Effort–reward imbalance at work, overcommitment personality and diet quality in Central and Eastern European populations

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ABSTRACT

This study aims to investigate the associations between work stress defined by the Effort–Reward Imbalance (ERI) model and diet quality, and to examine the potential role of overcommitment (OC) personality in ERI–diet relationships. A cross–sectional study was conducted in random population samples of 6,340 men and 5,792 women (age 45–69 years) from Czech Republic, Russia and Poland. Dietary data were collected using Food Frequency Questionnaire. Healthy Diet Indicator (HDI) was constructed by 8 nutrient/food intakes (HDI components) to reflect the adherence to World Health Organisation dietary guideline. The extent of imbalance between effort and reward was measured by effort–reward (ER) ratio; effort score was put in enumerator and reward score was multiplied by a factor adjusting for unequal number of items in denominator. Logistic regression and linear regression were used to assess the associations between exposures (ER ratio and OC) and outcomes (HDI components and HDI) after adjustment for confounders and mediators. The results showed that high ER ratio and high OC were significantly associated with unhealthy diet quality. For a 1–standard deviation (SD) increase in ER ratio, HDI was reduced by 0.030 and 0.033 SD in men and women; for a 1–SD increase in OC, HDI was decreased by 0.036 and 0.032 SD in men and women. Modifying role of OC in ERI–diet relationships was non–significant. To improve diet quality at workplace, a multiple–level approach combining organizational intervention for work stress and individual intervention for vulnerable personality is recommended.

Introduction

Health behaviours – such as unhealthy diet, alcohol drinking, smoking and physical inactivity have been found to increase risks of chronic diseases[1]. High intakes of saturated fat and cholesterol are associated with high levels of low–density lipoprotein fraction of cholesterol and triglyceride, which increase risks of coronary heart disease and atherosclerosis. In contrast, high intakes of fruit and vegetable reduce risks of coronary heart disease, stroke, hypertension, diabetes and cancer[2]. Diet quality is defined by the adherence to dietary guidelines associated with health outcomes like chronic diseases[3]. Diet is influenced by a wide range of psychosocial factors; in particular, chronic stress was found to influence individual's psychological and physiological responses, resulting in food choice towards high–fat and high–carbohydrate content[4].

Work stress, a common type of chronic stress in adults, has been measured comprehensively after theoretical development of the Demand–Control (DC) model and the Effort–Reward Imbalance (ERI) model. The DC model proposes that job task profiles defined by low control and high demand (job strain) may elicit sustained stress reactions. Job strain was found to predict unhealthy diet[5,6,7,8], as well as other health behaviours like drinking, smoking, and physical inactivity[9,10,11].

In the ERI model, work stress is defined by the violation of social reciprocity in terms of high extrinsic effort (heavy workload, interruption, responsibility, overtime, physical demands and increasing demands) and low reward (salary, esteem, promotion prospect and job security). The ERI model has been found to predict obesity, high blood cholesterol, hypertension, diabetes, and cardiovascular diseases[12,13,14]. ERI is suggested to influence above chronic diseases through psychobiological processes (autonomic, endocrine and immune activation) and health behaviours[15]. There have been empirical studies showing that high ER ratio predicted health behaviours – drinking, smoking and physical inactivity[16,17,18], but evidence for the link between ERI and diet is lacking. As mentioned earlier, evidence shows that unhealthy diet increases risks of chronic diseases; diet may mediate the impacts of ERI on chronic diseases. Thus, it is plausible to suggest a potential link between ERI and diet.

The ERI model incorporated a personality construct – overcommitment (OC), thereby enabling examination of the potential role of personality in work stress–outcome relationships. OC reflects a cognitive–motivational pattern of coping with

demands characterized by high need for control, excessive striving at work, and inability to withdraw from work; high OC persons tend to maintain excessive effort under inadequate reward[19]. The concept of OC is similar to "workaholism" – being overly concerned about work, to be driven by strong and uncontrollable work motivation, and to spend so much energy and effort into work that it impairs relationships, leisure activities and health[20].

OC was primarily assumed to have main effect on health outcomes (high OC increases the risk of poor health) or modifying effect on ERI–outcome relations (those with high ERI and high OC have an even higher risk of poor health). The review of 45 studies found that main effect of OC was supported in 17 out of 27 studies (63%), but modifying effect was supported in only 3 out of 12 studies (25%)[13]. However, very little literature has examined the potential role of OC in the associations between ERI and health behaviours. Two studies have reported negative findings on main effect of OC on smoking, without testing modifying effect of OC[21,22].

In Central and Eastern European (CEE) countries, diet is characterized by high consumption of saturated fat and sugar but low intake of fruit and vegetable[23]. Diet is suggested to increase risks for chronic diseases, contributing to mortality gap between Western Europe and CEE[24]. Socioeconomic and political transformations occurred in CEE since 1989; dramatically changing working environments result in the highest levels of job insecurity among European countries[25]. Work stress defined by the DC and ERI models has been found to predict cardiovascular diseases, poor health, and high alcohol consumption in CEE[26,27,28]. Thus, to study the ERI–diet associations would contribute to deeper understanding on the mechanisms via which work stress influences chronic diseases.

