A Simple Chart to Communicate Health-behaviors-associated Mortality

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Research

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Abstract

Background

Unhealthy behaviors are trending towards the increase. This study aimed to develop a risk chart for personalized risk communication, which can induce a positive health behavioral change.

Methods

2235 middle-aged men were followed-up for a median time of 28 years. Participants were assessed for their smoking status, quality of diet expressed in Healthy Nordic Diet (HND) index, and alcohol drinking. After controlling for age, Body Mass Index (BMI), physical activity, and comorbidities, the variables' association with all-cause mortality was analyzed.

Results

Smoking, quality of diet, and alcohol drinking were associated to mortality risk with hazard ratios (HRs) of 2.72 [95% confidence interval (CI) 2.22–3.33], 0.88 (95% CI 0.82–0.94), and 1.13 (95% CI 1.10–1.17) respectively. Based on the analysis model [area under the curve (AUC) 72.8, 95% CI 70.8–74.9], a risk chart was presented allowing risk comparison across different predictors and for different combinations of health behaviors. A dysergic BMI-smoking interaction was found, suggesting that the individual effects of smoking and high BMI on mortality were lower when they co-existed. The combined effects of obesity (BMI > 30), smoking, poor quality diet (HND 1), and high level of alcohol consumption (475 g/week) associated with nearly 10 folds the mortality risk of a normal weight individual engaged in healthy behavior regardless of age, and a loss of 20 years in median survival.

Conclusion

The risk chart can be used as a tool to offer personalized risk communication and improve individuals' perception of the risk associated to their health behaviors.

Key-points

- Unhealthy behaviors associated with 10 folds the risk of mortality of individuals with healthy behaviors.
- A median of 20 years lost could be attributed to unhealthy behaviors.
- The individual effects of smoking and high BMI on mortality were lower when they co-existed.
- Personalized risk prediction models could be turned into health promotional tools that allow better risk communication.
Introduction

Unhealthy behaviors are putatively responsible for about half the global all-cause mortality.[1–3] Four health behaviors, namely unhealthy diet, tobacco smoking, physical inactivity, and alcohol consumption account for an extensive proportion of deaths.[2, 4] Although their role in major non-communicable diseases is well established, these health behaviors are prevalent in populations.[1, 5]

As lowering the prevalence of unhealthy behaviors is thought to decrease the proportion of premature mortality[6], disease prevention efforts are increasingly focusing on health literacy.[7] Despite the surge in the availability of health information, unhealthy behaviors are, however, increasing, even in some developed countries.[8] According to a study from the Netherlands, although participants appeared to have an understanding of disease causation, they did not have an adequate perception of risk.[9]

Risk perception is crucial to the process of behavioral change. Inducing a strong willingness to change behavior in an individual often requires a shift in their perception of risk, which depends not only on health information, but also on the way this information is conveyed and presented to that particular individual.[10] The importance of risk communication in the induction of individual's behavioral change is well established, and some forms of presentation of risk were found to favor more efficient improvement in risk perception than others.[11] An example would be that, albeit communicating risk as a numeric probability is generally encouraged, numeric health information presented in a too precise form can be challenging for individuals to comprehend and believe. Risk probabilities presented in the form of integers had higher ratings of perceived reliability than those with one or more decimal places.[12]

Different circumstances call for different risk communication strategies. For example, risk communication related to a future outcome was found to be more convincing if conveyed in a form that shows how the future risk is influenced by behavior modification.[13] In such circumstances, risk communications that carried messages implying both threat and health benefit were associated with the highest efficacy.[14] Also, tailoring the communicated risk to the profile of the individual allows not only more accurate estimates of risk, but also better motivation to take action.[15] The concept of personalized risk assessment is particularly useful when it comes to communicating the risk of multiple health behaviors. Personalized risk assessment allows individuals to compare their risk to different risk profiles which might significantly affect risk perception and decision-making.[16]

