PARENTAL SEPARATION AND OFFSPRING MORBIDITY IN ADULTHOOD: A DESCRIPTIVE STUDY OF THE NORTHERN FINLAND BIRTH COHORT 1966.

Heidi Varis[a,b,*], Maria Hagnäs[a,b,c], Ilona Mikkola[b], Tanja Nordström[a,d,e], Katri Puukka[f], Anja Taanila[a,**], Sirkka Keinänen-Kiukaanniemi[a,d,g,h **]

[a]Center for Life Course Health Research, University of Oulu, Finland

[b]Rovaniemi Health Centre, Rovaniemi, Finland

[c]Endocrine Section, Department of Clinical and Experimental Medicine, University of Catania, and Diabetes and Obesity Unit, Garibaldi Hospital, Catania, Italy

[d]Medical Research Center Oulu, Oulu University Hospital and University of Oulu

[e]Infrastructure for Population Studies, Faculty of Medicine, University of Oulu

[f]NordLab Oulu, Oulu University Hospital and Department of Clinical Chemistry, University of Oulu

[g]Unit of Primary Care, Oulu University Hospital, Oulu, Finland

[h]Healthcare and Social Services of Selänne, Pyhäjärvi, Finland

[*]Corresponding author at: University of Oulu, Center for Life Course Health Research, P.O. Box 8000, FI-90014 University of Oulu, Finland, Tel. +358 294 48 0000, Fax +358 8 344 084

E-mail: hvaris@student.oulu.fi

[**] With Equal Contribution

Abstract

Aims

Rates of parental separation have increased dramatically in recent decades. We evaluated the association of individuals' childhood family structure with their somatic health over 46- years of follow-up.

Methods

Data were drawn from the Northern Finland Birth Cohort, an ongoing project in which 12 058 participants born in 1966 have been followed from their 24th gestational week. Based on information supplied at age 14, family structure was categorized as 'single-parent family' and 'two-parent family'. The anthropometric information, data from blood samples and medical history were collected from postal questionnaires and clinical examinations routinely performed at the ages of 31 and 46 years.

Results

The study population comprised a total of 10 895 individuals; 85% (n=9253) were offspring of two-parent families and 15% (n=1642) of single-parent families. Type 2 diabetes (p=0.032) or prediabetes (p=0.007), psychoactive drug problem (p<0.001) and sexually transmitted diseases (p<0.001), were more common in the single-parent family group than in the participants from two-parent families. Additionally, among males back diseases (p=0.002), and among females hypertension (p=0.003) and ovary infection (p=0.024) were more frequent in individuals affected by parental death than in those from two-parent families.

Conclusions

Our results indicate the association of childhood family structure with offspring morbidity during 46- years follow-up. The lifetime morbidity was observed to be higher among offspring from single-parent family compared to two-parent family offspring. Public and

scientific concern about the consequences of parental separation on the offspring' health exist, therefore support from health care professionals and society is warranted.

Keywords

Family structure; Morbidity; Offspring; Parental Divorce; Physical Health; Single-parent

Background and aims

An individual's family structure is important for the development of their emotional and physical health [1]. It has been observed that the two-parent family model (with both parents living in the same household as the offspring) is better for the offspring's development than the single-parent family [2]. Divorce rates have increased significantly in the European countries since the 1960s [3]. Among families with children the proportion of single-parent families make up approximately 14% of all families in Europe [4]. More than half of all divorces happen when the offspring is under the age of 18 years [5].

All types of parental separations are associated with deficits in the offspring's health and well-being from childhood to adulthood [6]. Even when parental separation occurs during the prenatal period, it can affect some subsequent health conditions in the offspring [7]. One study found that children affected by parental divorce were 50% more likely to develop health problems than children from a two-parent family [8]. Other studies have observed that the offspring from divorced families have more somatic health problems and diseases, such as overweight and obesity [7,9] and asthma [9] than those who live with both parents. However, some studies have found that the offspring's cardiometabolic health is unaffected by parental separation [10,11].

Associations between parental separation and mental health problems in the offspring have been reported [6,8,12]. Children living in a single-parent family are twice as likely to exhibit stress [5] and lower levels of well-being [5,6] than those who live with both parents. Additionally, parental separation may have an impact on the offspring's drug, tobacco and alcohol use [10,11,13]. Parental death has also been shown to associate with lower levels of

well-being and increased number of psychiatric diagnosis in the offspring [6,14]. Young adults who have suffered parental bereavement often exhibit problems similar to those who have experienced parental divorce, such as poor mental and physical health and health risk behavior [15].

Although previous studies have examined the associations between parental separation and the mental and somatic health of the offspring, to our knowledge, there is a lack of large—scale comprehensive studies investigating the long-term association of parental separation with somatic health of the offspring. In the present descriptive study, we therefore examined the association between individuals' family structure at the age of 14 years (two-parent vs. single-parent family with subgroups) and their morbidity over a 46-year follow-up period in the context of a large birth cohort study. Our hypothesis was that lifetime morbidity is more common among offspring of single-parent family compared to offspring of two-parent family.

