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Factors associated with six-year weight change in young and middle-aged adults in the Young Finns Study

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Abstract

Objective. To examine factors associated with weight change and obesity risk in young and middle-aged adults. Subjects/methods. The Young Finns Study with its 923 women and 792 men aged 24–39 years at baseline were followed for six years. Variables associated with the weight change were investigated with regression models. Results. The average weight change was 0.45 kg/year in women and 0.58 kg/year in men. In women, weight change was steady across all ages. In men, weight changes were more pronounced in younger age groups. In women (weight gain ≥ 2 kg, n = 490), medication for anxiety, low occupational status, high baseline BMI (body mass index), high intake of sweet beverages, high childhood BMI, high salt (NaCl and/or KCl) use, low number of children, low childhood family income, high stature and low level of dependence (a temperament subscale) were associated with increased weight gain (in the order of importance). In men (weight gain ≥ 2 kg, n = 455), high stature, high intake of french fries, low intake of sweet cookies, young age, recent divorce, low intake of cereals, high intake of milk, depressive symptoms, rural childhood origin, high baseline BMI and unemployment were associated with more pronounced weight gain. Sedentariness (screen-time) was associated with weight gain only in young men. Physical activity and genetic risk for high BMI (score of 31 known variants) were not consistently associated with weight change. Conclusions. Socio-economic factors, temperamental and physical characteristics, and some dietary factors are related with weight change in young/middle-aged adults. The weight change occurring in adulthood is also determined by childhood factors, such as high BMI and low family income.

Key Words: Adiposity, adult, overweight, population, risk factors, weight gain, weight loss, young adult

Introduction

Obesity is an important risk factor for cardiovascular disease and other disorders [1]. In Finland, 20.4% of the men and 19.0% of the women are obese (body mass index, BMI ≥ 30 kg/m²) [2]. Alarmingly, weight gain is more common in young adults than in the average members of the adult population [3]. Although it is clear that it is an imbalance between energy intake and energy expenditure that leads to weight gain, there are numerous behavioral, environmental and genetic determinants which can modulate this balance. Recent reviews evaluating various interventions have not been able to identify any optimum set of strategies which could prevent the weight gain [4–6].
Cohort studies conducted in both Finland and the US have previously provided data on weight gain in adults, but they have not focused on young adults. In the 1980s in Finland, changes in weight and in BMI were monitored for 17,294 Finnish men and women, aged 20–92 years, with a follow-up of an average of 5.7 years. Weight loss was associated with old age and high initial BMI, whereas a weight gain was most common among participants aged < 50 years at entry, even in those subjects with a high initial BMI [7]. When investigated in detail (n = 12,669), the weight gain (≥ 5 kg/5 years) was found to be highest in those individuals with low levels of education, chronic diseases, little leisure-time physical activity or heavy alcohol consumption, and for those who became married or stopped smoking during the follow-up. Parity and energy intake predicted the weight gain only in women [8]. In the combined Nurse’s Health Study (NHS + NHSII) and the Health Professionals Follow-up Study (120,877 middle-aged or older men and women), four-year weight gain was most strongly associated with the intake of potato chips, potatoes, sugar-sweetened beverages, unprocessed red meats, and processed meats, and inversely with the intake of vegetables, whole grains, fruits, nuts, and yogurt. Other lifestyle factors independently associated with the weight gain e.g. low physical activity, alcohol use, smoking cessation, sleep (< 6 or > 8 hours of sleep), and duration of television watching [9].

Determinants of adulthood obesity have been extensively investigated also in the Young Finns Study (YFS) population [10,11]. There are also a few population and intervention studies focusing on weight change in young adults, but heterogeneity and the short duration of these studies make it difficult to interpret the data [6,12,13]. In addition, due to rapid urbanization and internationalization, there has been changes in lifestyle in the Finnish society, such as a decrease in work-related physical activity, and these have occurred during the past three to four decades [14]. Therefore, potential childhood and adolescence behavioral, environmental, anthropometric and biochemical weight regulators and recently discovered genetic risk markers for BMI [15] were investigated to examine which of these factors could be associated with weight gain in a contemporary population of young and middle-aged Finnish adults. For example, it has been previously found that temperament traits, such as novelty seeking (directly) and reward dependence ( inversely) are related to BMI [16], and therefore, the impacts of these subscales were investigated in the present study.

Materials and methods

Subjects
In 1980, 4,320 children and adolescents, aged 3, 6, 9, 12, 15 and 18 years, and living in five university cities and 12 adjacent rural communities, were randomly chosen from the Finnish national population register. A total of 3,596 subjects participated in the examination in 1980 [17]. Follow-up examinations with weight measurements were carried out in 2,276 subjects (63.3%) aged 24–39 years in 2001 (baseline for the present study) and for 2,170 subjects (60.3%) aged 30–45 years in 2007. Those women who were pregnant in either 2001 or 2007 or had missing pregnancy information in 2001 were excluded. In addition, one male outlier with a weight loss more than 50 kg was removed. Thus, the final study set consisted of 923 women and 792 men for whom the body mass index (kg/m²) information was available for the period 2001–2007.

The study was carried out in accordance with the recommendations of the Declaration of Helsinki. All participants provided written informed consent, and the study protocol was approved by a local ethics committee (minutes 3/2001, 12/2006 and 9/2010).

Anthropometric data
Distributions of different continuous variables and their normality were statistically and visually tested (Table I). Stature or height and weight were measured in childhood (1980) and at adulthood baseline (2001) and follow-up (2007). Baseline weight and BMI (body mass index) (kg/m²) were log-transformed. Due to the wide childhood age range (from 3–18 years) resulting in age-related differences in anthropometrics, the childhood BMI was Z-scored separately for different age groups. To investigate hereditary risk for high BMI, a sum variable created from dichotomous mother’s + father’s BMI in childhood (1 = belonging to the lowest 50% in BMI values vs. 2 = belonging to the highest 50%) was calculated (receiving values from 2–4). Parent’s anthropometric data and study subjects’ birth weight and length as newborns were collected during the interview.