Our study assesses the risk of all-cause mortality attributed to the effects of the four major health behaviors and develops a simple paper-based risk chart that allows personalized risk communication and comparisons across different risk factors. The study draws from a cohort of Finnish middle-aged men; this age category represents 16% of Finnish society.[17] In middle-age, the cumulative effects of childhood and adult behavioral and socio-environmental risk factors start manifesting as common chronic diseases.[18] From a health promotional perspective, middle-age is a favorable target for interventions on health behaviors, with an appreciable potential to enhance health outcomes.[19]
Methods

Data source

We based our study on the population-based Kuopio Ischaemic Heart Disease Risk Factor Study (KIHD) which has followed up 2682 Finnish men aged 42–60 years after their initial examination between March 1984 and December 1989.[20] KIHD is continuously updated with data on the participants’ health status from various Finnish registries.[21]

After excluding subjects with missing values (n = 10), subjects with a zero level of physical activity (less than 15 MET-hours per year) (n = 67), and subjects who were abstinent from alcohol at baseline (n = 370), we retained 2235 participants for the analysis. The approach of excluding subjects with an exposure level of zero is justifiable when the unexposed are likely to constitute a population that differs considerably from the exposed due to factors other than the exposition or the controlled variables. The analyzed subjects have been followed-up for a median of 28 years and a maximum of 35 years by the end of 2018.

Variables measurement

In KIHD, each participant had their baseline variables measured at the beginning of their enrollment.[20] Quality of diet, tobacco smoking, leisure-time physical activity, and alcohol drinking constituted our input variables of interest. Diet was assessed based on 4-day food records.[22] The quality of diet was then evaluated using a slightly modified Baltic Sea Diet Score as an indicator of healthy Nordic Diet (HND). The index, considered as a validated measure of diet quality based on commonly consumed foods in Nordic countries, summarizes six foods and food group variables and three nutrient variables (supplementary material 1).[23] We excluded alcohol from the healthy HND index, because we used alcohol consumption as a separate factor. Physical activity was assessed by several methods including self-recordings and interviews. Leisure-time physical activity was computed in hours of metabolic-equivalent of task (MET-hour).[24] Tobacco smoking status and alcohol consumption level were assessed through self-reported questionnaires. Waist circumference was measured by a study nurse in baseline examination.

As additional covariates, we included age, body mass index (BMI), and morbidity. To evaluate morbidity status at baseline we computed the Charlson Comorbidity Index (CCI) based on self-reported data regarding previous health events.[25]

The outcome of interest was all-cause mortality. Cause of Death Registry served as the source of information on the mortality status (Statistics Finland, License number: TK-53-1770-16). The Finnish equivalent of the social security number was used to link the participants to their mortality status in the registry. Period at risk was defined in days from baseline either to the date of death or until the end of follow-up in December 31, 2018.

Data analysis
We used Cox regression model[26] to model the effects of health behaviors on mortality. The initial model included age, BMI, and smoking status as categorical covariates and period at risk, CCI, HND, physical activity, and alcohol consumption as continuous covariates. Computation was performed by means of R programming language V4.0.0. The Cox regression model was built and diagnosed with the R package: Survival Analysis[27] V3.1, and receiver operator characteristic (ROC) curves were generated using the R package: Risk Regression.[28]

We used Chi-square and Mann Whitney U tests to compare participants' baseline characteristics in respect to the survived and dead participants. We used ROC curves to compute the area under the curve (AUC) for discriminatory power of the models.

We used the Schoenfeld method[29] to test the proportional hazard assumption for the global model and individual variables. The initial model violated the proportional hazard assumption. However, excluding CCI and physical activity, and converting smoking status from a three levels (never smoker, previous smoker, current smoker) factor to a two-level (nonsmoker, smoker) factor slightly improved the global assumption of the model and its individual variables at the expense of a slight reduction in concordance and discriminative accuracy (supplementary material 2). In addition to the model's assumption considerations, as physical activity did not markedly affect the factors’ effect estimates [Hazard Ratio (HR) 1.05, 95% confidence interval (CI) 0.92–1.19 for each unit of 10 MET-hour leisure-time physical activity per day], and as survivors and non-survivors did not differ in baseline leisure-time physical activity (Table 1), and for more simplicity in presenting the risk chart for usage, we decided to omit leisure-time physical activity from the final model analysis.