Methods

Study design

This retrospective study examined data gathered as part of the Northern Finland Birth Cohort 1966 (NFBC1966) project. Based in the two northernmost former provinces of Finland (Oulu and Lapland), the NFBC1966 is a large, ongoing prospective, general population-based longitudinal birth cohort that comprises 96.3% of all live births in the regions with expected delivery date between first January 1966 and 31st December 1966 (initially a total of 12 231 individuals) [16]. Data on all members of the cohort were first collected during the 24th

gestational week and subsequently at predetermined time points (birth and at ages one, 14, 31 and 46 years). Postal questionnaires were sent to participants at the ages of 14, 31, and 46 years and clinical examinations were performed at the ages of 31 and 46 years [17]. All participants provided written consent, and the research plan was approved by the Ethics Committee of the Northern Ostrobothnia Hospital, Oulu, Finland.

Study protocol

Based on information supplied by the cohort members at age 14, the family structure of each was categorized as being from a two-parent family or a single-parent family. The single parent families were further subdivided as follows: ‘one parent not living at home’; ‘father or mother deceased’ and ‘no information on father’.

The prevalence of diseases was based on information from the NFBC questionnaires at the ages of 31 and 46. For the purposes of the present study, an individual was classified as having any particular disease if they reported having received a diagnosis at either 31 or 46 years of age, or both. The participants diagnosed conditions were grouped into 24 classifications, which in turn were placed into seven categories (Supplementary Table 1). The postal questionnaires at the ages of 31 and 46 years differed in terms of how they asked about diabetes and thyroid diseases. At age 31, the relevant questions were general ”diabetes”, and ”thyroid diseases”, whereas the questions at age 46 specifically asked for information on type 1 diabetes, type 2 diabetes, hypothyroidism and hyperthyroidism. Multimorbidity was defined as the presence of at least two chronic diseases.

Data on the following variables were recorded by a nurse or physician at the clinical examinations at the age of 46 years: Weight (to an accuracy of 0.1 kg), height (to an accuracy

of 0.1 cm) and waist circumference (WC) (cm) were measured in light clothing [17]. Body mass index (BMI) was calculated as the weight in kilograms divided by the square of the height in meters. In accordance with the classification provided by the World Health Organization, BMI (kg/m^2) was categorized as underweight (BMI <18.5 kg/m^2), normal weight (BMI 18.5–24.9 kg/m^2), overweight (BMI 25–29.9 kg/m^2) or obese (BMI ≥ 30 kg/m^2). WC and BMI were additionally examined according to gender. Systolic and diastolic blood pressure levels (mmHg) were measured in a seated position after 15 minutes of rest using an automated device (Omron Digital Automatic Blood Pressure Monitor Model M10-IT; Omron Kyoto Japan) and an appropriately sized cuff on the right arm. The measurements were performed twice at the age of 31 years and three times at age 46 years and thereafter the mean of blood pressure values was calculated [17].

At age 46 years blood samples were collected after an overnight fasting period. They were centrifuged and analysed immediately in NordLab Oulu, a testing laboratory (T113) accredited by Finnish Accreditation Service (FINAS) (EN ISO 15189). Fasting plasma glucose (mmol/l) was analysed by as previously described [18]. Plasma total cholesterol (mmol/l), high-density lipoprotein (HDL) cholesterol (mmol/l), low-density lipoprotein (LDL) cholesterol (mmol/l) and triglycerides (mmol/l) were determined using an enzymatic assay method. The concentration of HbA1c and the concentration of total hemoglobin were measured by immunochemical assay method. The ratio is reported as percent HbA1c (NGSP). (All methods by Advia 1800, Siemens Healthcare Diagnostics Inc., Tarrytown, NY, USA).

Metabolic syndrome (MetS) was defined according to the International Diabetes Federation criteria as follows: abdominal obesity (WC ≥94 cm in men or ≥ 80 cm in women) and at least two of the following: blood pressure ≥ 130/85 or previously diagnosed hypertension; HDL

cholesterol < 1.03 mmol/l in men or < 1.29 in women; triglycerides > 1.7 mmol/l fasting plasma glucose level ≥ 5.6 mmol/l or previously diagnosed type 2 diabetes [19]. The present study did not take information on diabetes treatment into account when making the classifications. Participants who did not report a diagnosis of diabetes in the questionnaire at the age of 46 were screened retrospectively for pre-diabetes and diabetes by examination of their fasting plasma glucose (FPG) and HbA1c

We examined disease prevalence and anthropometrics in the before mentioned categories of family unit to determine any impact of family structure on later clinical outcomes. To get a better overview of the present study sample, we included sociodemographic variables in our analyses.

Statistical methods

Continuous clinical outcome measures are presented as mean and standard deviation (SD), and categorical questionnaire variables as proportions. Analysis of variance (ANOVA) was used to evaluate the associations between study groups and clinical outcome measures, while associations between study groups and categorical variables were examined using contingency tables with $\chi 2$-test, or Fisher's Exact Test when appropriate. Differences between study groups among continuous variables were evaluated with Tukey's Honest Significant Difference test or Games-Howell post hoc tests. The Benjamini-Hochberg (B-H) false discovery rate procedure was used to control for type I error caused by multiple comparisons in the contingency tables. All p-values (p and B-Hp) were two-tailed and a p-value <0.05 was considered statistically significant. Statistical analyses were performed using IBM SPSS Statistics, Version 25 (IBM Corporation and its licensors 1989, 2017). Because of the large

study size (also after omitting the missing data) and numerous of non-continuous variables, missing data were omitted from the analysis rather than being imputed.