Dietary data
The non-quantitative dietary (use frequency) questionnaire (2001) [18] covered habitual eating patterns, special diets and food choices. It also included questions on the consumption of certain food groups which may play a role in weight regulation [19], such as milk, milk products; porridge, cereals and muesli (cereals); french fries; water-cooked potatoes; fruits and vegetables; meat and meat products; fish; eggs; cookies; sweets; and sugar-sweetened beverages. The habitual consumption was assessed as a frequency (ranging from never or rarely = 1, to daily = 6). The responses were converted into portions per month (ranging from never or rarely = 0, to daily = 30, high/low use frequency being equivalent to the terms
Table I. Study characteristics, and Pearson correlations of different variables with the 6-year weight change and baseline body mass index.

| Characteristics                                                                 | Women                                          | Men                                            |
|--------------------------------------------------------------------------------|------------------------------------------------|------------------------------------------------|
|                                                                              | N    | Mean ± SE | Weight change | Baseline BMI | N    | Mean ± SE | Weight change | Baseline BMI |
| Six-year weight change (2001–2007) (kg)                                       | 923  | 2.73 ± 0.21 | –0.06         | 792          | 3.49 ± 0.23 | –0.098**       |
| 2001 data (adolescent baseline)                                               |      |            |               |              |      |            |               |              |
| Baseline weight (kg)b                                                         | 923  | 67.3 ± 0.43 | –0.04         | 792          | 82.8 ± 0.50 | –0.05         | 0.899**       |
| Height (cm)                                                                   | 923  | 166 ± 0.20  | 0.05          | 792          | 180 ± 0.23  | 0.083*         | 0.02          |
| Baseline body mass index, BMI (kg/m²)b                                        | 923  | 24.38 ± 0.149 | –0.06       | 792          | 25.64 ± 0.138 | –0.098** | 0.239**       |
| Baseline age (years)                                                          | 923  | 32.1 (24 to 39) | 0.04       | 792          | 31.9 (24 to 39) | –0.135** |                  |
| Hormones and inflammatory markers                                            |      |            |               |              |      |            |               |              |
| Serum leptin (ng/mL)b                                                         | 919  | 15.2 ± 0.31  | –0.02         | 789          | 5.3 ± 0.15  | –0.06         | 0.716**       |
| Serum adiponectin (µg/mL)b                                                    | 919  | 10.9 ± 0.14  | 0.102**      | 789          | 7.3 ± 0.12  | 0.082*         | –0.275**      |
| Serum insulin (mU/L)b                                                         | 920  | 7.73 ± 0.19  | 0.03         | 790          | 7.60 ± 0.21 | –0.07         | 0.517**       |
| Serum testosterone (nmol/L)b                                                  | 917  | 1.6 ± 0.02   | 0.02         | 757          | 18.5 ± 0.27 | 0.05          | –0.305**      |
| Serum sex hormone binding globulin (nmol/L)b                                 | 918  | 82.6 ± 2.0   | 0.04         | 757          | 30.3 ± 0.4  | 0.093*         | –0.428**      |
| Serum C reactive protein (mg/L)b                                              | 920  | 2.13 ± 0.14  | –0.06        | 790          | 1.45 ± 0.12 | –0.03         | 0.422**       |
| Serum amyloid A-1 (µg/mL)b                                                    | 917  | 23.5 ± 1.9   | 0.00         | 790          | 21.1 ± 2.9  | –0.02         | 0.277**       |
| Dietary habits and alcohol use                                                |      |            |               |              |      |            |               |              |
| Milk use (categorized 1 = low to 3 = high)c                                   | 888  | 1.76 ± 0.03  | –0.03         | 772          | 2.11 ± 0.03 | 0.06          | 0.092*        |
| Monthly use of industrial milk products (portions)                           | 862  | 39.0 ± 0.59  | 0.02         | 746          | 36.1 ± 0.65 | –0.01         | –0.05         |
| Monthly use of cereals (portions)                                            | 877  | 10.9 ± 0.35  | 0.05         | 763          | 8.5 ± 0.36  | –0.143**       | 0.00          |
| Monthly use of french fries (portions)                                       | 880  | 1.74 ± 0.06  | 0.04         | 772          | 2.97 ± 0.12 | 0.101**       | –0.04         |
| Monthly use of water-cooked potatoes (portions)                              | 887  | 13.1 ± 0.28  | 0.02         | 768          | 13.7 ± 0.29 | –0.089*       | 0.083**       |
| Monthly use of vegetables and fruits (portions)                              | 875  | 38.9 ± 0.53  | –0.01        | 761          | 32.5 ± 0.57 | –0.03         | –0.157**      |
| Monthly use of meat and meat products (portions)                             | 874  | 26.9 ± 0.50  | 0.02         | 764          | 35.9 ± 0.52 | –0.02         | 0.04          |
| Monthly use of fish (portions)                                                | 883  | 4.15 ± 0.12  | 0.01         | 772          | 3.76 ± 0.12 | –0.04         | 0.05          |
| Monthly use of eggs (portions)                                                | 880  | 3.88 ± 0.14  | 0.05         | 764          | 4.32 ± 0.16 | –0.03         | 0.078*        |
| Monthly use of sugar-sweetened soft drinks (portions)                        | 889  | 5.07 ± 0.23  | 0.05         | 772          | 8.43 ± 0.29 | 0.05          | –0.03         |
| Monthly use of sweet cookies (portions)                                       | 889  | 11.4 ± 0.29  | –0.108*      | 772          | 11.6 ± 0.32 | –0.06         | –0.02         |
| Monthly use of sweets and chocolate (portions)                               | 888  | 9.71 ± 0.25  | –0.04        | 768          | 8.45 ± 0.26 | –0.01         | –0.05         |
| Frequency to add salt (NaCl and/or KCl) to the food                           | 890  | 1.22 ± 0.01  | 0.078*       | 774          | 1.28 ± 0.02 | 0.02          | 0.04          |
| (1 = never, 2 = when needed following tasting, 3 = always)                   |      |            |               |              |      |            |               |              |
| Alcohol use (categorized 1 = low to 4 = high)c                                | 913  | 2.17 ± 0.03  | –0.04        | 779          | 2.82 ± 0.04 | 0.05          | 0.00          |
| Genetic factors                                                               |      |            |               |              |      |            |               |              |
| Genetic risk score for BMI (17 = low to 40 = high)                            | 842  | 27.34 ± 0.111 | –0.01       | 725          | 27.59 ± 0.124 | 0.01 | 0.198**      |
| Parent’s BMI in 1980 (sum of dichotomous mother and father BMI, 2 = low to 4 = high) | 898  | 3.03 ± 0.02 | –0.01         | 768          | 3.00 ± 0.03 | –0.01         | 0.286**       |
| Psychological factors                                                         |      |            |               |              |      |            |               |              |
| Depressive symptoms (1 = low level to 5 = high level)                         | 764  | 2.15 ± 0.03  | 0.072*       | 606          | 1.92 ± 0.02 | 0.04          | 0.144**       |
| Novelty seeking (NS) (1 = low level to 5 = high level)                        | 766  | 3.03 ± 0.01  | 0.00         | 606          | 2.92 ± 0.02 | 0.07          | 0.123**       |
| Exploratory excitability (1–5)                                                 | 766  | 3.32 ± 0.02  | –0.01        | 606          | 3.25 ± 0.02 | 0.01          | 0.05          |
| Impulsiveness (1–5)                                                           | 766  | 2.85 ± 0.02  | 0.03         | 606          | 2.77 ± 0.02 | 0.06          | 0.109**       |

