidemiological investigations have often focused on isolated risk factors. While such approaches have generated valuable insights, growing evidence suggests that cancer development is rarely attributable to a single determinant. Contemporary models increasingly recognise the importance of multifactorial causation, whereby inherited susceptibility, lifestyle characteristics, occupational exposures, environmental hazards, and chronic disease processes combine to influence cancer risk.

The present pilot study was undertaken to examine these interacting influences within a cohort of medically diagnosed cancer patients. Rather than evaluating a single risk factor in isolation. This study sought to explore the prevalence and co-occurrence of multiple recognised cancer-related risk factors and to investigate their associations with cancer type and age at diagnosis.

The aim was not to establish causation, but to generate exploratory evidence relevant to a, and to identify potential avenues for future research and prevention strategies.

METHODS

This pilot survey study analysed responses from 134 individuals who reported a medically diagnosed cancer. Participants completed a structured questionnaire examining demographic characteristics, age at diagnosis, cancer type, family history of cancer, tobacco and alcohol use, dietary habits, physical activity, occupational and environmental exposures, and chronic infections or inflammatory conditions associated with increased cancer risk.

Descriptive statistics were used to summarise demographic and clinical characteristics. Associations between categorical variables were examined using chi-square analyses with effect sizes reported as Cramér's V. Multivariable logistic regression models were used to examine predictors of early age at diagnosis, and selected cancer outcomes. Exploratory ordinal logistic regression and exploratory factor analysis were conducted to investigate broader patterns among risk factors and precursor variables.

Given the exploratory nature of the study and the modest sample size, findings were interpreted as hypothesis-generating rather than definitive evidence of causation. The findings presented in this study provide compelling evidence that challenges several widely held assumptions about cancer risk and diagnosis. Contrary to the common belief that cancer affects older adults the data indicates that a substantial proportion of respondents were diagnosed at relatively young ages, with more than half receiving a diagnosis by age 40 and most by age 50. These findings call into question the view that cancer is primarily a disease of later life and are consistent with recent literature documenting the growing importance of early-onset cancers across multiple cancer types (Ugai et al., 2022). The results also question the assumption that cancer risk is largely driven by isolated factors. Instead, most respondents exhibited a high burden of multiple risk factors, including family history, tobacco use, occupational or environmental exposures, and chronic infections or inflammatory conditions. Notably, 70.9% of the sample fell into the high-burden category, suggesting that cancer risk is often multifactorial and accumulates across domains, rather than reflecting a single dominant cause (Wu et al., 2018). In addition, the data questions the idea that specific cancers are associated with narrow or uniform risk profiles. Strong associations were observed between lung cancer and environmental exposures, such as diesel exhaust and asbestos, while breast cancer was strongly associated with family history. Taken together, these findings highlight the limitations of simplified models of cancer causation and suggest a more integrated understanding of cancer risk maybe. They further suggest the need to re-examine prevailing assumptions in cancer epidemiology and to adopt broader, more inclusive approaches to risk assessment and prevention.

Sample Characteristics and Cancer Profile

The analytic sample included 134 respondents who reported having been diagnosed with cancer by a medical professional. Age at diagnosis was available for 133 respondents. Most respondents were diagnosed by age 50, including 74 respondents (55.6%) diagnosed at age 40 or younger and 115 respondents (86.5%) diagnosed at age 50 or younger, a pattern consistent with broader concern about early-onset cancer across multiple cancer types (Ugai et al., 2022). Using the midpoint-coded age variable, the mean estimated age at diagnosis was 39.66 years (*SD* = 11.12), and the mean estimated current age was 45.23 years (*SD* = 10.42). The sample was 57.9% female and 41.4% male, with one respondent preferring not to report sex at birth. The sample was predominantly White/Caucasian (91.0% of valid responses). Educational attainment was high: 66.9% reported a bachelor's degree and 21.1% reported a postgraduate degree.

The most frequently reported cancer type was breast cancer (*n* = 64, 47.8%), followed by skin cancer (*n* = 43, 32.1%), prostate cancer (*n* = 33, 24.6%), lung cancer (*n* = 25, 18.7%), colorectal cancer (*n* = 16, 11.9%), leukemia/lymphoma (*n* = 13, 9.7%), and other or unspecified cancer types (*n* = 10, 7.5%). Because respondents could select more than one cancer type, categories were not mutually exclusive. Overall, 32 respondents (23.9%) reported two or more cancer-type categories.

Cancer-Related Risk and Precursor Variables

Risk-related responses were common in the sample. Among respondents with valid data, 99 of 132 (75.0%) reported an immediate family history of cancer, and 112 of 133 (84.2%) reported ever smoking tobacco. Among those with smoking data, 47 respondents (35.3%) were current smokers and 86 (64.7%) were former smokers. Based on midpoint-coded variables, the mean estimated pack-years was 14.19 (*SD* = 11.79). Weekly or more frequent alcohol use was reported by 59 respondents (44.0%), and 45 of 133 respondents (33.8%) reported five or more servings of red or processed meat per week. In addition, 82 of 133 respondents (61.7%) reported 10 or fewer servings of fruits and vegetables per week, and 74 of 133 respondents (55.6%) reported sedentary or light physical activity.