Based on the identified research gaps, the aims of this study are: (1) to investigate the associations between ERI and diet quality in the CEE populations, and (2) to examine the potential role of OC personality in ERI–diet relationships.

Methods

Study design and population

The data come from the HAPIEE study (Health, Alcohol and Psychosocial factors In Eastern Europe). Random samples of 45–69 year–old men and women were selected from population registers in 6 towns (Havízov/Karviná, Jihlava, Ústí

nad Labem, Liberec, Hradec Králové, and Kromezíz) in the Czech Republic, Krakow in Poland, and Novosibirsk in Russia from 2002 to 2005. From 28,947 subjects recruited (overall response rate 61%), ineligible people – retired (14,060), unemployed (1,178) and housewives (307) were excluded. Next, those with missing values in employed status (131), exposure variables (518) and dietary outcomes (621) were excluded; additionally, subjects with missing values for more than 15 items in Food Frequency Questionnaire were excluded. The final sample consisted of 12,132 subjects (6,340 men and 5,792 women).

Each participant independently completed a structured questionnaire and had a medical examination. This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects were approved by the ethical committees in University College London and all three countries. Written informed consent was obtained from all subjects. The methodology of the HAPIEE study was described in detail by Peasey et al[29].

Dietary outcomes

Dietary data were collected using the Food Frequency Questionnaire (FFQ) adapted from Willett et al and used previously in the Whitehall II Study[30]. Due to inclusion of country–specific dishes, Czech, Polish and Russian FFQs consisted of 136, 148 and 147 food items, respectively. For each food item, a country–specific portion size was specified and its nutrient content was based on the McCance and Widdowson Food Composition Data and local food composition tables[31]. Subjects were asked how often they had consumed that amount of the item during the last 3 months, with 9 responses ranging from "never or less than once per month" to "6 or more times per day". Daily intakes of nutrients were calculated by multiplying the frequency of food consumed per day with the nutrient content of the specified portion size. This methodology was described in detail by Boylan et al[23].

Healthy Diet Indicator (HDI), a diet quality score, was constructed to reflect the adherence to the World Health Organisation (WHO) dietary guideline for prevention of chronic diseases (2003)[32]. Huijbregts et al developed this approach to identify diet quality associated with chronic diseases[33]. According to the WHO guideline, 8 nutrient intakes or food intakes were selected: (1) percentages of total energy intakes without energy provided by alcohol (as alcohol consumption differed considerably between countries) from saturated fatty acid, polyunsaturated fatty acid, protein,

total carbohydrate, and free sugar; (2) nutrient intakes of non–starch polysaccharide and cholesterol; (3) food intakes of fruit and vegetable. Next, a dichotomous variable was generated for each nutrient or food intake; if one's intake was within the WHO recommended range this variable was coded as 1 (healthy intake), otherwise it was coded as 0 (unhealthy intake). The HDI score was the sum of 8 dichotomous variables (HDI components), so each subject has a score value ranging from 0 to 8 (Table 1).

Monounsaturated fatty acid was not included because the WHO guidelines did not take them into account. Sodium chloride was not included, as only information on sodium content in foods was available but unknown amount of salt was added during preparation of meals or at the table.

Exposure variables

The ERI model is operationalized as a standardized self–report measure containing 23 items, defining 3 unidimensional scales: extrinsic effort, reward, and overcommitment with each item rated on a 5 point (extrinsic effort and reward) or 4 point (overcommitment) scale. Extrinsic effort is measured by 6 items on demanding aspects of work environment: quantitative load, qualitative load, physical load, and increasing load. Reward is assessed by 11 items on financial reward, esteem reward, promotion prospect and job security[19]. The extent of imbalance between extrinsic effort and reward is measured by effort–reward (ER) ratio; extrinsic effort score is put in enumerator, and reward score is multiplied by a correction factor (6/11) adjusting for unequal number of items in denominator. The ERI questionnaire was translated into all 3 languages, back translated to confirm accuracy of original translations and validated in the pilot of HAPIEE study[34].

Overcommitment (OC) is assessed by 6 items and its score is created by summing them up: (1) I get easily overwhelmed by time pressures at work. (2) As soon as I get up in the morning I start thinking about work problems. (3) When I get home, I can easily relax and switch off work. (4) People close to me say I sacrifice too much for my job. (5) Work is still on my mind when I go to bed. (6) If I postpone something that I was supposed to do today, I have trouble sleeping at night.

Potential confounders

A variable is considered a confounder if it meets three criteria: it must be a risk

factor for the outcome; it must be associated with the exposure in the study population; it cannot be a mediator in the causal path between the exposure and the outcome[35]. Potential confounders were selected from the HAPIEE study if they were known risk factors for poor diet: demographics (age and marital status) and socioeconomic indicators (educational level, occupational class, and deprivation)[36]. These variables were significantly ($p < 0.05$) associated with at least one exposure variable.