(Continued)
Table I. (Continued)

| Characteristics                                                                 | Women          |       | Men          |       |
|--------------------------------------------------------------------------------|----------------|-------|--------------|-------|
|                                                                                  | \( N \)        | Mean \( \pm \) SE | Weight change \( r \) | Baseline BMI \( r \) | \( N \) | Mean \( \pm \) SE | Weight change \( r \) | Baseline BMI \( r \) |
| Extravagance (1–5)                                                              | 767            | 3.28 \( \pm \) 0.03 | 0.079* | 0.07   | 607 | 3.00 \( \pm \) 0.03 | 0.080* | 0.084* |
| Disorderliness (1–5)                                                            | 767            | 2.67 \( \pm \) 0.02 | 0.077* | 0.04   | 607 | 2.65 \( \pm \) 0.02 | 0.03  | 0.112** |
| Reward dependence (RD) (1 = low level to 5 = high level)                        | 766            | 3.52 \( \pm \) 0.01 | -0.099** | -0.06 | 606 | 3.17 \( \pm \) 0.02 | 0.03  | 0.00   |
| Sentimentality (1–5)                                                            | 767            | 3.30 \( \pm \) 0.02 | -0.02  | -0.02  | 607 | 2.88 \( \pm \) 0.02 | 0.03  | 0.081* |
| Attachment (1–5)                                                                | 767            | 3.86 \( \pm \) 0.02 | -0.085* | -0.05 | 607 | 3.39 \( \pm \) 0.03 | 0.05  | 0.00   |
| Dependence (1–5)                                                                | 766            | 3.39 \( \pm \) 0.02 | -0.089* | -0.04 | 606 | 3.24 \( \pm \) 0.02 | -0.05 | -0.08  |
| Medication for anxiety (0 = no, 1 = yes)                                        | 923            | 0.03 \( \pm \) 0.006 | 0.070* | 0.109** | 792 | 0.02 \( \pm \) 0.004 | 0.07  | -0.05  |
| Low back pain during the latest 12 months (1 = no, 2 = yes)                     | 916            | 1.71 \( \pm \) 0.015 | 0.140** | 0.01  | 786 | 1.63 \( \pm \) 0.017 | -0.01 | -0.01  |
| **Living habits**                                                                |                |       |              |       |
| Smoker (0 = no, 1 = yes)                                                         | 900            | 0.19 \( \pm \) 0.01 | 0.02  | -0.02  | 775 | 0.27 \( \pm \) 0.02 | 0.075* | -0.04  |
| Leisure-time physical activity (5 = low to 15 = high)                            | 859            | 1.00 \( \pm \) 0.08 | -0.04  | -0.128** | 742 | 9.8 \( \pm \) 0.09  | 0.01  | -0.02  |
| Exercise intensity (1 = low to 3 = high)                                        | 908            | 2.12 \( \pm \) 0.02 | -0.05  | -0.05  | 779 | 2.31 \( \pm \) 0.02 | -0.03 | -0.00  |
| Screen-time (minutes/day)                                                        | 865            | 105 \( \pm \) 2.3 | 0.01  | 0.140** | 747 | 125 \( \pm \) 2.8  | 0.082* | 0.03   |
| **Living environment**                                                           |                |       |              |       |
| Number of grandparents born in West-Finland (0–4)                                | 703            | 1.35 \( \pm \) 0.06 | 0.06  | -0.02  | 589 | 1.48 \( \pm \) 0.07 | -0.05 | -0.02  |
| Living with a partner (0 = no, 1 = yes)                                          | 916            | 1.74 \( \pm \) 0.01 | -0.04  | 0.083* | 784 | 1.73 \( \pm \) 0.02 | -0.05 | 0.166** |
| Occupational status (1 = low to 3 = high)                                        | 809            | 1.97 \( \pm \) 0.23 | -0.099** | -0.095** | 686 | 1.86 \( \pm \) 0.032 | -0.04 | -0.04  |
| Educational status (1 = low to 3 = high)                                         | 919            | 2.11 \( \pm \) 0.17 | -0.097** | -0.104** | 788 | 2.11 \( \pm \) 0.01 | -0.04 | 0.125** |
| Home location (1 = city centre to 4 = rural areas)                              | 898            | 2.29 \( \pm \) 0.02 | -0.06  | 0.107** | 778 | 2.33 \( \pm \) 0.035 | 0.01  | 0.133** |
| Unemployed within the latest year (number of months)                             | 664            | 1.24 \( \pm \) 0.115 | -0.097* | 0.111** | 577 | 0.73 \( \pm \) 0.097 | 0.04  | -0.03  |
| Children (number)                                                                | 923            | 1.26 \( \pm \) 0.042 | -0.04  | 0.122** | 792 | 0.97 \( \pm \) 0.042 | -0.05 | 0.137** |
| Divorced between the years 1992 and 2001 (0 = no, 1 = yes)                      | 904            | 1.10 \( \pm \) 0.010 | 0.068* | 0.02   | 780 | 1.07 \( \pm \) 0.009 | 0.01  | 0.03   |
| **1980 data (childhood)**                                                        |                |       |              |       |
| **Childhood anthropometry and other childhood factors**                          |                |       |              |       |
| Birth weight (g)                                                                | 785            | 3340 \( \pm \) 20 | 0.04  | 0.02   | 667 | 3570 \( \pm \) 20 | 0.02  | 0.04   |
| Birth length (cm)                                                               | 781            | 50.0 \( \pm \) 0.08 | 0.03  | 0.01   | 660 | 50.7 \( \pm \) 0.09 | 0.03  | 0.03   |
| Birth weight/birth length                                                        | 778            | 68.5 \( \pm \) 0.32 | 0.04  | 0.02   | 657 | 70.1 \( \pm \) 0.37 | 0.02  | 0.03   |
| Childhood body mass index (kg/m²)                                               | 920            | 17.9 \( \pm \) 0.10 | 0.03  | 0.506** | 782 | 18.0 \( \pm \) 0.11 | 0.01  | 0.525** |
| Childhood high-sensitive C-reactive protein (mg/L)                              | 911            | 1.09 \( \pm \) 0.11 | 0.05  | -0.01  | 778 | 0.94 \( \pm \) 0.10 | 0.02  | -0.07  |
| Childhood family income (1 = low to 8 = high)                                   | 884            | 4.87 \( \pm \) 0.065 | -0.092** | -0.113** | 762 | 4.90 \( \pm \) 0.068 | -0.01 | -0.06  |
| Childhood home location (1 = city centre to 4 = rural areas)                    | 920            | 2.68 \( \pm \) 0.031 | 0.04  | 0.01   | 788 | 2.65 \( \pm \) 0.034 | -0.02 | 0.06   |