Occupational or environmental exposures were also often reported. Overall, 114 respondents (85.1%) reported at least one listed exposure. The most common exposure was industrial chemicals or solvents (*n* = 71, 53.0%), followed by diesel exhaust (*n* = 53, 39.6%), asbestos (*n* = 51, 38.1%), radiation (*n* = 38, 28.4%), and pesticides/herbicides (*n* = 30, 22.4%). Chronic infections or inflammatory conditions associated with increased cancer risk were reported by 82 of 120 respondents with valid data (68.3%). The mean simple risk-factor count was 5.39 (*SD* = 1.75) out of 9 possible indicators, and 95 respondents (70.9%) were classified in the high-burden group (five or more risk factors), consistent with broader evidence that cancer risk often reflects the combined influence of multiple non-intrinsic factors rather than a single isolated exposure (Wu et al., 2018).

Bivariate Associations

Bivariate analyses were interpreted descriptively because many cross-tabulations were exploratory, multiple comparisons were conducted, and some tables had small, expected

cell counts. Family history was strongly associated with breast cancer status. Among respondents with breast cancer, 60 of 64 (93.8%) reported an immediate family history of cancer, compared with 39 of 68 respondents (57.4%) without breast cancer, x^2 (1, N = 132) = 23.29, p < .001, V = .42. Breast cancer status also varied by current age group, x^2 (5, N = 133) = 23.52, p < .001, V = .42. Reporting two or more cancer-type categories was associated with family history as well: 30 of 32 respondents (93.8%) with multiple cancer-type categories reported a family history, compared with 69 of 100 respondents (69.0%) without multiple cancer-type categories, x^2(1, N = 132) = 7.92, p = .005, V = .25.

Several exposure-related associations were observed, consistent with broader evidence that non-intrinsic and environmental factors contribute substantially to cancer risk (Wu et al., 2018). Lung cancer status was associated with diesel exhaust exposure, x^2 (1, N = 134) = 17.08, p < .001, V = .36; 19 of 25 respondents with lung cancer (76.0%) reported diesel exposure, compared with 34 of 109 respondents without lung cancer (31.2%). Lung cancer was also associated with asbestos exposure, x^2(1, N = 134) = 11.69, p < .001, V = .30, industrial chemical/solvent exposure, x^2(1, N = 134) = 4.46, p = .035, V = .18, and chronic infection/inflammatory condition status, x^2(1, N = 120) = 8.17, p = .004, V = .26. Leukemia/lymphoma status was associated with radiation exposure, x^2 (1, N = 134) = 16.71, p < .001, V = .35; 10 of 13 respondents with leukemia/lymphoma (76.9%) reported radiation exposure, compared with 28 of 121 respondents without leukemia/lymphoma (23.1%). This table, however, included one expected cell count below 5 and should be interpreted cautiously.

Earlier diagnosis was also related to risk burden and exposure status. Diagnosis at age 50 or younger was associated with any listed occupational/environmental exposure, x^2(1, N = 133) = 21.69, p < .001, V = .40; 105 of 115 respondents diagnosed by age 50 (91.3%) reported an exposure, compared with 9 of 18 respondents diagnosed after age 50 (50.0%). Risk-factor burden was also associated with diagnosis at age 50 or younger, x^2 (2, N = 133) = 16.63, p < .001, V = .35. Specifically, 88 of 95 respondents in the high-burden group (92.6%) were diagnosed by age 50, compared with 24 of 31 (77.4%) in the moderate-burden group and 3 of 7 (42.9%) in the low-burden group.

Multivariable Logistic Regression Models

In exploratory logistic regression, the model predicting diagnosis at age 40 or younger from family history, smoking, pack-years, alcohol use, red/processed meat intake, fruit/vegetable intake, physical activity, occupational/environmental exposure, and chronic infection/inflammatory condition status was not statistically significant, x^2(9, N = 119) = 14.39, p = .109, Nagelkerke R^2 = .153. The parallel model predicting diagnosis at age 50 or younger was statistically significant, x^2 (9, N = 119) = 22.41, p = .008, Nagelkerke R^2 = .153. In that model, the absence of any listed occupational/environmental exposure was associated with lower odds of diagnosis by age 50 relative to having an exposure, OR = 0.14, 95% CI [0.03, 0.64], p = .012.

A second adjusted model predicting diagnosis at age 40 or younger included family history, smoking, pack-years, any listed occupational/environmental exposure, chronic infection/inflammatory condition status, sex at birth, and current age. This model was significant, x^2 (7, N = 119) = 47.10, p < .001, Nagelkerke R^2 = .438. Male sex was associated with lower odds of diagnosis at age 40 or younger relative to female sex, OR = 0.29, 95% CI

[0.12, 0.74], p = .009, and each one-year increase in estimated current age was associated with lower odds of diagnosis at age 40 or younger, *OR* = 0.85, 95% *CI* [0.79, 0.92], p < .001. The adjusted model predicting multiple cancer-type categories was statistically significant overall, x^2 (7, N = 119) = 22.27, p = .002, Nagelkerke R^2 = .250, but no individual predictor reached p < .05.