Results

There was initially a total of 12 231 individuals in NFBC and at the age of 14 total respondents were 10 895 individuals. In the present study, two groups were formed based on info at age 14: 1) two-parent family (n=9253) and 2) single-parent family (n=1642). Single-parent family was additionally divided into three subgroups: 1) father or mother deceased (n=710), 2) one parent not living at home (n=882) and 3) no information on father (n=50). Subgroups 'one parent not living at home' and 'no information on father' were combined (n=932). Number of respondents in follow-ups at 31-year was 8767 (71.7% of the initial study population) and at 46-year 6868 (56.2% of the initial study population) from postal questionnaires and 6033 (49.3% of the initial study population) and 5861 (47.9% of the initial study population), respectively, from clinical examinations [20]. The number of responses varied between participants.

Table 1 shows the cohort's anthropometric characteristics and cardiovascular measurements at the age of 46. The mean levels of LDL cholesterol and triglycerides were significantly higher in individuals from a single-parent family than those from a two-parent family (p=0.044 and p=0.006, respectively). Furthermore, mean FPG was significantly higher in the 'one parent not living at home' subgroup (p=0.012) than in the 'two-parent family' group.

Table 2 presents the descriptive statistics for the sociodemographic variables of the study population. Information from postal questionnaires completed by the cohort mothers during

pregnancy and at the offspring ages of 14 and 46 years. During pregnancy, almost the half of the two-parent families lived in an owner-occupied dwelling, while in the single-parent family subgroup ‘one parent not living at home/no information on father’ only one-third of the families occupied their own property (p<0.001). Mothers in the two-parent family group were significantly older during pregnancy than mothers in the single-parent family subgroup ‘one parent not living at home/no information on father’ (27.8 and 25.3 years, respectively [p<0.001]). Occupational status during pregnancy was higher among mothers in the single-parent family subgroup ‘one parent not living at home/no information on father’ than in those from two-parent families (p<0.001). Grand multiparity was more frequent in two-parent families than in the single-parent family subgroup ‘one parent not living at home/no information on father’ (p<0.001). The offspring of two-parent families were significantly more likely to be in a relationship and less likely to be divorced or separated than those in the ‘one parent not living at home/no information on father’ single-parent family subgroup (p=0.007). These associations remained statistically significant after B-H correction.

The lifetime prevalence of diseases and health conditions of the study population at the age of 31 and 46 years, by childhood family structure at the age of 14 years are shown in Table 3. When compared with the two-parent family group, the following conditions were significantly more common in the subgroup ‘one parent not living at home/no information on father’ than in the two-parent family group: psychoactive drug problem (p<0.001) and sexually transmitted diseases (p<0.001). These associations remained statistically significant after B-H correction. (Table 3)

Table 4 shows the lifetime prevalence of the diseases and conditions stratified by sex. The following diseases were significantly more common in female offspring in the single-parent family subgroup ‘one parent not living at home/no information on father’ than in females from two-parent families: metabolic syndrome (p=0.014), type 2 diabetes (p=0.032),

hypothyroidism (p=0.005), ophthalmopathy or eye injury (p=0.009), nervous system diseases (p=0.019) and respiratory diseases (p=0.003). Only the association with hypothyroidism and respiratory diseases remained significant after B-H correction. Additionally, among females, obesity (p=0.040), hypertension (p=0.003), type 2 diabetes (p=0.030) and ovary infection (p=0.024) were more common in the 'father or mother deceased' subgroup than in the two-parent family group, however none of these associations remained significant after B-H correction. Prediabetes (p=0.007), degenerative back disease or other back diseases (p=0.047), depression (p<0.001), bone fractures (p=0.005) and multimorbidity (p=0.040) were significantly more common in the male offspring from the single-parent family subgroup 'one parent not living at home/no information on father' than in males from two-parent families. Only the associations with prediabetes, depression and bone fractures remained significant after B-H correction. Furthermore, degenerative back disease or other back diseases (p=0.002) were more common in males from the 'father or mother deceased' subgroup than in males from two-parent families, however this association did not remain significant after B-H correction. (Table 4)

Discussion

In this large prospective population-based study of 10 895 individuals, we observed that adulthood lifetime morbidity was associated with childhood family structure, being higher in the offspring of single-parent families than in the offspring of two-parent families. This pattern was particularly apparent in type 2 diabetes or prediabetes, use of psychoactive drugs and sexually transmitted diseases. Morbidity was especially high in the combined subgroup 'one parent not living at home/no information on father' and among females in single-parent

families. Although, the significance of these effects may be considered minor at the individual level, they are relevant at the overall population level.

Metabolic syndrome, a cluster of obesity-related cardiometabolic risk factors, was more prevalent among females in the combined subgroup ‘one parent not living at home/no information on father’ than in the two-parent family group. We also observed that LDL-cholesterol and triglyceride values were higher in the single-parent family group than in the two-parent family group. This aligns with a previous report that parental loss in childhood is associated with the development of metabolic syndrome in adulthood [21], however controversial findings have been reported [11]. An elevated prevalence of type 2 diabetes has been reported in adults that were affected in childhood by parental death [22] or separation [23], which our findings confirm. The increased cardiometabolic risk factors among single-parent family offspring might be partly explained by the higher prevalence of type 2 diabetes.

We observed that the drinking and other drug problems were more common in the single-parent group. This aligns with previous findings that people affected by parental separation start alcohol consumption earlier [24], and are more likely to develop problematic drinking habits in adulthood [10]. Earlier alcohol initiation is linked with a higher risk of alcohol dependence in later life [25], which might partly explain the higher prevalence of problem drinking in adults who were affected by parental separation in childhood. Our finding regarding psychoactive drug problems is supported by the previous report from the NFBC1966 cohort that overdosing by drugs resulting to hospitalization is more common in the offspring from single-parent families [13]. We also observed a higher prevalence of sexually transmitted diseases in the single-parent family group. One previous study has also

found that people who have experienced at least one childhood adversity, such as parental separation, have a higher likelihood of sexually transmitted diseases [12]. Furthermore, parental separation has been linked with sexual risk behaviour patterns such as earlier sexual debut [12, 24] and having a greater number of sexual partners [26].