For age, subjects were 45–69 years old at the baseline. Marital status was coded as: (1) married or cohabiting, (2) single, and (3) divorced or widowed. Education was categorized as: (1) primary or less, (2) vocational, (3) secondary, and (4) university. Occupational grade was categorized as: (1) manager or professional, (2) non–manual workers, and (3) manual workers. Material deprivation was assessed by 3 questions on how often subject's household had difficulties to buy enough food or clothes and to pay bills for electricity, heating and housing. The answers to each question were coded from "never" (0) to "always" (3). Deprivation score, the sum of 3 responses ranged from 0 to 9, was dichotomized into low (0 to 3.9) and high (4 to 9).

Potential mediators

Potential mediators (depression, problem drinking, and current smoker) in the causal path between the exposure and the outcome were selected from the HAPIEE study. ERI is found to predict depression[34], alcohol drinking[16], and smoking[17], all of which may influence diet. Depression is associated with low motivation for planning and eating healthy diet, decreased appetite or overeating[37]. High alcohol consumption results in restraint from eating or overeating[38]. Although smoking suppresses appetite temporarily, smokers tend to consume more saturated fatty acid, more sugars, and less fruit and vegetable[39].

Depressive symptoms were measured by Center for Epidemiologic Studies Depression scale (CESD), consisting of 20 self–reported items ranged from 0 to 60; CESD >= 16 was defined as having clinically relevant depressive symptoms[40]. Problem drinking was screened by CAGE questionnaire consisting of 4 items with 2 responses (0= no, 1= yes). With a cut–off point of 2, sensitivity and specificity are high in relation to alcohol abuse and dependence[41]. Smoking status was measured by the question: "do you smoke cigarettes?" Those with the first 2 answers (yes,

regularly, at least one cigarette a day; yes, occasionally, less than one cigarette a day) were classified as current smokers; others (no, I smoked in the past but I stopped; no, I have never smoked) were classified as current non–smokers.

Statistical analysis

Descriptive characteristics in the sample were analyzed by country and by gender. Crude associations between exposure variables (ER ratio and OC) and HDI were not very different across country–specific strata and across gender–specific strata (p for heterogeneity > 0.1). Data for three countries were pooled for further analyses, but men and women were analyzed separately as most studies that examined the effects of work stress on health behaviours and diet[5,6,7,8].

Binary logistic regression was used to assess the associations between exposure variables and 8 HDI components (dichotomous outcomes), respectively, after adjustment for confounders and potential mediators. Odds ratio (OR) represents the odds of an outcome occurring (e.g., healthy intake) given the exposure, compared to the odds of the outcome occurring in the absence of that exposure. For continuous exposures (ER ratio and OC), OR per unit is the odds of having healthy intake of the HDI component for a 1–unit increase in the exposure; OR per SD is the odds of having healthy intake of the HDI component for a 1–SD increase in the exposure.

The associations between exposure variables and HDI (a continuous outcome) were evaluated by linear regression with the following four steps: these associations were adjusted for confounders and ER ratio in Model 1, adjusted for confounders and OC in Model 2, adjusted for confounders, ER ratio and OC in Model 3, and additionally adjusted for potential mediator (depression, problem drinking and current smoker) in Model 4. Beta (β) coefficient reflects change in the outcome for a 1–unit increase in the exposure. Standardized β coefficient reflects change of standard deviation (SD) in the outcome for a 1–SD increase in the exposure; the same standardized units allow for comparing relative strength between different exposure variables. For model fit, R^2 explains how much of variation of the outcome is explained by independent variables in the model.

To evaluate modifying role of OC in ERI–HDI relationships, linear regression was conducted for HDI regressed by OC, ER ratio, and the interaction term between OC and ER ratio after adjustment for confounders and mediators. By comparing log likelihoods of the models with and without this interaction term, likelihood–ratio

(LR) test was adopted to test the significance of the interaction term. All analyses were conducted with STATA 12 software (StataCorp LP, College Station, Texas, USA).

Results

In this sample of 6,340 men and 5,792 women, the mean age is 55.0 years (SD 6.0) in men and 53.0 years (SD 5.3) in women. Descriptive statistics for confounders and mediators are presented by country and by gender in Table 2.

Table 3 shows descriptive characteristics of dietary outcomes by country and by gender. The means of overall HDI scores are highest in the Czech Republic and lowest in Poland in both men and women. By comparing the percentages of total energy in Table 3 with the WHO recommended ranges in Table 1, less than 10% of subjects consumed WHO suggested ranges of saturated fatty acid and free sugars; most of them consumed too much. Only 10–20% of subjects met WHO recommended ranges of total carbohydrate and protein; most of them consumed too little total carbohydrate but too much protein.

In Table 4, logistic regression is used to assess the associations between ERI and 8 HDI components, respectively, after adjustment for confounders and mediators. In men, higher ER ratio is marginally and significantly ($p < 0.1$) associated with less healthy intakes of free sugars and cholesterol. In women, higher ER ratio is significantly ($p < 0.05$) related to less healthy intakes of saturated fatty acid and non-starch polysaccharide. For example, OR of having healthy intake of saturated fatty acid is 0.84 ($p= 0.019$) for a 1–SD (0.25) increase in ER ratio in women.