The final Cox regression model included smoking status (nonsmoker, smoker), alcohol consumption (units of 100 g/week), and HND index (a score ranging from 1 to 24) as main covariates. Additional covariates were: age (42–47, 47–52, 52–57, and 57–62) and BMI (normal weight ≤ 25, slight overweight 25–27.5, overweight 27.5–30, and obese ≥ 30 kg/m²). As only four study participants were underweight (BMI ≤ 18.5), we joined them to the normal weight category. The model also accounted for multiplicative interactions between BMI categories and smoking status.

**Results**

Within the 34.8 years of follow-up (median time 27.8 years), 1395 out of the 2235 study participants died. The median age of death was 75 years with a minimum of 45 years. The oldest study participants were 92 years old at the end of follow-up, December 31, 2018. At baseline, those who survived the entire follow-up period were younger, less frequently smokers, less frequently obese, had a healthier diet, and consumed less alcohol (p-value < 0.001 in each case) (Table 1).
Table 1  
Baseline characteristics of the study population

|                                | Survivors | Non-Survivors | Total   | P-values* |
|--------------------------------|-----------|---------------|---------|-----------|
| Number of participants (%)     | 840 (38)  | 1395 (62)     | 2235    | < 0.001   |
| Age a                          | 50.4 (5.6)| 54.5 (4.3)    | 52.9 (5.2)| < 0.001   |
| Age category (%)               |           |               |         |           |
| 42–47                          | 219 (26.1)| 80 (5.7)      | 299 (13.4)| < 0.001   |
| 47–52                          | 191 (22.7)| 116 (8.3)     | 307 (13.7)|           |
| 52–57                          | 380 (45.2)| 279 (20.0)    | 329 (14.7)|           |
| 57–62                          | 50 (6.0)  |               |         |           |
| BMI category (%)               |           |               |         |           |
| normal weight ≤ 25             | 322 (38.3)| 392 (28.1)    | 714 (31.9)| < 0.001   |
| slight overweight 25–27.5      | 268 (31.9)| 299 (21.4)    | 442 (19.8)|           |
| overweight 27.5–30             | 143 (17.0)| 277 (19.9)    | 384 (17.2)|           |
| obese ≥ 30                     | 107 (12.7)|             |         |           |
| Smokers (%)                    | 187 (22.3)| 581 (41.6)    | 768 (34.4)| < 0.001   |
| Healthy Nordic Diet index a    | 12.6 (3.8)| 11.6 (4.0)    | 12.0 (4.0)| < 0.001   |
| Alcohol consumption in g/week a| 68.8 (95.2)| 97.5 (163.5) | 86.7 (142.4)| < 0.001   |
| Leisure-time physical activity in MET-hours/day a | 4.7 (3.6) | 4.8 (4.3) | 4.8 (4.0) | 0.094 |
| Charlson Comorbidity Index a   | 0.5 (1.0) | 0.9 (1.2)     | 0.8 (1.1) | < 0.001   |
| Follow-up time in years a      | 31.6 (1.6)| 19.5 (8.8)    | 24.1 (9.1)| < 0.001   |

* results presented as mean (SD)  
* Chi-square and Mann Whitney U tests to compare survived and dead participants

The Cox model found associations of tobacco smoking, unhealthy diet, and alcohol consumption with mortality (Fig. 1). Tobacco smokers had the HR of 2.72 (95% CI 2.22–3.33) in contrast to nonsmokers. Each 5 units increase in healthy HND index was associated with 12% lower risk of mortality (HR 0.88,
95% CI 0.82–0.94). Each increase of 100 grams per week in alcohol intake associated with a 13% increase in mortality risk (HR 1.13, 95% CI 1.10–1.17). BMI categories “slight overweight”, “overweight”, and “obese” were also linked to a higher risk of mortality in contrast to “normal weight” with HRs of 1.29 (95% CI 1.06–1.57), 1.63 (95% CI 1.33–2.00), and 1.96 (95% CI 1.59–2.40), respectively. The interaction of BMI categories with smoking associated with a protective effect on mortality risk with HRs of 0.67 (95% CI 0.51–0.88), 0.59 (95% CI 0.43–0.80), and 0.57 (95% CI 0.41–0.80) for the respective interaction of “slight overweight”, “overweight”, and “obese” with smoking. In other words, when smoking and increased BMI co-existed in the same study participant, as they interacted with each other, their individual effects on mortality risk were lower than when they did not co-exist. In term of goodness of fit and accuracy, the model had a Likelihood ratio test of 598.8 (p-value < 0.001), a concordance index of 0.69 (SE 0.007), and an AUC of 72.8 (95% CI 70.8–74.9) (supplementary material 2).