Variable characteristics (mean \( \pm \) SE) and Pearson correlation coefficients. *\( p < 0.05 \); **\( p < 0.01 \); *BMI was presented as a reference variable; bLog-transformed, categorized or dZ-scored variables were used in statistical analyses.
high/low intake, consumption or use). There were two exceptions: Consumption of milk was assessed as glasses per day, and total alcohol use was expressed as drinks per day (one drink was equivalent to one glass of wine or one bottle of beer). Transformations were unable to normalize all of the distributions. Therefore, milk use was categorized into 3 (i.e. values from 1–3) and alcohol use into 4 (i.e. values from 1–4) groups equal in their number of subjects. Additional salt intake was assessed by asking the frequency to add salt (NaCl and/or KCl) to the food: 1 = never, 2 = when needed following tasting, 3 = always.

Other questionnaires

Adulthood (2001) medication for anxiety, smoking and the low back pain during the latest year (no = 0 vs. yes = 1), number of children, daily time used to watch television and to play computer games (screen-time in minutes, log-transformed), number of unemployment months within the latest year, current living with a partner (no = 0 vs. yes = 1) and divorce history since the year 1992 (no = 0 vs. yes = 1), and the number of subject’s grandparents born in West-Finland (levels of some cardio-metabolic risk factors have been shown to be higher in subjects originating from eastern Finland [20]) were characterized by questionnaires and/or during an interview. Leisure-time physical activity index (2001) [21] was categorized into score values from 5–15, and exercise intensity from 1–3 (1 = usually not becoming out of breath or sweating, 2 = becoming out of breath and sweating slightly, 3 = becoming out of breath and sweating considerably). Socioeconomic status (SES)-related variables (2001), i.e. educational status (1 = comprehensive school, 2 = secondary education, not academic, 3 = academic) and occupational status (1 = manual, 2 = lower-grade non-manual, 3 = higher-grade non-manual) were also characterized. Childhood and adulthood home locations were categorized from 1–4 (1 = city centre, 2 = suburban, 3 = rural centre (village), 4 = rural remote) and childhood family incomes from 1 = low to 8 = high.

Personality (2001) was evaluated based on Cloninger’s sociobiological model and the Temperament and Character Inventory estimated from this model [22] which comprises four temperament traits, i.e. novelty seeking, harm avoidance, reward dependence and persistence, each of them consisting of subscales. Previously, novelty seeking and reward dependence have been shown to play a role in the development of obesity [16]. In the present study, their subscales were used: Exploratory excitability, impulsiveness, extravagance and disorderliness of novelty seeking, and sentimentality, attachment and dependence of reward dependence (see Supplementary Material for descriptions, available online at http://informahealthcare.com doi/abs/10.3109/00365513.2014.992945). Each scale was assessed with a five-point Likert scale. Depressive symptoms were assessed with a modified version of Beck’s Depression Inventory by using 21 items on a five-point scale, ranging from 1 (strongly disagree) to 5 (strongly agree) [23].