Parental separation has been shown to have both short- and long-term negative effects on several domains of the offspring's societal functioning [6]. The results of the present study appear to support these findings.

This study has several strengths, including its large cohort size and its longitudinal design. The NFBC1966 cohort is a representative, unselected population-based sample of Finnish adults. A further strength is the lifetime study setting of the NFBC1966 project, which includes multiple cross-sectional clinical examinations. The participation rate remained high during the 46 years of follow-up and a large variety of diseases were taken in account in the present analysis. Blood samples were examined and measurements of other relevant components were taken by trained physicians or study nurses using identical procedures. Also, all blood samples were analysed in the same laboratory by the same method.

Family status was recorded at the time when the participants were 14 years old. However, a potential limitation of the study is presented by the fact that no subsequent parental separation was documented and therefore the number of offspring who were affected by parental separation could be higher than reported here. Similarly, no information was available regarding later reconciliation or the introduction of step-parents, any of which might have had an effect on the participants' family dynamics. The formation of our single-parent family subgroups was hindered by small numbers of participants, which necessitated the

combination of the ‘one parent not living at home’ and ‘no information on father’ groups. It must be acknowledged that the practical effect on the offspring’s daily life may have varied significantly between these subgroups: when the father is not living at home he could nevertheless be a part of the offspring’s life, even if not on a daily basis. This is very different to there being no information about the father – in this situation the father has no contact whatsoever with the offspring. A further potential weakness of the study may be that disease diagnoses (although made by a doctor) were self-reported by the participants, which may have led to inaccuracies in the data. Our study was descriptive in design, and therefore did not control for factors that may confound findings in this complex area. Therefore, the influence and possible causal roles of such factors should be explored in future studies.

Conclusions

In conclusion, to the best of our knowledge, we can state that our study was the first to extensively investigate the prevalence of somatic diseases in the offspring of single-parent families over 46 years of follow-up. The findings of our descriptive study suggest that some somatic diseases are more prevalent in individuals brought up in single-parent families than in those from two-parent families. Clinicians should be aware of the potential association between patients’ family structure and their somatic health, and consider targeted disease screening in people from single-parent families. Active research on the associations of parental separation with the offspring’s somatic health should be continued, particularly considering the influence of confounding factors. There is public and scientific concern about this issue, and therefore support from health care professionals and society is warranted.

Funding

The Northern Finland Birth Cohort 1966 was supported by the University of Oulu Grant no. 65354 and no. 2400069, Oulu University Hospital Grant no. 2/97, 8/97 and no. 24301140 , Ministry of Health and Social Affairs Grant no. 23/251/97, 160/97, 190/97, National Institute for Health and Welfare, Helsinki Grant no. 54121, Regional Institute of Occupational Health, Oulu, Finland Grant no. 50621, 54231 and ERDF European Regional Development Fund Grant no. 539/2010 A31592. Heidi Varis has received funding from the Juho Vainio foundation and the Finnish General Practice foundation.

Conflict of interest

None declared.

References

[1] Troxel WM and Matthews KA. What are the costs of marital conflict and dissolution to children's physical health?. Clin Child Fam Psychol Rev 2004;7:29-57. DOI: 10.1023/B:CCFP.0000020191.73542.b0.

[2] McMunn AM, Nazroo JY, Marmot MG, et al. Children's emotional and behavioural well-being and the family environment: findings from the Health Survey for England. Soc Sci Med 2001;53:423-40. DOI: 10.1016/S0277-9536(00)00346-4.

[3] Amato PR. The Consequences of Divorce for Adults and Children: An Update. Društvena istraživanja - Časopis za opća društvena pitanja 2014;23:5-24. DOI: 10.5559/di.23.1.01.

[4] Anonymous Eurostat: https://ec.europa.eu/eurostat/statistics-explained/index.php/Household_composition_statistics (Accessed 30 Oct 2020).

[5] Dissing AS, Dich N, Andersen AN, et al. Parental break-ups and stress: roles of age & family structure in 44 509 pre-adolescent children. Eur J Public Health 2017;27:829-34. DOI: 10.1093/eurpub/ckx040.

[6] Amato PR and Keith B. Parental Divorce and the Well-Being of Children: A Meta-Analysis. Psychol Bull 1991;110:26-46. DOI: 10.1037/0033-2909.110.1.26.

[7] Hohwü L, Zhu JL, Graversen L, et al. Prenatal Parental Separation and Body Weight, Including Development of Overweight and Obesity Later in Childhood. PLoS One 2015;10(3):e0119138. DOI: 10.1371/journal.pone.0119138.

[8] Uphold-Carrier H and Utz R. Parental Divorce Among Young and Adult Children: A Long-Term Quantitative Analysis of Mental Health and Family Solidarity. J Divorce Remarriage 2012;53:247-66. DOI: 10.1080/10502556.2012.663272.

[9] Scharte M and Bolte G. Increased health risks of children with single mothers: the impact of socio-economic and environmental factors. Eur J Public Health 2013;23:469-75. DOI: 10.1093/eurpub/cks062.