To better illustrate the effect of different combinations of health behaviors on mortality risk we computed a relative risk score of mortality (RRSM) for a generated series of profiles with predefined parameters. We used the model to estimate a prediction of the absolute mortality risk for each of the theoretical profiles. The RRSM of each dummy profile was then computed as the relative risk to the absolute mortality risk of the profile with the ideal health behavior. Therefore, the RRSM represents a relative measure of the mortality risk associated to the combined effects of unhealthy behaviors regardless of follow-up time.

The predefined parameters represent a variety of health behavioral profiles that were determined by different levels of the model’s variables. The chosen levels of HND index correspond to minimum, 1st quartile, 3rd quartile, and maximum values of HND index in the cohort population. The chosen levels for alcohol consumption correspond to 2nd percentile, median, and 98th percentile values in the cohort population. We focused on these values to illustrate contrasts. The ideal health behavioral profile, which is attributed an RRSM of 1, corresponds to a normal weight 42–47 years old person who does not smoke, has a HND index of 24, and consumes 1 gram of alcohol per week. RRSMs of other health behavioral profiles represent risks that are relative to this reference ideal profile. Age-adjusted RRSMs are reported in supplementary material 3.

Rounded RRSMs at different ages are presented in Fig. 2. Throughout age categories, the chart shows an inverse relationship between HND index and RRSM, and a direct relationship between alcohol consumption and RRSM. Smoking was clearly associated with higher RRSMs across the profiles. The chart also shows a trend of risk increase with higher values of BMI. Moreover, the risk associated to combined smoking and high BMI was lower than the product of risks associated to smoking and BMI separately, illustrating a dysergic smoking-BMI interaction.

An overweight smoker aged 42–47 who consumes 475 g of alcohol per week and has a poor-quality diet (HND 1), has nearly the same RRSM as a normal weight nonsmoker aged 57–62 who has a healthy diet (HND 24) and drinks less heavily (≤ 43 g of alcohol per week). Irrespective of BMI category, a 42–47 years old smoker with a low-quality diet (HND 1) has nearly the same RRSM as a 52–57 years old person who has a better-quality diet and who does not smoke. These examples conveyed, in terms of age,
the risk associated to the combined effects of different health behaviors. Also, if a person for example has a low quality diet (HND 1), they can be shown that the risk associated to their diet is equivalent to the risk associated to drinking 475 g of alcohol per week, or close to the risk associated to obesity (in nonsmokers), in comparison to the ideal health behavioral profile (supplementary material 3 for more precise estimates).

The mortality risk associated to unhealthy behaviors directly translates into life-years lost as illustrated in Fig. 3, which presents estimated median ages of survival for different combinations of predictors’ parameters. The estimates are valid for the age category 57–62 as estimated median survivals are not available for all health behavioral profiles in other age categories.

The ideal health behavioral profile had an estimated median age of survival of 90.72 years (95% CI 88.49–93.38). The interaction BMI-smoking also appeared in median ages of survival; the deleterious effect of smoking was clearly more marked in normal BMI category than in all others. The lowest estimates of median survival age (around 70 years) were found in profiles of heavy drinking (475 g of alcohol per week) smokers who had a low-quality diet (HND 1), with little variation across BMI categories.

The combined effects of obesity, smoking, poor quality diet (HND 1), and high level of alcohol consumption (475 g/week) might be associated with nearly 10 folds the mortality risk of a normal weight individual engaged in healthy behavior regardless of age (Fig. 2), and a loss of about 20 years in median survival (Fig. 3).