The associations between OC and 8 HDI components after adjustment for confounders and mediators are evaluated by logistic regression (Table 4). In men, higher OC is at least marginally and significantly ($p < 0.1$) associated with less healthy intakes of saturated fatty acid, polyunsaturated fatty acid, free sugars, and fruit and vegetable. In women, higher OC is at least marginally and significantly associated with less healthy intakes of saturated fatty acid, polyunsaturated fatty acid, and non-starch polysaccharide. For instance, OR of having healthy intake of polyunsaturated fatty acid is 0.90 ($p= 0.005$) for a 1–SD (3.56) increase in OC in women.

In Table 5, linear regression is used to assess the associations between exposure

variables and HDI. In Model 1 (adjusted for confounders and ER ratio), for a 1–SD increase in ER ratio, HDI is decreased by 0.052 (standardized β) and 0.042 SD in men and women, respectively. In Model 2 (adjusted for confounders and OC), for a 1–SD increase in OC, HDI is decreased by 0.056 and 0.052 SD in men and women, respectively. In Model 3 (adjusted for confounders, ER ratio and OC) and Model 4 (additionally adjusted for mediators), the ERI–HDI associations and OC–HDI associations attenuate substantially but remain significant ($p < 0.05$). Men is taken for example, standardized β coefficients for ERI–HDI associations are –0.052, –0.039 and –0.030 in Model 1, 3 and 4, respectively; standardized β coefficients for OC–HDI associations are –0.056, –0.044 and –0.036 in Model 2, 3 and 4, respectively.

In Model 4, for a 1–SD increase in ER ratio, HDI is reduced by 0.030 and 0.033 SD in men and women. For a 1–SD increase in OC, HDI is reduced by 0.036 and 0.032 SD in men and women, respectively. When effort and reward subscales are entered separately into regression model, effort is negatively associated with HDI in men (standardized $\beta = -0.027$, $p = 0.079$) and women (standardized $\beta = -0.036$, $p = 0.025$); reward is positively associated with HDI in men (standardized $\beta = 0.058$, $p < 0.001$) but not in women (standardized $\beta = 0.017$, $p = 0.252$).

The associations of confounders and mediators with HDI in Model 4 are shown in Table 5. In men, divorced or widowed, manual workers, high deprivation, problem drinking and current smoker are associated with low HDI ($p < 0.1$). In women, young age, high deprivation and problem drinking are associated with low HDI.

For assessing modifying role of OC in ERI–HDI relationships, linear regression is conducted for HDI regressed by OC, ER ratio, and the interaction term between OC and ER ratio after adjustment for confounders and mediators. LR test shows that the interaction term is not significant in men ($p = 0.219$) and in women ($p = 0.431$).

Discussion

To our knowledge, this large cross–sectional survey from CEE populations is the first study to provide evidence for the links between the ERI model and a range of dietary indicators. High ER ratio and high OC personality are both associated with unhealthy diet quality; modifying role of OC in ERI–diet relationships is non–significant. This study provides additional evidence for the potential role of OC in

ERI–outcome associations, an area where current literature is not entirely consistent.

Associations between effort–reward imbalance and diet quality

Our results found inconsistent effects of ERI on individual HDI components, probably reflecting gender or individual differences in dietary responses to work stress[42]. Higher ER ratio was associated with less healthy intakes of free sugars and cholesterol in men; higher ER ratio was related to less healthy intakes of saturated fatty acid and non-starch polysaccharide in women (Table 4). However, overall impacts of ERI on HDI appeared robust; for a 1–SD increase in ER ratio, HDI is reduced by 0.030 and 0.033 SD in men and women, respectively (Table 5).

These findings imply that work stress defined by ER ratio is associated with people's choice of overall diet quality, which is linked to risks of chronic diseases. There are at least two potential mechanisms linking work stress to diet based on existing evidence. In biological pathway, work stress can influence individual's physiological responses (e.g., increased activities of hypothalamus–pituitary–adrenal axis and elevated levels of cortisol and insulin), resulting in food choice towards high–fat and high–carbohydrate content[4]. In psychological pathway, work stress (viewed as primary cognitive appraisal – perception of severity of the threat) can affect one's problem–focused or emotion–focused coping. Engaging in risky health behaviour is an emotion–focused coping, which temporarily relieves psychological distress and distracts attention from stressful situation[43].

Additionally, our finding provided evidence supporting that the effect of ERI on diet might be partially mediated by depression, alcohol drinking and smoking, as the ERI–diet associations were substantially reduced after adjustment for these mediators. Evidence reported that high ER ratio predicted depression[34], alcohol drinking[16], and smoking[17], all of which may influence diet via mechanisms like overeating or restraint from eating[37,38,39]. In fact, direct evidence showed that high ER ratio was associated with overeating in obese men in Japan[44]. The British Whitehall II cohort study found that work stress predicted increased body weight in obese men, but reduced body weight in thin men; no corresponding effects were reported in women[45].