[10] Lacey R, Kumari M and McMunn A. Parental separation in childhood and adult inflammation: The importance of material and psychosocial pathways. Psychoneuroendocrinology 2013;38:2476-84. DOI: 10.1016/j.psyneuen.2013.05.007.

[11] Soares ALG, Gonçalves H, Matijasevich A, et al. Parental Separation and Cardiometabolic Risk Factors in Late Adolescence: A Cross-Cohort Comparison. Am J Epidemiol 2017;185:898-906. DOI: 10.1093/aje/kwx007.

[12] Anderson J. The impact of family structure on the health of children: Effects of divorce. Linacre Q 2014;81:378-387. DOI: 10.1179/0024363914Z.00000000087.

[13] Sauvola A, Mäkikyro T, Jokelainen J, et al. Single-parent family background and physical illness in adulthood: a follow-up study of the Northern Finland 1966 Birth Cohort. Scand J Public Health 2000;28:95-101.

[14] Haapea M, Nordström T, Räsänen S, et al. Parental death due to natural death causes during childhood abbreviates the time to a diagnosis of a psychiatric disorder in the offspring: a follow-up study. Death Stud 2020; 17:1-10. DOI: 10.1080/07481187.2020.1725928.

[15] Feigelman W, Rosen Z, Joiner T, et al. Examining longer-term effects of parental death in adolescents and young adults: Evidence from the national longitudinal survey of adolescent to adult health. Death Stud 2017;41:133-43. DOI: 10.1080/07481187.2016.1226990.

[16] Rantakallio P. The longitudinal study of the Northern Finland birth cohort of 1966. Paediatr Perinat Epidemiol 1988;2:59-88. DOI: 10.1111/j.1365-3016.1988.tb00180.x.

[17] Keto J, Ventola H, Jokelainen J, et al. Cardiovascular disease risk factors in relation to smoking behaviour and history: a population-based cohort study. Open heart 2016;3:e000358. DOI:10.1136/ openhrt-2015-000358

[18] Similä T, Auvinen J, Puukka K, et al. Impaired glucose metabolism is associated with tooth loss in middle-aged adults: The Northern Finland Birth Cohort Study 1966. Diabetes Res Clin Pract 2018;142:110-19. DOI: 10.1016/j.diabres.2018.05.035.

[19] Alberti G, Zimmet P and Shaw J. The metabolic syndrome—a new worldwide definition. Lancet 2005;366:1059-62. DOI: 10.1016/S0140-6736(05)67402-8.

[20] Nurul Hanis Ramzi, Andrianos M Yiorkas, Sylvain Sebert, et al. Relationship between BMI and emotion-handling capacity in an adult Finnish population: The Northern Finland Birth Cohort 1966. PLoS One 2018;13(9):e0203660. DOI: 10.1371/journal.pone.0203660.

[21] Alciati A, Gesuele F, Casazza G, et al. The Relationship between Childhood Parental Loss and Metabolic Syndrome in Obese Subjects. Stress Health 2013;29:5-13. DOI: 10.1002/smi.1435.

[22] Lown EA, Lui CK, Karriker-Jaffe K, et al. Adverse childhood events and risk of diabetes onset in the 1979 National longitudinal survey of youth cohort. BMC Public Health 2019;19:1007. DOI: 10.1186/s12889-019-7337-5.

[23] Amemiya A, Fujiwara T, Shirai K, et al. Association between adverse childhood experiences and adult diseases in older adults: a comparative cross-sectional study in Japan and Finland. BMJ Open 2019;9:e024609. DOI: 10.1136/bmjopen-2018-024609.

[24] McCutcheon VV, Agrawal A, Kuo SI, et al. Associations of parental alcohol use disorders and parental separation with offspring initiation of alcohol, cigarette and cannabis use and sexual debut in high-risk families. Addiction 2018;113:336-45. DOI: 10.1111/add.14003.

[25] Guttmannova K, Bailey JA, Hill KG, et al. Sensitive Periods for Adolescent Alcohol Use Initiation: Predicting the Lifetime Occurrence and Chronicity of Alcohol Problems in Adulthood. J Stud Alcohol Drugs 2011;72(2):221-231. DOI:10.15288/jsad.2011.72.221

[26] Cleveland HH and Gilson M. The Effects of Neighborhood Proportion of Single-Parent Families and Mother-Adolescent Relationships on Adolescents' Number of Sexual Partners. J Youth Adolesc 2004; 33:319-329.

Table 1. The characteristics of the study population at the age of 46. Subgroups categorized according to their childhood family structure at the age of 14 years.