Laboratory measurements

Overnight fasting serum was used for all of the laboratory analyses. Due to the wide childhood age range (from 3–18 years), childhood high-sensitive C-reactive protein (CRP) was Z-scored separately for different age groups following log-transformation, and adulthood (2001) CRP log-transformed (measured by standard methods). In 2001, fasting insulin levels were measured by microparticle enzyme immunoassay (Abbott Laboratories, Diagnostic Division, Dainabot, Japan), and serum leptin [24], total adiponectin [24], testosterone [25], sex hormone binding globulin (SHBG) [25] and amyloid A-1 [26] levels (all of these were log-transformed), as previously described.

Genome-wide analysis was performed with Illumina Bead Chip (Human 670K). Genetic BMI risk score was formed using 31 previously published single nucleotide polymorphism as an arithmetic sum variable of the risk alleles [15]. This score has been shown earlier to associate with BMI (p < 1 × 10⁻⁷) in a meta-analysis using data on 249,796 individuals [15].

Statistical analysis

SPSS statistical software (version 20) was used for statistical analyses. Distributions of different continuous variables and their normality were visually and statistically tested (Kolmogorov-Smirnow test). Pearson’s (Table I) and Spearman’s (Supplementary Table I, available online at http://informahealthcare.com doi/abs/10.3109/00365513.2014.992945) correlation coefficients were used to evaluate bivariate correlations. In linear regression models with mean substitution, bBMI (baseline BMI) was always included into the model, following a stepwise method (p for entry 0.05 and p for removal 0.10) for rest of the variables. Potential weight regulation-related explanatory variables, offered for the models (described in Table I), were selected based on the current literature and on their availability as baseline or childhood factors for most of the subjects in our study cohort. The purpose was to identify ‘overall determinants’ of the weight change. For this reason, also variables which were potentially causally dependent on each other were offered for the same models. However, strongly associating variables, such as occupational and educational status or parallel temperament subscales, were not allowed to be entered into the same model. In such cases, the variable showing the strongest crude correlation with the
weight change was offered for the final model. In addition, some variables dependent on obesity or weight change, such as serum lipids, hypertension and type 2 diabetes were excluded and not included in the presented data.

Due to significant sex-interactions in several weight change-determinants characterized (generalized linear models with the sex, explanatory variable and sex*explanatory variable interaction term, data not shown), men and women were studied separately. In particular, there was a difference in the age-related weight change between the sexes (Figure 1). For this reason, subjects aged 24–27 years at baseline, and those aged 30 years or more, were also separately investigated (Table IIA). We also studied separately those aged 30 years or more, were also separately

Investigated (Table IIB). The subject was assessed to have gained weight if the weight had increased more than 2 kg during the follow-up (accuracy of delta weight was approximated to be ±2 kg). To study the factors associated with incident obesity (0 = BMI < 30 kg/m² in 2001 vs. 1 = BMI ≥ 30 kg/m² in 2007), we also calculated odds ratios using logistic regression models for the study variables (see Table III for details).

Results

Bb BMI (baseline BMI) was 24.4 kg/m² (range 16–47 kg/m²) in women (n = 923) and 25.6 kg/m² (16–44 kg/m²) in men (n = 792). One hundred women (10.8%) and 103 men (13.0%) were obese (bBMI ≥ 30 kg/m²) at baseline. The mean 6-year weight increase was 2.7 kg (–28.8–35.5 kg) in women and 3.5 kg (–25.5–31.3 kg) in men (Table I). In men, the highest mean weight gain was observed among the 24 years (5.3 kg [–20.8–30.7 kg]) and the 27 years old men (4.2 kg [–11.3–23.5 kg]) (Figure 1). In contrast, women experienced the smallest mean weight gain in those age groups, i.e. 1.8 kg (–22.1–16.8 kg) in 24-year-olds, and 2.2 kg (–28.8–23.7 kg) in 27-year-olds (Figure 1).

Bivariate correlations

The baseline characteristics and bivariate Pearson’s correlation coefficients for the potential determinants of the weight change and bBMI (as reference) are shown in Table I. Overall, the associations were stronger with the bBMI than with the 6-year weight change, and with some variables, such as with serum total adiponectin levels, bBMI and weight change showed opposite associations. In women (n = 923), a high serum total adiponectin level, high extravagance of novelty seeking, additional salt use, disorderliness of novelty seeking, depressive symptoms, medication for anxiety and a recent divorce were directly associated with weight gain (in the order of importance). On the other hand, consumption of sweet cookies, high occupational and educational status, recent unemployment, high childhood family income, and high dependence and attachment of reward dependence were associated with weight loss. In men (n = 792), consumption of french fries, high SHBG level, high stature, and high total adiponectin level, screen-time (TV watching + computer game playing), extravagance of novelty seeking and smoking were associated with the weight gain, whereas consumption of cereals, older age, high bBMI and high use of water-cooked potatoes were associated with weight loss. In respect to the potential variables, leisure-time physical activity and genetic BMI risk score did not show any crude association with the weight change. The corresponding Spearman’s correlation coefficients have been presented in Supplementary Table I, available online at http://informahealthcare.com/doi/abs/10.3109/00365513.2014.992945.

The increase in the number of children (2001–2007) was not associated with any weight change, in either women or men (data not shown in tables). However, quitting smoking during the follow-up was associated with an increase in body weight in men (r = 0.141, p < 0.001, data not shown in tables). There was an inverse relationship between serum total adiponectin and insulin concentrations at baseline (data not shown in tables).