Discussion

In this study, we examined the effects of health behaviors and their different combinations on all-cause mortality in 2235 middle-aged men. We aimed to present the results of our investigation in a way that allows simple and practical communication of the risk of combined unhealthy behaviors on mortality. Our focus on modifiable risk factors pinpoints the reversibility of the conveyed risk and elucidates the potential of exposure reduction. Tobacco smoking, alcohol drinking, and unhealthy dietary habits were associated with an increase in mortality risk. As in other studies that considered the relation between different health behaviors and survival[4], the association between mortality and tobacco smoking per se was particularly strong, with an HR over 2.7.

With linked data from electronic health registers and death certificates, the population cohort approach that we adopted in our study is one of the leading methods in estimating the risk of unhealthy behaviors. As in Manuel et al. (2016), we found that the effect of diet on mortality was not as important as that of tobacco smoking.[4] High BMI, also an important influencer of chronic disease and health outcomes,[30] was associated with a considerable increase in mortality per se. A meta-analysis of BMI and all-cause mortality association found that mortality increased with each BMI level: (15-18.5), (18.5–20), and (25-27.5).[31] This conclusion suggests that stratifying our normal weight BMI category might allow a more appropriate definition of the reference category for BMI. Also, Freedman et al. (2006)[32] found that
smokers with a BMI > 35 were at a substantially high cardiovascular mortality risk. Our model could thus benefit from a stratification of the obese BMI category.

Regression without interaction already accounts for the fact that multiple factors reduce lifespan independently. However, when an interaction between two variables is significant, it illustrates either a synergic effect or, its opposite, a dysergic effect between them. In our study, the only statistically significant interaction we found was between smoking and BMI. We found that a high BMI was associated with a lower effect size of tobacco smoking on mortality risk. It is common for smokers to have lower BMIs than nonsmokers. That is often explained by the appetite-suppressive effect of nicotine. [33] Our smoker participants also appeared to have lower BMIs (supplementary material 4). A possible explanation of the sense of the BMI-smoking interaction could be that higher BMIs might be associated with a lower quantity of cigarette smoking. An increased waist circumference in smokers, as Lv et al. (2015)[34] suggested, can be another possible explanation of the smoking-BMI interaction; in our study, smokers were found to have a larger waist circumference than nonsmokers of the same high BMI category (supplementary material 5). Controlling for waist circumference and magnitude of tobacco smoking exposure might improve our model and bring more clarity to the nature of the BMI-smoking interaction. Sedentary behavior is another factor that might influence the relation between BMI, smoking and mortality; smokers were found to have higher rates of sedentary behavior than nonsmokers.[35] Although our study participants’ leisure time physical activity level differed significantly by smoking status (supplementary material 4), suggesting a more marked sedentary behavior in smokers, as sedentary behavior is a risk factor for both obesity and adverse health outcomes, this association would, however, rather increase the magnitude of mortality risk linked to the effect of combined smoking and high BMIs.

In addition to findings related to single risk factors, our study clearly shows that combining the effects of unhealthy behaviors on all-cause mortality yields considerably high HRs. The combined effect of tobacco smoking, alcohol drinking, and unhealthy diet, especially when associated with obesity, increased the mortality risk by nearly 10 folds, in comparison to a normal-weight individual free of unhealthy behaviors. To allow comparisons of different combinations of health behaviors and individual health behaviors of different natures, inspired by the work of Jackson et al. (1993)[36] and Pyörälä et al. (1994)[37], we created a simple paper-based risk-chart presented as Fig. 2. Such comparisons permit healthcare workers to better communicate the risk of unhealthy behaviors, based on an individualized assessment of risk. The chart aimed for information simplicity and risk comparability.

While many obstacles such as the preferability of qualitative over quantitative information and the abstract nature of the concept of risk hamper personalized risk information[38, 39], individualized prediction models are important supports of health information and effective vectors of risk communication, especially when there is a need to evaluate combined risk factors.

**Strengths And Limitations**
Our study has many strengths. It is based on a cohort of high-quality scientific standards with a comprehensive baseline examination and a follow-up time that is long enough to allow proper survival studies on chronic diseases. Moreover, the registries used for the ascertainment of cases are dependable and there was no loss to follow-up in the cohort. As inaccuracy of forecasts and overfitting are common limitations to the internal and external validity of clinical prediction models, we evaluated the ROC curve of our model and obtained an AUC of 72.8 (95% CI 70.8–74.9), which seems decent considering that we aimed for assessment simplicity more than prediction power.