	Total population n=10 895		**Two-parent family** n=9253 (84.9%)		**Single-parent family** n=1642 (15.1%)			**Single-parent family**								
								One parent not living at home n=882 (8.1%)			**Father or mother deceased** n=710 (6.5%)			**No information on father** n=50 (0.5%)		
	Mean	SD	Mean	SD	Mean	SD	p	Mean	SD	p	Mean	SD	p	Mean	SD	p
Weight (kg)	78.6	16.6	78.6	16.6	78.6	16.6	0.954	79.2	17.5	0.897	78.1	15.9	0.964	76.1	13.8	0.890
Height (cm)	170.9	9.1	171.0	9.1	170.2	9.1	0.040	170.6	9.3	0.869	169.9	9.0	0.155	169.1	8.3	0.750
Body mass index, kg /m^2	26.8	4.9	26.8	4.9	27.0	4.9	0.192	27.1	5.1	0.598	27.0	4.7	0.892	26.7	4.9	0.999
Waist circumference (cm)	91.7	13.5	91.6	13.5	92.3	13.7	0.158	92.8	14.0	0.317	91.9	13.4	0.971	90.5	13.1	0.985
Systolic blood pressure (mmHg)	125.3	16.0	125.5	16.1	123.7	15.6	0.004	123.0	15.6	0.017	124.5	15.6	0.664	123.8	14.4	0.956
Diastolic blood pressure (mmHg)	84.7	10.8	84.7	10.8	84.4	10.8	0.391	84.2	10.6	0.759	84.6	11.0	0.930	83.4	10.7	0.986
LDL-cholesterol (mmol/l)	3.5	0.9	3.4	0.9	3.5	0.9	0.044	3.5	0.9	0.645	3.5	1.0	0.237	3.4	1.0	0.999
HDL-cholesterol (mmol/l)	1.6	0.4	1.6	0.4	1.5	0.4	0.307	1.5	0.4	0.983	1.5	0.4	0.667	1.5	0.4	0.998
Triglycerides (mmol/l)	1.3	0.8	1.3	0.8	1.4	1.1	0.006	1.4	1.3	<0.001	1.3	0.7	0.956	1.2	0.6	1.000
Fasting plasma glucose (mmol/l)	5.5	0.9	5.5	0.9	5.6	0.9	0.167	5.7	1.1	0.012	5.5	0.6	0.774	5.4	0.6	0.932

LDL-cholesterol, low density lipoprotein cholesterol; HDL-cholesterol, high density lipoprotein. Data are presented as mean (SD, standard deviation) of the population, except for description of family structure (given as number and percentage). P-value presents the differences between groups. Comparison to two parent family values was performed with Tukey HSD or Games-Howell post hoc tests.

Field Code Changed

Table 2. The descriptive statistics for the sociodemographic variables of the study population. Data were gathered from postal questionnaires completed by cohort mothers during pregnancy and by the offspring at the ages of 14 and 46 years. Subgroups categorized according to childhood family structure at the age of 14 years.

		Two-parent family n=9253 (84.9%)		**One parent not living at home/No inormation on father** n=932 (8.1%)				**Father or mother deceased** n=710 (6.5%)			
		n	%	n	%	p	B-Hp	n	%	p	B-Hp
	Gender										
	Male	4706	50.9	434	46.6	0.012	0.085	351	49.4	0.465	
	Female	4547	49.1	498	53.4			359	50.6		
	Family has owner-occupied dwelling	4114	44.8	279	30.2	<0.001	<0.001	385	54.5	<0.001	<0.001
	Mother's age	27.8 (6.46)		25.3 (5.91)		<0.001		31.5 (7.40)		<0.001	
Pregnancy	**Mother's occupational status***										
	No occupation	2810	31.2	263	29.6			225	32.4		
	Lowest social class	2366	26.3	121	13.6	<0.001	<0.001	242	34.8	<0.001	<0.001
	Highest social class	3822	42.5	504	56.8			228	32.8		
	Number of children in the family										
At the age of 14 years	One child	400	4.4	121	13.2			25	3.6		
	2-4 children	6297	68.8	629	68.4	<0.001	<0.001	362	51.5	<0.001	<0.001
	Grand multiparity (5 or more children)	2458	26.8	169	18.4			316	45.0		
	Education level										
	Basic or less	205	3.6	25	5.1			27	6.5		
	Secondary	3883	68.8	340	69.2	0.199		295	70.9	0.002	0.003
	Tertiary	1560	27.6	126	25.7			94	22.6		
At the age of 46 years	**Marital status**										
	Cohabiting/in relationship	4418	78.4	353	71.9			309	74.3		
	Unmarried	633	11.2	77	15.7	0.007	0.015	51	12.3	0.120	
	Divorced/separated	557	9.9	58	11.8			55	13.2		
	Widower	25	0.4	3	0.6			1	0.2		

Data are presented as number and percentage of the population, except for mother's age (given as mean and SD, standard deviation). *Mother's occupational status was classified: 'No occupation' (housewife), 'lowest social class' (unskilled workers, farmers and farmers' wives) and 'highest social class' (professionals and skilled workers). P-value presents the differences between groups. Differences between study groups and continuous variables were evaluated with Tukey's Honest Significant Difference test or Games-Howell post hoc tests. B-Hp, Benjamini-Hochberg corrected p-value.