Despite existing evidence on the link between the DC model and diet, our findings on ERI–diet associations might strengthen the knowledge gap due to the advantage of the ERI model. The DC model reflected social concerns on industrial

workers' control in the 1970s[46]. The diminished industrial setting of working environments might reduce the prevalence of this exposure[47]. In this era of globalization, tight managerial control is shifted to flexibility, self-regulation and decentralization. The ERI model emphasizing psychosocial reward in career prospect, self-esteem and job security might be more sensitive in explaining the nature of work stress in modern occupations[48].

Associations between overcommitment and diet quality

Our results found that higher OC was significantly associated with lower HDI. OC reflects a cognitive–motivational pattern characterized by high need for control, excessive striving at work, and inability to withdraw from work. Siegrist initially developed OC as a distinct individual pattern of coping with work demands (need for control), which evolved from Type A behaviour (characterized by hostility, aggression, urgency, competitiveness and hard driving)[49]. Type A persons have high need for control over environment and tend to feel loss of control; their coping response is to assert control over environment[50].

Very little literature is available on the potential role of OC in relationships between ERI and health behaviours; two studies reported no main effect of OC on smoking without examining modifying effect of OC[21,22]. This study is probably the first to support main effect of OC on health behaviours (diet), and modifying role of OC is non–significant. However, the effect of OC on diet may be somewhat supported by previous studies demonstrating the impact of Type A behaviour (or its component hostility) on health behaviours[51]. For example, Type A behaviour was associated with high consumption of saturated fatty acid, cholesterol, and vegetable in a cohort study of 10,602 men in Northern Ireland and France [52].

Our results reported that the OC–HDI associations attenuated after adjustment for ER ratio (comparison of standardized β between Model 2 and 3 in Table 5), suggesting that the effect of OC on HDI might be mediated or confounded by ERI. Type A behaviour at adolescence was found to predict high ER ratio at adulthood[53]. Personality may influence work stress via cognitive–behavioural mechanisms: selection (e.g. Type A persons select themselves into highly competitive tasks), perception (e.g. Type A persons tend to perceive high levels of work stress), and stressor creation (e.g. Type A persons create work stressors for themselves by provoking interpersonal conflict)[54]. Thus, it is likely that high OC affects high ER

ratio which results in low HDI. If ERI is considered a mediator in the OC–HDI causal path, it would not be viewed as a confounder[35].

On the other hand, our results found that the ERI–HDI associations were reduced after adjustment for OC (comparison of standardized β between Model 1 and 3 in Table 5), suggesting that the effect of ERI on HDI might be mediated or confounded by OC. In contrast to classical perspective suggesting that personality do not change, the meta-analysis found that personality continues to change moderately throughout adulthood[55]. Work stress was found to induce changes in personality[56]. Thus, it might be plausible that high ER ratio affects high OC which then influences low HDI.

By the life course approach, there might be a "bidirectional" relationship between personality (OC) and work environment (ERI) across life span; personality can shape work experience, and work experience may has moderate impact on personality[57].

Methodological issues

Several methodological issues should be considered when interpreting our results. First, FFQ is the primary method to gather dietary information from large population samples, as it is inexpensive and representative for average long–term diet. However, FFQ method tends to be semi–quantitative, rather than fully quantitative, probably resulting in overestimation or underestimation of dietary intakes[58]. Thus, assigning HDI scores may be imprecise and introduce some misclassification, but the ranking of subjects in terms of HDI should be unbiased.

Second, the validity of FFQ regarding fruit, vegetable and micronutrient intakes was found acceptable by estimating correlations with plasma biomarker in a random subsample of HAPIEE study[59]. Nevertheless, other HDI components have not been tested for validity. Third, the HDI was constructed by Huijbregts' original approach (HDI components coded as dichotomous variables). However, Jankovic et al proposed a new HDI approach which applied continuous scoring to obtain greater variation between individuals, and it may provide more precise estimation for diet quality[3].

Fourth, a cross–sectional study often has difficulty in determining the time order between the exposure and the outcome. Reverse causality that unhealthy diet may cause high levels of work stress cannot be ruled out. Albeit less likely than the

other causal direction, poor diet may elicit physiological (e.g., pro–inflammatory state) and psychological problems (e.g., depression)[60], which may render persons more sensitive to work stress. Moreover, the cross–sectional design does not allow identification of the causal chains between OC, ERI and diet; a future cohort study is needed in order to draw firm conclusion on the relationships.

Fifth, although potential confounders were adjusted in our analyses, there may be residual confounders not taken into account, leading to underestimation or overestimation of the exposure–outcome relationships. For example, chronic stressors outside workplace (e.g., work–family conflicts or family stressors) were known risk factors for unhealthy diet but unavailable in the HAPIEE study[61].

Finally, it is unclear to what extent our findings can be generalized beyond these study samples covering urban populations in Czech Republic, Russia and Poland; however, socioeconomic and health indicators suggest that these study populations approximately represent their national populations. Evidence shows that the effects of ERI on self–rated health and alcohol drinking in CEE are generally similar to those found in Western Europe[28,62]. As evidence for the ERI–diet association is lacking in existing literature, it is possible that our findings might be generalized to the European populations.