Cancer-specific logistic models were also exploratory. The lung-cancer model was statistically significant, x^2(9, N = 120) = 22.76, p = .007, Nagelkerke R^2 = .270; absence of diesel exposure was associated with lower odds of lung cancer relative to diesel exposure, *OR* = 0.25, 95% *CI* [0.07, 0.85], p = .026. The breast-cancer model was significant, x^2(8, N = 132) = 36.95, p < .001, Nagelkerke R^2 = .326; absence of family history was associated with lower odds of breast cancer relative to family history, *OR* = 0.07, 95% *CI* [0.02, 0.23], p < .001. The skin-cancer model was not significant overall, x^2 (6, N = 133) = 12.07, p = .060, although absence of pesticide/herbicide exposure was associated with lower odds of skin cancer, *OR* = 0.33, 95% *CI* [0.13, 0.85], p = .021. The leukemia/lymphoma model was significant, x^2 (6, N = 120) = 18.16, p = .006, Nagelkerke R^2 = .294; absence of radiation exposure was associated with lower odds of leukemia/lymphoma, *OR* = 0.08, 95% *CI* [0.02, 0.39], p = .002.

Exploratory Ordinal Regression

An exploratory ordinal logistic regression was also conducted with the six-level age-at-diagnosis category as the ordered outcome. The model included family history, ever smoking, 16 or more pack-years, weekly or more frequent alcohol use, red/processed meat intake, low fruit/vegetable intake, sedentary or light physical activity, any listed occupational/environmental exposure, and chronic infection/inflammatory condition status. Complete-case analysis included 119 respondents. Because 275 observed outcome-by-predictor cells (77.7%) had zero frequencies, this model should be interpreted cautiously. The final model did not improve on the intercept-only model, x^2 (9, N = 119) = 3.64, p = .933, and explained extraordinarily minor variation in the ordered age-at-diagnosis categories, Nagelkerke R^2 = .015, McFadden R^2 = .004. No predictor had a statistically significant location coefficient, with all ps ≥ .473. Thus, the ordinal model did not provide evidence that the included risk and precursor variables jointly distinguished earlier from later age-at-diagnosis categories.

Exploratory Factor Analysis

An exploratory factor analysis was conducted on 13 binary cancer-related precursor/risk indicators using principal axis factoring with varimax rotation. Sampling adequacy was acceptable for exploratory purposes, KMO = .675, and Bartlett's test of sphericity was significant, x^2 (78) = 300.31, p < .001. The unconstrained eigenvalue-greater-than-one solution attempted to extract five factors but did not fully converge within 25 iterations, so constrained two- and three-factor solutions were examined. The two-factor solution explained 26.81% of the extracted variance after rotation. Factor 1 was defined most strongly by chronic infection/inflammatory condition (.76), asbestos exposure (.73), family history (.60), ever smoking (.50), and diesel exposure (.48). Factor 2 was defined by weekly or more frequent alcohol use (.70), low fruit/vegetable intake (-.58), 16 or more pack-years

(.47), and red/processed meat intake (.39). The three-factor solution explained 31.87% of the extracted variance, but the third factor was comparatively weak and cross-loaded with asbestos exposure, sedentary/light activity, and pesticide/herbicide exposure. Factor 1 broadly reflected a medical/occupational-and-inherited risk profile, Factor 2 reflected an unhealthy lifestyle/consumption risk profile, and Factor 3 reflected a weaker, less coherent mixed environmental/activity exposure profile. Reliability was low for both the occupational/environmental exposure indicators (Cronbach's α = .27) and the lifestyle risk indicators (Cronbach's α = .09), indicating that these indicators should be treated as separate risk markers rather than internally consistent scales.

CONCLUSION

The findings are consistent with contemporary evidence suggesting that cancer risk reflects the interaction of multiple determinants rather than a single explanatory pathway. Although the study's limitations require cautious interpretation, the results support continued investigation of integrated models of cancer development and prevention.

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The cohort demonstrated a substantial cumulative burden of risk factors. Seventy-five percent of respondents reported an immediate family history of cancer, 84.2% reported tobacco exposure, 85.1% reported at least one occupational or environmental exposure, and 68.3% reported chronic infections or inflammatory conditions associated with increased cancer risk. More than 70% of respondents were classified within the high-risk burden category.

Significant associations were identified between family history and breast cancer status, environmental exposures and lung cancer, radiation exposure and leukemia/lymphoma, and cumulative risk burden and earlier age at diagnosis. Exploratory factor analysis identified distinct clusters of inherited, occupational, and lifestyle-related risk factors, supporting a multifactorial interpretation of cancer risk.