Field Code Changed

Table 3. The lifetime prevalence of diseases and health conditions

	Two-parent family n=9253 (84.9%)		One parent not living at home/ No information on father n=932 (8.6%)				Father or mother deceased n=710 (6.5%)			
Diseases and health conditions	n	%	n	%	p	B-Hp	n	%	p	B-Hp
Body mass index (BMI) ***										
Underweight BMI <18.5 kg /m^2	28	0.6	2	0.5	1.000		5	1.4	0.056	
Normal weight BMI 18.5-24.9 kg /m^2	1880	39.0	164	39.1			126	35.2		
Overweight BMI 25-29.9 kg /m^2	1917	39.7	143	34.1	0.192		144	40.2	0.378	
Obese BMI ≥30 kg /m^2	996	20.7	110	26.3	0.074		83	23.2	0.153	
Cardiovascular diseases										
Heart diseases	434	5.7	44	6.2	0.553		29	5.2	0.638	
Hypertension	1675	22.0	151	21.4	0.740		150	26.7	0.010	0.175
Measured blood pressure ≥140/90 ***	1562	32.3	126	30.1	0.354		112	31.4	0.725	
Endocrinological diseases										
Prediabetes ***[a]	706	14.7	73	17.5	0.085		51	14.2	0.926	
Screen detected diabetes	183	3.8	22	5.3			15	4.2		
Diabetes *	98	1.4	10	1.5	0.861		4	0.8	0.322	
Type 1 Diabetes **	40	0.7	0	0	0.072		2	0.5	0.767	
Type 2 Diabetes **	148	2.7	21	4.3	0.043	0.125	15	3.7	0.268	
Metabolic syndrome ***	1479	27.8	153	33.0	0.018	0.070	110	27.0	0.774	
Central obesity ***	3071	64.1	271	65.6	0.556		233	65.3	0.689	
Thyroid diseases *	129	1.8	14	2.2	0.649		11	2.1	0.735	
Hypothyroidism **	244	4.4	37	7.6	0.002	0.013	14	3.4	0.382	
Hyperthyroidism **	47	0.8	8	1.7	0.077		4	1.0	0.777	
Nervous system diseases										
Organ of hearing diseases	878	11.6	81	11.6	1.000		72	12.9	0.374	
Ophthalmopathy or eye injury	1882	24.7	203	28.8	0.016	0.070	148	26.3	0.39	
Nervous system diseases	1714	22.5	193	27.3	0.004	0.020	127	22.6	0.958	
Musculoskeletal diseases										
Rheumatoid arthritis or other arthritis	834	10.9	2	11.6	0.615		66	11.7	0.576	
Degenerative back disease or other back diseases	1852	24.3	193	27.3	0.082		170	30.4	0.001	0.035
Genital, urinary and bowel diseases										
Gastrointestinal diseases	549	7.2	55	7.8	0.595		45	8.0	0.500	
Urethritis	560	7.3	58	8.2	0.408		37	6.6	0.556	
Ovary infection	216	3.2	32	5.0	0.021	0.073	26	5.1	0.029	0.338
Prostatitis	225	3.3	21	3.5	0.812		15	3.0	0.796	
Sexually transmitted diseases	2400	31.5	303	42.9	<0.001	<0.001	183	32.6	0.605	
Mental diseases										
Psychosis	100	1.3	14	2.0	0.171		6	1.1	0.705	
Depression	804	10.5	111	15.8	<0.001	<0.001	49	8.7	0.197	
Other mental disorder	352	4.6	41	5.8	0.163		30	5.3	0.467	
Psychoactive drug problem	205	2.7	39	5.5	<0.001	<0.001	22	3.9	0.108	
Other diseases and health conditions										
Cancer	158	2.1	14	2.0	0.892		14	2.5	0.540	
Anemia	1356	17.8	144	20.5	0.082		107	19.1	0.458	
Respiratory diseases	1326	22.1	154	27.6	0.003	0.018	106	24.6	0.255	
Atopy	2133	34.6	210	37.2	0.230		149	33.5	0.643	
Bone fractures	2393	31.4	250	35.5	0.028	0.089	176	31.4	1.000	
Multimorbidity										
No chronic disease	3956	51.8	325	46.0			269	47.9		
One chronic disease	2461	32.2	236	33.4	0.002	0.014	184	32.7	0.063	
Two or more chronic diseases	1216	15.9	145	20.5			109	19.4		

P-value presents the differences between groups. Comparison to two-parent family values was performed with χ2-test or Fisher Exact Test. Data presented as lifetime prevalences at both time points, except for diabetes and thyroid diseases at the 31* and 46** years. Data collected from clinical examination at the age 46 *** years. [a] Prediabetes was defined in accordance with the Finnish Current Care Guidelines. B-Hp, Benjamini-Hochberg corrected p-value.

Table 4. The lifetime prevalence of diseases and health conditions stratified by sex