Multivariable models

When all women were examined as a single group (n = 923, data not shown in tables), low bBMI (beta
|                  | Women aged 24 to 27 years \( (n = 265) \) | Men aged 24 to 27 years \( (n = 226) \) | Women aged 30 to 39 years \( (n = 658) \) | Men aged 30 to 39 years \( (n = 566) \) |
|------------------|------------------------------------------|------------------------------------------|------------------------------------------|------------------------------------------|
| **Subjects with weight gain (delta weight > 2 kg)** |                                |                                |                                |                                |
| **A: Age stratification** |                                |                                |                                |                                |
| Women aged 24 to 27 years \( (n = 265) \) | | | | |
| Unemployed within the latest year (number of months) | \(-0.526 \pm 0.182 \) p.002 | Screen-time (minutes/day)\( a \) | | |
| Baseline body mass index (kg/m\(^2\)) | \(-14.738 \pm 0.153 \) p.011 | Monthly use of french fries (portions) | | |
| Occupational status (1 = low to 3 = high) | \(-1.724 \pm 0.150 \) p.011 | Unemployed within the latest year (number of months) | | |
| Monthly use of sugar-sweetened soft drinks (portions) | \(0.114 \pm 0.147 \) p.013 | Baseline body mass index (kg/m\(^2\)) | | |
| Depressive symptoms (1 = low level to 5 = high level) | \(1.539 \pm 0.142 \) p.016 | Baseline body mass index (kg/m\(^2\)) | | |
| Birth length (cm) | \(0.422 \pm 0.139 \) p.018 | Baseline body mass index (kg/m\(^2\)) | | |

B: Subjects with weight gain (delta weight > 2 kg)

|                  | Women with increased weight \( (n = 490) \) | Men with increased weight \( (n = 455) \) |
|------------------|------------------------------------------|------------------------------------------|
| Medication for anxiety (0 = no, 1 = yes) | \(3.535 \pm 0.138 \) p.001 | Height (cm) | \(0.193 \pm 0.253 \) p.<.001 |
| Occupational status (1 = low to 3 = high) | \(-1.054 \pm 0.134 \) p.002 | Monthly use of french fries (portions) | \(0.229 \pm 0.158 \) p.<.001 |
| Baseline body mass index (kg/m\(^2\)) | \(8.231 \pm 0.132 \) p.007 | Monthly use of sweet cookies (portions) | \(-0.088 \pm 0.154 \) p.001 |
| Monthly use of sugar-sweetened soft drinks (portions) | \(0.088 \pm 0.125 \) p.003 | Age (years) | \(-0.139 \pm 0.141 \) p.002 |
| Childhood body mass index (kg/m\(^2\)) | \(0.617 \pm 0.122 \) p.011 | Divorced between the years 1992–2001 (no = 0, yes = 1) | \(2.769 \pm 0.131 \) p.003 |
| Frequency to add salt (NaCl and/or KCl) to the food (1 = never to 3 = always) | \(1.345 \pm 0.117 \) p.005 | Monthly use of cereals (portions) | \(-0.068 \pm 0.127 \) p.004 |
| Children (number) | \(-0.443 \pm 0.117 \) p.006 | Milk use (categorized 1 = low to 3 = high) | \(0.782 \pm 0.126 \) p.004 |
| Childhood family income (1 = low to 8 = high) | \(-0.257 \pm 0.106 \) p.013 | Depressive symptoms (1 = low level to 5 = high level) | \(1.172 \pm 0.124 \) p.005 |
| Height (cm) | \(0.082 \pm 0.103 \) p.015 | Childhood home location (1 = city centre to 4 = rural areas) | \(0.563 \pm 0.108 \) p.015 |
| Dependence of RD (1 = low level to 5 = high level) | \(-0.801 \pm 0.083 \) p.047 | Baseline body mass index (kg/m\(^2\)) | \(7.588 \pm 0.101 \) p.024 |

Linear stepwise regression with fixed baseline BMI (p < 0.05 for entry and p > 0.1 for removal). \( a \) Log-transformed, \( b \) Z-scored or \( c \) categorized variables were used in statistical analyses. Variables offered for the models are presented in Table I. Strongly associating variables, such as occupational and educational status were not allowed to enter into the same model. In such a case, the variable showing the strongest crude correlation with the weight change was offered for the model. RD, reward dependence.
Table III. Odds ratios for incident obesity (BMI ≥ 30 kg/m²) in which initially non-obese subjects (BMI < 30 kg/m²) were followed for six years.

| Baseline or childhood variables                                      | OR  | Lower | Higher | p     |
|---------------------------------------------------------------------|-----|-------|--------|-------|
| **Women (63 obese of 823 females, 7.7%)**                           |     |       |        |       |
| Standardized (Z-score) log-transformed variables:                   |     |       |        |       |
| Body mass index (kg/m²)                                            | 14.4| 7.8   | 26.4   | <0.001|
| Serum insulin (μU/mL)                                               | 1.93| 1.36  | 2.74   | <0.001|
| Serum adiponectin (μg/mL)                                          | 1.67| 1.15  | 2.43   | 0.007 |
| Non-standardized variables:                                        |     |       |        |       |
| Monthly use of french fries (portions)                             | 1.19| 1.01  | 1.39   | 0.033 |
| Exploratory excitability of NS (1 = low level to 5 = high) level)  | 1.99| 0.99  | 4.01   | 0.05  |
| **Men (61 obese of 689 males, 8.9%)**                               |     |       |        |       |
| Standardized (Z-score) log-transformed variables:                  |     |       |        |       |
| Body mass index (kg/m²)                                            | 39.3| 16.8  | 92.4   | <0.001|
| Serum adiponectin (μg/mL)                                          | 1.64| 0.12  | 2.38   | 0.010 |
| Serum amyloid A-1 (μg/mL)                                          | 1.42| 1.03  | 1.96   | 0.034 |
| Serum sex hormone binding globulin (nmol/L)                       | 1.41| 0.98  | 2.03   | 0.07  |
| Non-standardized variables:                                        |     |       |        |       |
| Milk use (categorized 1 = low to 3 = high)                         | 2.20| 1.36  | 3.56   | 0.001 |
| Smoker (0 = no, 1 = yes)                                           | 0.28| 1.12  | 0.65   | 0.003 |
| Monthly use of french fries (portions)                             | 1.13| 1.01  | 1.25   | 0.027 |
| Childhood family income (1 = low to 8 = high)                     | 0.81| 0.66  | 1.00   | 0.047 |

Odds ratios were expressed for one SD increase in the predictor variable (standardized log-transformed variables), or for one categorical unit increase (non-standardized variables). Baseline BMI was always included in the model. Forward method (p-entry = 0.07 and p-removal = 0.15) was used for rest of the variables described in Table 1. Missing values were replaced by the series mean. CI, confidence interval; NS, novelty seeking; OR, odds ratio.