However, we also recognize some limitations. The proportional hazards assumption contradicts with the changing nature of health behaviors. Baseline measurements are unable to reflect previous rates, future rates, and patterns of exposure. A model that supports time-varying covariates might provide more accurate results.

The restrictions related to our demographic, namely sample size, age group, gender, and regional nature seriously hinder the generalizability of our results and the usage of the risk-chart. Also, data on our study participants’ physical activity was too left skewed to allow sufficient statistical power. While BMI can partly reflect physical activity level, its role in chronic disease and frailty remains indisputable. Finally, HND index would need a separate chart to be assessed, and considering that HND index is region-specific, the external validity and practicality of the study might be further limited.

**Conclusion**

We have developed a paper-based risk chart that illustrates the all-cause mortality risk associated to three main health behaviors. The chart appeared useful in comparing risk across predictors of different natures as well as in illustrating the concept of interaction. As unhealthy behaviors tend to occur concomitantly, their effects on mortality should be assessed as combined and in relation to each other to enable more reliable mortality predictions. The simple presentation of such research findings relevant to health promotion might constitute an efficient individualized risk communication channel and create an opportunity for a change towards a healthier behavior.

**Declarations**

**Ethics approval and consent to participate**

The KIHD study protocol was approved by the Research Ethics Committee of the University of Kuopio and have been performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

**Availability of data and materials**
The department of Public Health of the University of Eastern Finland can be approached with a reasonable request for permission to obtain the data. The corresponding author, Mounir Ould Setti, can provide the computing code required to replicate the results reported in the submission.

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**Conflicts of Interest**

The authors declare no conflict of interest to disclose.

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**Figures**

| Variable                  | N    | Hazard ratio          | p-value |
|---------------------------|------|-----------------------|---------|
| Age category              |      |                       |         |
| 42–47                     | 299  | -                     | Reference|
| 47–52                     | 307  | —                     | 1.72(1.29, 2.28) <0.001 |
| 52–57                     | 1300 | ■                     | 3.75(2.98, 4.72) <0.001 |
| 57–62                     | 329  | ■                     | 6.98(5.42, 8.99) <0.001 |
| BMI                       |      |                       |         |
| normal weight             | 714  | ■                     | Reference|
| slight overweight         | 695  | ■                     | 1.29(1.06, 1.57) 0.010 |
| overweight                | 442  | ■                     | 1.63(1.33, 2.00) <0.001 |
| obese                     | 384  | ■                     | 1.96(1.59, 2.40) <0.001 |
| Smoking                   |      |                       |         |
| nonsmoker                 | 1467 | ■                     | Reference|
| smoker                    | 768  | ■                     | 2.72(2.22, 3.33) <0.001 |
| Alcohol intake            | 2235 | ■                     | 1.13(1.10, 1.17) <0.001 |
| HND                       | 2235 | ■                     | 0.88(0.82, 0.94) <0.001 |
| Interaction - slight overweight:smoker | 2235 | ■ | 0.67(0.51, 0.88) 0.005 |
| Interaction - overweight:smoker | 2235 | ■ | 0.59(0.43, 0.80) <0.001 |
| Interaction - obese:smoker | 2235 | ■ | 0.57(0.41, 0.80) 0.001 |

**Figure 1**

Forest plot of the Cox model’s hazard ratios Note. Alcohol intake, units of 100 g/week; HND, Healthy Nordic Diet index (units of 5)
**Figure 2**

Relative risk score of mortality (RRSM) in different profiles of health behaviors and different age categories.
Figure 3

Estimated median ages of survival for different health behavioral profiles – computed for age category 57-62 Note. HND, Healthy Nordic Diet; rare drinking corresponds to a level of alcohol consumption of 1 g per week; heavy drinking corresponds to a level of alcohol consumption of 475 g per week; whiskers illustrate the 95% CI.
Supplementary Files

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- Supplementarymaterial.docx