Implications for practice and policy

Workplace has emerged as an important environment for delivering behaviour change interventions targeted at diet, smoking and physical activity. Workplace may offer healthy food served at cafeterias and education on healthy diet. Sorenson et al integrated intervention to reduce exposure to occupational hazards with intervention to improve health behaviours; the rate of behaviour changes in integrated program was twice as high as that focusing on health behaviours only[63]. Since the effect of work stress on diet was found in this study, organizational interventions should address potential occupational hazards – work stress.

The strategy of organizational interventions based on the ERI model is to restore the balance between extrinsic effort and reward at work. In terms of extrinsic effort, interventions can focus on reduction of overtime work, even distribution of workload and responsibility, and provision of holidays. In terms of reward, social skill training improves supervisor's leadership behaviours, resulting in increased esteem reward. Introduction of additional benefits can increase non–monetary

reward. Provision of vocational training and steps for promotion can ensure employees' job security[64].

The association between OC personality and diet was found in our study. A meta-analysis from 36 studies found that individual interventions based on cognitive–behaviour therapy (CBT) produced larger effects than others[65]. It is plausible to suggest targeting cognitive–behaviour mechanisms via which personality can influence health behaviours. Aust et al conducted an intervention to reduce the impact of OC; this program included self–observation for perception of arousal, relaxation training, management of conflict with supervisors, and coping with anger[66]. Limm et al conducted a group prevention program to foster awareness of stress situations based on ERI model and to provide coping strategies with stressful situations; the program reduced perceived stress reactivity, sympathetic activation, and ER ratio[67]. While it is difficult to induce strong changes in personality itself, to change individual's tendency in cognition and behaviour appears practical.

Our finding of potentially bidirectional relationships between ERI and OC implies that interventions can focus on both working environments and individuals in order to disrupt cumulated effects in the reciprocal relations. Individual interventions are effective at individual–level outcomes like health behaviours, but organizational interventions have positive impacts on organizational–level outcomes like reducing exposure to work stressors. Superior results would be expected from combining individual and organizational interventions (a multi–level perspective) over a single type[68]. Organizational interventions for work stress and health behaviours can be implemented if resources are available; individual interventions for personality vulnerable to work stress can be adopted according to individual needs.

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Conflict of Interest

None.

Authorship

The authors' contributions are as follows: S. C., A. Peasey and H. P. contributed to formulating the research questions, analysing the data and writing the article. D. S. and J. C. contributed to critically revising the article for important content. M. B., S. M., A. Pajak and R. K. contributed to the study design and the data collection in three countries. All authors read and approved the final version of the manuscript.

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Table 1. Eight components of Healthy Diet Indicator

Individual HDI component:	Dichotomous value	
Nutrient or food intakes	1= Within WHO suggested range	0= Otherwise
1. Saturated fatty acid	1= < 10% of total energy	0= > 10% of total energy
2. Polyunsaturated fatty acid	1= 6–10% of total energy	0= < 6% or > 10% of total energy
3. Protein	1= 10–15 % of total energy	0= < 10% or > 15% of total energy
4. Total carbohydrate	1= 55–75% of total energy	0= < 55% or > 75% of total energy
5. Free sugars	1= < 10% of total energy	0= > 10% of total energy
6. Non–starch polysaccharide	1= > 20 g/day	0= < 20 g/day
7. Cholesterol	1= < 300 mg/day	0= > 300 mg/day
8. Fruit & vegetable	1= > 400 g/day	0= < 400 g/day

HDI, Healthy Diet Indicator; WHO, World Health Organisation.

Table 2. Descriptive characteristics of confounders and mediators by country and gender