$[\beta] = -0.175, p < 0.001$), childhood BMI ($\beta = 0.112, p = 0.003$), high serum insulin level ($\beta = 0.103, p = 0.006$), low unemployment ($\beta = -0.095, p = 0.003$), low occupational status ($\beta = -0.092, p = 0.006$), low consumption of sweet cookies ($\beta = -0.091, p = 0.005$), high serum total adiponectin level ($\beta = 0.089, p = 0.008$), medication for anxiety ($\beta = 0.088, p = 0.007$), low childhood family income ($\beta = -0.083, p = 0.012$), older age ($\beta = 0.081, p = 0.014$), low level of reward dependence ($\beta = -0.079, p = 0.014$), high extravagance of novelty seeking ($\beta = 0.074, p = 0.023$) and additional salt consumption ($\beta = 0.064, p = 0.045$) were associated with the weight gain (in the order of importance). In men ($n = 792$), low consumption of cereals ($\beta = -0.148, p < 0.001$), low bBMI ($\beta = -0.118, p = 0.005$), younger age ($\beta = -0.097, p = 0.008$), child BMI ($\beta = 0.082, p = 0.046$) and high stature ($\beta = 0.077, p = 0.029$) were associated with the weight increase. In women, the model accounted for 8.5% and in men 5.2% of the variation in the weight change.

**Age stratification**

24 to 27 years. In women ($n = 265$), employment, low bBMI, low occupational status, high use of sweet beverages, depressive symptoms, and birth length were linked to a weight increase (in the order of importance, Table IIA). In men ($n = 226$), the variables associated with the weight gain were high screen-time, high use of french fries, unemployment, low bBMI and high stature. In women, the model accounted for 13.4% and in men 9.6% of the variation in the weight change.

30 to 39 years. In women ($n = 658$), low childhood family incomes, home location in urban areas, low educational status, low use of sweet cookies, high stature, medication for anxiety, and low level of attachment of reward dependence were associated with a weight increase (accounting for 8.4% of the variation in the weight change) (in the order of importance, Table IIA). In men ($n = 566$), low use of cereals, high childhood BMI, and low bBMI were linked to weight gain (accounting for 4.4% of the variation in the weight change).

**Subjects with increased weight (delta weight > 2 kg)**

When these women ($n = 490$) (Table IIB) were assessed separately, the following factors were most clearly associated with weight gain: Medication for anxiety, low occupational status, bBMI, high use of sweet beverages, childhood BMI, additional salt use, low number of children, low childhood family incomes, high stature and low level of dependence of reward dependence (in the order of importance). In men ($n = 455$), high stature, high use of french fries, low use of sweet cookies, young age, recent divorce, low use of cereals, high milk use, depressive symptoms, childhood home location in rural areas, high bBMI and unemployment were associated with the
greater weight gain. In women, the model accounted for 18.7% and in men 20.6% of the variation in the weight change.

**Models using pure stepwise regression**

Alternative models with pure stepwise regression (bBMI was also estimated with the stepwise method) provided rather similar outcomes with the primary data presented above (data not shown). However, hormonal variables (in particular serum total adiponectin level in women and SHBG in men) seemed to enter into several multivariable models (instead of bBMI). In addition, the impact of childhood BMI was less pronounced in these models (data not shown).

**Models for incident obesity**

Odds ratios for incident obesity (BMI ≥ 30 kg/m², 63 obese females of 823 women and 61 obese males of 689 men) showed that high bBMI, high serum insulin and total adiponectin levels, and high intake of french fries were the risk factors in women (Table III). In men, high bBMI, high serum adiponectin and amyloid A-1 levels, high milk use, non-smoking, high use of french fries, and low childhood family incomes were the risk factors for incident obesity.

**Discussion**

Our purpose was to identify the individual factors associated with a weight change and incident obesity in young and middle-aged Finnish adults. The findings can be summarized as follows: The weight gain of young men started earlier than in women, and young age was a risk factor only in men. Anthropometric variables, i.e. bBMI (baseline BMI) (low bBMI predicted weight gain and high bBMI incident obesity) and/or height; personality, i.e. a high tendency towards novelty seeking or its subscales and a low value towards reward dependence or its subscales in women, and depressive symptoms and medication for anxiety in particular in women; sedentary behavior, i.e. high screen-time in the youngest men; and environmental factors, i.e. low educational and occupational status and employment in women, and unemployment in men, were linked to weight gain. In respect to childhood factors, in particular low family incomes in women, and high childhood BMI in both sexes were important risk factors. In contrast, the impacts of physical activity and the genetic BMI risk factor score were very low. Previously, some gene-based relationships with adulthood obesity have been characterized in the YFS [11]. In addition, unhealthy diet (i.e. low consumption of cereals in men, and high use of french fries in men and women with incident obesity, and additional salt (NaCl and/or KCl) consumption in women with the weight gain) seemed to exert some impact on the weight change.

Young adults are more vulnerable to experience a weight gain than their older counterparts [27]. Some critical steps have been identified, i.e. puberty, teenagers leaving home to go to university/college, couples in the early stages of cohabitation, pregnancy, smoking cessation and the child rearing years, all probably being associated with some increased consumption of food and less physical activity [3].

As anticipated, low bBMI predicted weight gain in most of the models investigated. Nonetheless, our findings reveal that high childhood BMI might independently (following adjustment for bBMI in adulthood) predict a weight gain. Earlier, we have detected a link between child BMI and adulthood obesity [11]. Furthermore, it has been shown that young adult age is associated with increased weight gain [7,27]. On the basis of the present findings, this seems to be the case only in men in the age range investigated.

Low SES has been associated with a weight gain in adolescents [28] and young adults [29]. In the YFS, low childhood SES and low school performance have predicted higher obesity in adults [30,31]. Thus, the present findings from women support these previous observations. However, the reasons why low educational or occupational status can provoke a weight gain will require further detailed investigation. Some of the present study variables which parallel low SES, i.e. low childhood family income (in women) and childhood home location in rural areas (in men with weight gain) were also associated with weight gain. Recent unemployment was associated with a weight loss in women, but with a weight gain in men.

In respect to temperament-related variables, it has been noted previously in the YFS that high novelty seeking and low tendency toward reward dependence are associated with increased weight gain [16]. The present study supported these published findings since a high level of extravagance of novelty seeking (women as a single group), and low level of attachment (older women, Table IIA) or dependence (women with > 2 kg weight gain, Table IIB) of reward dependence were independently associated with a weight gain in women. The use of the subscales instead of the total scale helps to pinpoint which dimensions inside the temperament traits are responsible for the associations between temperament and BMI found in previous studies [16]. As discussed previously [16], high novelty seeking has been linked to snacking and impulsivity, i.e. stressed individuals with high novelty seeking might be particularly prone to succumb to the temptations of unhealthy comfort foods. There may be also neurobiological mechanisms to explain why stressed people experience...
eating as being more rewarding. On the other hand, a high reward dependence is characterized by the need for social acceptance, and for this reason, the presence of cultural pressure may encourage subjects with high reward dependence to reduce their weight [16]. In the present study, a high score for depressive symptoms and medication for anxiety were also associated with the weight gain. Previously, the use of some anti-psychotic drugs has also been linked to a weight gain in young adults [32].

There seem to be inverse associations between serum SHBG levels and the waist-to-hip ratio [33], and serum total adiponectin concentration and the metabolic syndrome [24], but also between these variables and the bBMI (Table I). In contrast, serum total adiponectin (in men and women), SHBG levels (a trend in men) and insulin (in women), were directly associated with incident obesity (Table I). In multivariable models among women (as a single group), serum total adiponectin and insulin levels were also linked with the weight increase. Since low bBMI seems to predict weight gain in our population, it is plausible that high total adiponectin levels (among those subjects with low bBMI) could be directly associated with a future weight gain. In earlier studies, adiponectin and high molecular weight adiponectin [34] have been related to insulin sensitivity. We analyzed serum total adiponectin and insulin levels, and noted that there was an inverse cross-sectional relationship between these two variables at baseline.

With regard to dietary variables, the inverse association of consumption of cereals with the weight gain in men was the most evident finding, but in addition, additional salt intake, consumption of sugar-sweetened soft drinks (e.g., in women with weight gain), and also that of french fries (particularly in men and women with incident obesity) and milk (in men with weight gain and incident obesity) appeared to have some impact as risk factors. Earlier in the YFS, an increasing frequency of consuming sugar-sweetened soft drinks from childhood to adulthood has been shown to associate with adulthood overweight in women [35]. Overall, these food/nutrient findings are rather consistent with the previous observations [9,19]. In contrast, total alcohol use (expressed as drinks per day) did not show any association with the weight change in the present study. It is important to note that those individual food groups which provide associations with the weight change may simply act as indicators of the dietary or living habits in general. Thus, their independent role as direct weight regulators may be low.

A daily duration of 45–60 min of moderate intensity physical activity has been claimed to prevent the transition from normal weight to overweight or obesity [36]. In the CARDIA study, high intensity physical activity for 20 years seemed to combat the weight gain in young women, aged 18–30 years at baseline [37]. In the models examined in the present study, high leisure-time physical activity and the intensity of the physical activity were not associated with the weight change. A recent review also concluded that despite the well-established health benefits of physical activity, it may not be a key determinant in preventing weight gain [38]. On the contrary, the time used to watch TV + play computer games (screen-time) was associated with gaining weight in the youngest men participating in the present study. Overall, this sedentary behavior which is also associated with the consumption of unhealthy energy-rich food items [39] seems to be more important than physical activity in the regulation of weight especially in young men.

As a shortcoming, there were quite a high number of potential determinants of weight change for which there were rather few subjects with complete data, in particular in the sub-group analyses, and this could lead to inappropriate statistically significant associations. In addition, some determinants, such as nutrition or physical activity, were difficult to measure. Typically, there is a tendency for overweight/obese subjects to underestimate their consumption of sweets and to overestimate their amount of physical activity. Furthermore, low use frequency of sweets may result in high portion sizes when the sweets are consumed. These possibilities may explain some of the unexpected or non-existing associations characterized. Since body composition was not assessed, it is unclear whether an increase in muscle mass might partially explain the weight gain in young men. Since it was an observational study, the YFS was not set up to establish causality. Furthermore, the generalizability of the findings is limited to white European subjects [17]. In addition, we did not investigate the effects of inadequate sleep [40], smoking cessation [41], initial exposure to contraceptive use [12], or energy intake (the short dietary questionnaire did not permit this kind of detailed investigation) on the weight gain. Lack of data reflecting energy intake (and expenditure) may at least partly explain why the multivariable regression models explained only from 4.4–20.6% of the variation in the weight change. The main strength of the YFS is its longitudinal study design from childhood to adulthood with its large study population and its high participation rate. Due to inadequate resources for reporting, these important data have not been published earlier.

In summary, our findings suggest that childhood factors, such as BMI and family income, and adulthood factors, such as BMI, educational and occupational status, temperament, mental problems, sedentary behavior, and certain nutrients play key roles in weight regulation, some in women, others in men, and many in both sexes.
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Supplementary Material available online

Supplementary Table I and short descriptions of the temperament variables used.