Confounders and mediators	Czech Republic		Russia		Poland	
	Men N= 1814	Women N= 1708	Men N= 2544	Women N= 2332	Men N= 1982	Women N= 1752
Age, Mean (SD)	54.1 (5.5)	52.3 (4.7)	56.1 (6.2)	53.8 (5.8)	54.5 (5.8)	52.6 (5.0)
Marital status, N (%)						
Married or cohabiting	1527 (84.6)	1236 (72.5)	2290 (90.0)	1464 (62.8)	1802 (91.2)	1220 (69.9)
Single	53 (2.9)	45 (2.6)	64 (2.5)	135 (5.8)	68 (3.4)	156 (8.9)
Divorce or widowed	226 (12.5)	423 (24.8)	190 (7.5)	733 (31.4)	107 (5.4)	369 (21.1)
Educational level, N (%)						
Primary or less	55 (3.0)	150 (8.8)	175 (6.9)	90 (3.9)	76 (3.8)	89 (4.4)
Vocational	723 (40.0)	453 (26.7)	561 (22.1)	772 (33.1)	440 (22.2)	184 (10.5)
Secondary	605 (33.4)	847 (49.7)	882 (34.7)	681 (29.2)	613 (30.9)	732 (41.8)
University	426 (23.6)	256 (15.0)	926 (36.4)	789 (33.8)	853 (43.0)	746 (42.6)
Occupational class, N (%)						
Manager or profession	486 (27.0)	281 (16.9)	676 (26.6)	467 (20.0)	595 (30.4)	338 (19.7)
Non-manual worker	729 (40.6)	1087 (65.2)	864 (34.0)	1404 (60.2)	940 (48.1)	1155 (67.4)
Manual worker	583 (32.4)	300 (18.0)	1004 (39.5)	461 (19.8)	421 (21.5)	222 (12.9)
Deprivation, N (%)						
Low (0–3.9)	1569 (86.6)	1399 (82.1)	1805 (71.0)	1238 (53.1)	1595 (80.8)	1278 (73.4)
High (4–9)	242 (13.4)	305 (17.9)	739 (29.1)	1094 (46.9)	379 (19.2)	463 (26.6)
Depression, N (%)						
CESD < 16	1563 (88.3)	1327 (79.3)	1662 (88.2)	1266 (71.9)	1686 (85.9)	1299 (80.8)
CESD ≥ 16	208 (11.7)	346 (20.7)	222 (11.8)	495 (28.1)	275 (14.1)	432 (19.2)
Problem drinking, N (%)						
No	1584 (88.9)	1611 (96.9)	2054 (80.7)	2288 (98.0)	1617 (88.8)	1407 (98.0)
Yes	198 (11.1)	52 (3.1)	490 (19.3)	46 (2.0)	204 (11.2)	29 (2.0)
Current smoker, N (%)						
No	1202 (66.9)	1204 (71.2)	1276 (50.2)	2003 (85.8)	1262 (63.8)	1153 (66.0)
Yes	595 (33.1)	488 (28.8)	1268 (49.8)	331 (14.2)	717 (36.2)	595 (34.0)

N, number; SD, standard deviation; CESD, Center for Epidemiologic Studies Depression scale.

Table 3. Descriptive statistics of dietary outcomes by country and gender

Dietary outcomes	Czech Republic				Russia				Poland			
	Men N= 1814		Women N= 1708		Men N= 2544		Women N= 2332		Men N= 1982		Women N= 1752	
HDI overall score:												
Mean, SD	2.0	1.1	2.6	1.2	1.8	1.0	2.0	1.1	1.5	1.1	1.9	1.2
Total energy (MJ/d):												
Mean, SD	9.1	3.1	8.4	3.0	12.2	3.6	10.4	3.2	9.9	3.0	9.0	2.7
Saturated fatty acid (g/d):												
Mean, SD	32	13	29	13	48	20	40	16	40	16	35	14
% of total energy	13%		13%		15%		15%		15%		15%	
% meeting WHO range*	6%		10%		3%		3%		2%		5%	
PUFA (g/d): Mean, SD	15	7	14	6	26	10	25	10	13	6	12	5
% of total energy	6%		6%		8%		9%		5%		5%	
% meeting WHO range*	57%		59%		71%		62%		17%		16%	
Protein (g/d): Mean, SD	96	35	87	30	125	38	107	33	106	32	95	29
% of total energy	18%		17%		17%		17%		18%		18%	
% meeting WHO range*	9%		14%		12%		19%		7%		10%	
Total carbohydrate (g/d):												
Mean, SD	240	96	238	96	287	85	253	82	267	86	262	87
% of total energy	44%		48%		40%		41%		46%		49%	
% meeting WHO range*	5%		16%		1%		2%		7%		17%	
Free sugar (g/d): Mean, SD	110	57	128	67	126	49	125	48	124	54	133	59
% of total energy	20%		26%		17%		20%		21%		25%	
% meeting WHO range*	4%		1%		5%		2%		3%		1%	
NSP (g/d): Mean, SD	17	9	19	10	18	6	18	6	19	7	19	8
% meeting WHO range*	19%		29%		22%		19%		27%		32%	
Cholesterol (mg/d):												
Mean, SD	326	141	283	123	544	253	413	165	424	195	357	144
% meeting WHO range*	49%		63%		11%		24%		22%		36%	
Fruit & vegetable (g/d):												
Mean, SD	452	396	678	582	379	255	450	305	456	267	559	347
% meeting WHO range*	56%		75%		52%		65%		65%		75%	

N, number; SD, standard deviation; WHO, World Health Organisation; PUFA, polyunsaturated fatty acid; NSP, non–starch polysaccharide; HDI, Healthy Diet Indicator.

* Percentage of HAPIEE subjects who meet the WHO recommended range of the HDI component (Table 1).

Table 4. Associations between exposure variables and 8 HDI components by logistic regression

8 HDI components	Men (n= 6340)				Women (n= 5792)			
	OR per unit†	95 % CI	OR per SD‡	P-value	OR per unit†	95 % CI	OR per SD‡	P-value
1. Association between ER ratio and diet after adjustment for OC, confounders & mediators*								
Saturated fatty acid	0.71	0.35, 1.46	0.92	0.349	0.51	0.30, 0.89	0.84	0.019
PUFA	0.84	0.65, 1.08	0.95	0.151	1.01	0.78, 1.32	1.00	0.893
Protein	0.89	0.59, 1.35	0.97	0.594	0.82	0.57, 1.18	0.95	0.301
Total carbohydrate	0.89	0.51, 1.54	0.97	0.671	0.76	0.51, 1.14	0.93	0.183
Free sugars	0.60	0.34, 1.05	0.87	0.098	0.65	0.30, 1.22	0.90	0.496
NSP	0.91	0.69, 1.21	0.97	0.513	0.73	0.55, 0.98	0.92	0.033
Cholesterol	0.82	0.63, 1.05	0.93	0.095	0.88	0.68, 1.13	0.97	0.329
Fruit & vegetable	0.83	0.66, 1.06	0.95	0.116	0.86	0.66, 1.12	0.96	0.273
2. Association between OC and diet after adjustment for ER ratio, confounders & mediators*								
Saturated fatty acid	0.95	0.90, 0.99	0.84	0.043	0.96	0.93, 1.01	0.88	0.099
PUFA	0.97	0.95, 0.99	0.92	0.020	0.97	0.95, 0.99	0.90	0.005
Protein	1.00	0.97, 1.03	1.01	0.862	0.98	0.96, 1.01	0.95	0.290
Total carbohydrate	0.98	0.94, 1.03	0.96	0.569	0.99	0.96, 1.02	0.97	0.554
Free sugars	0.96	0.92, 1.00	0.87	0.061	0.96	0.89, 1.04	0.88	0.294
NSP	0.98	0.96, 1.01	0.95	0.148	0.98	0.96, 1.00	0.93	0.072
Cholesterol	0.99	0.97, 1.01	0.97	0.466	1.00	0.98, 1.02	0.99	0.943
Fruit & vegetable	0.98	0.96, 1.00	0.94	0.079	0.99	0.97, 1.01	0.98	0.585

HDI, Healthy Diet Indicator; OR, odds ratio; CI, confidence interval; SD, standard deviation; PUFA, polyunsaturated fatty acid; NSP, non-starch polysaccharide.

* Binary logistic regression was used to assess the associations between exposure variables (ER ratio and OC) and 8 HDI components, respectively, after adjustment for confounders and mediators.

† OR per unit is the odds of having healthy intake of the HDI component for a 1–unit increase in the exposure.

‡ OR per SD is the odds of having healthy intake of the HDI component for a 1–SD increase in the exposure. 1 SD of ER ratio = 0.25 in men and women. 1 SD of OC = 3.65 in men and 3.56 in women.

Table 5. Associations between exposure variables and HDI by linear regression

Variables	Men (n= 6340)			Women (n= 5792)		
	Beta*	Standardized beta†	P–value	Beta*	Standardized beta†	P–value
Model 1: adjusted for confounders & ER ratio						
ER ratio	–0.224	–0.052	< 0.001	–0.198	–0.042	0.002
Model fit	$R^2 = 0.045$			$R^2 = 0.082$		
Model 2: adjusted for confounders & OC						
OC	–0.017	–0.056	< 0.001	–0.017	–0.052	< 0.001
Model fit	$R^2 = 0.045$			$R^2 = 0.081$		
Model 3: adjusted for confounders, ER ratio & OC						
ER ratio	–0.169	–0.039	0.005	–0.171	–0.037	0.017
OC	–0.013	–0.044	0.002	–0.014	–0.043	0.003
Model fit	$R^2 = 0.046$			$R^2 = 0.082$		
Model 4: additionally adjusted for potential mediators						
ER ratio	–0.126	–0.030	0.046	–0.153	–0.033	0.036
OC	–0.011	–0.036	0.015	–0.011	–0.032	0.040
Confounders						
Age	0.004	0.021	0.124	0.011	0.050	0.001
Marital status: Married						
Single	0.085	0.013	0.346	0.086	0.017	0.236
Divorce or widowed	–0.084	–0.022	0.098	–0.042	–0.016	0.301
Education: Primary / less						
Vocational	0.052	0.021	0.541	0.078	0.028	0.346
Secondary	0.008	0.004	0.921	0.052	0.021	0.519
University	0.078	0.034	0.360	0.138	0.054	0.101
Occupation: Manager						
Non-manual worker	–0.032	–0.014	0.446	–0.002	–0.001	0.969
Manual worker	–0.070	–0.032	0.042	0.027	0.008	0.655
Deprivation: Low						
High	–0.066	–0.024	0.084	–0.102	–0.039	0.008
Potential mediators						
Depression: Yes	–0.027	–0.008	0.552	–0.021	–0.008	0.606
Problem drinking: Yes	–0.194	–0.062	< 0.001	–0.264	–0.035	0.014
Current smoker: Yes	–0.192	–0.086	< 0.001	–0.048	–0.018	0.225
Model fit	$R^2 = 0.063$			$R^2 = 0.087$		

HDI, Healthy Diet Indicator; ER ratio, effort–reward ratio; OC, overcommitment.

* Beta (β) coefficient reflects change in HDI score for a 1–unit increase in the exposure.

† Standardized β coefficient reflects change of standard deviation (SD) in HDI score for a 1–SD increase in the exposure. 1 SD of ER ratio = 0.25 in men and women. 1 SD of OC = 3.65 in men and 3.56 in women. 1 SD of HDI = 1.08 in men and 1.18 in women.