	Males										Females									
	Two-parent family n= 4706 (50.9%)		**One parent not living at home/ No information on father** n=434 (46.6%)				**Father or mother deceased** n=351 (49.4%)				**Two-parent family** n= 4547(49.1%)		**One parent not living at home/ No information on father** n=498 (53.4%)				**Father or mother deceased** n=359 (50.6%)			
Diseases and health conditions	n	%	n	%	p	B-Hp	n	%	p	B-Hp	n	%	n	%	p	B-Hp	n	%	p	B-Hp
Body mass index (BMI) ***																				
Underweight BMI <18.5 kg/m2	4	0.2	1	0.6	0.322		1	0.7	0.266		24	0.9	1	0.4	0.433		4	1.9	0.102	
Normal weight BMI 18.5-24.9 kg/m2	642	30.1	51	31.3			40	27.2			1238	46.1	113	44.1			86	40.8		
Overweight BMI 25-29.9kg/m2	1041	48.8	64	39.3	0.186		78	53.1	0.358		876	32.6	79	30.9	0.937		66	31.3	0.632	
Obese BMI ≥ 30 kg/m2	448	21.0	47	28.8	0.187		28	19.0	0.990		548	20.4	63	24.6	0.162		55	26.1	0.040	0.350
Cardiovascular diseases																				
Heart diseases	243	6.5	26	8.8	0.139		17	6.6	0.967		191	4.9	18	4.4	0.665		12	4.0	0.475	
Hypertension	837	22.6	58	19.6	0.237		63	24.3	0.515		838	21.4	93	22.7	0.542		87	28.7	0.003	0.105
Measured blood pressure ≥ 140/90 ***	874	40.8	72	44.2	0.399		56	38.4	0.560		688	25.6	54	21.1	0.112		56	26.5	0.765	
Endocrinological diseases																				
Prediabetes ***[a]	414	19.4	45	27.4	0.007	0.049	25	17.0	0.382		292	10.9	28	11.0	0.795		26	12.3	0.103	
Screen detected diabetes	117	5.5	14	8.5			5	3.4			66	2.5	8	3.1			10	4.7		
Diabetes *	36	1.1	1	0.4	0.517		2	0.8	1.000		62	1.7	9	2.3	0.358		2	0.7	0.322	
Type 1 Diabetes **	25	1.0	0	0	0.254		2	1.1	0.693		15	0.5	0	0	0.635		0	0	0.620	
Type 2 Diabetes **	76	3.0	8	4.1	0.384		4	2.3	0.595		72	2.4	13	4.5	0.032	0.140	11	4.7	0.030	0.350
Metabolic syndrome ***	841	34.9	74	40.2	0.142		55	31.8	0.414		638	21.9	79	28.3	0.014	0.082	55	23.5	0.567	
Central obesity ***	1272	60.0	98	60.9	0.828		85	57.8	0.603		1799	67.3	173	68.7	0.662		148	70.5	0.344	
Thyroid diseases *	19	0.6	0	0	0.393		4	1.7	0.059		110	3.0	14	3.6	0.491		7	2.5	0.636	
Hypothyroidism **	43	1.7	5	2.5	0.256		2	1.1	0.766		201	6.6	32	11.0	0.005	0.044	12	5.1	0.367	
Hyperthyroidism **	12	0.5	3	1.5	0.085		0	0	0.360		35	1.2	5	1.8	0.389		4	1.7	0.353	
Nervous system diseases																				
Organ of hearing diseases	540	14.6	43	14.6	1.000		39	15.1	0.830		338	8.7	38	9.3	0.644		33	11.0	0.174	
Ophthalmopathy or injury	889	24.0	75	25.3	0.598		69	26.6	0.332		993	25.4	128	31.3	0.009	0.063	79	26.1	0.783	
Nervous system diseases	484	13.0	41	13.9	0.694		31	12.0	0.617		1230	31.4	152	37.1	0.019	0.095	96	31.7	0.912	
Musculoskeletal diseases																				
Rheumatoid arthritis or other arthritis	353	9.5	32	10.8	0.466		23	8.9	0.737		481	12.3	50	12.2	0.965		43	14.2	0.328	
Degenerative back disease or other back diseases	978	26.4	94	31.8	0.047	0.235	91	35.4	0.002	0.070	874	22.3	99	24.1	0.401		79	26.2	0.125	
Genital, urinary and bowel diseases																				
Gastrointestinal diseases	186	5.0	15	5.1	0.966		14	5.4	0.780		363	9.3	40	9.8	0.742		31	10.2	0.576	
Urethritis	64	1.7	5	1.7	0.962		4	1.5	1.000		496	12.7	53	13.0	0.860		33	10.9	0.372	
Ovary infection	2	0.1	0	0	1.000		0	0	1.000		214	5.5	32	7.8	0.051	0.200	26	8.6	0.024	0.420
Prostatitis	221	6.0	20	6.8	0.568		15	5.8	0.924		4	0.1	1	0.3	0.376		0	0	1.000	
Sexually transmitted diseases	781	21.1	94	31.8	<0.001	<0.001	51	19.7	0.603		1619	41.3	209	51.0	<0.001	<0.001	132	43.6	0.445	
Mental diseases																				
Psychosis	45	1.2	5	1.7	0.414		5	2.0	0.254		55	1.4	9	2.2	0.207		1	0.3	0.184	
Depression	270	7.3	46	15.6	<0.001	<0.001	17	6.6	0.676		534	13.6	65	15.9	0.215		32	10.6	0.131	
Other mental disorder	129	3.5	9	3.1	0.708		13	5.0	0.195		223	5.7	32	7.8	0.085		17	5.6	0.948	
Psychoactive drug problem	157	4.2	24	8.1	0.002	0.012	16	6.2	0.139		48	1.2	15	3.7	<0.001	<0.001	6	2.0	0.279	
Other diseases and health conditions																				
Cancer	37	1.0	5	1.7	0.235		2	0.8	1.000		121	3.1	9	2.2	0.314		12	4.0	0.402	
Anaemia	256	6.9	23	7.8	0.560		21	8.1	0.459		1100	28.1	121	29.5	0.547		86	28.5	0.890	
Respiratory diseases	575	20.3	53	22.6	0.394		46	23.4	0.307		751	23.8	101	31.3	0.003	0.035	60	25.6	0.523	
Atopy	800	27.5	64	27.6	0.980		47	24.0	0.283		1333	40.9	146	43.8	0.299		102	41.0	0.985	
Bone fractures	1469	39.7	141	48.0	0.005	0.044	100	38.6	0.736		924	23.6	109	26.6	0.179		76	25.2	0.540	
Multimorbidity																				
No chronic diseases	2032	54.7	145	49.0			134	51.7			1924	49.1	180	43.9			135	44.6		
One chronic disease	1162	31.3	95	32.1	0.040	0.233	84	32.4	0.576		1299	33.1	141	34.4	0.069		100	33.0	0.104	
Two or more chronic diseases	518	14.0	56	18.9			41	15.8			698	17.8	89	21.7			68	22.4		

P-value presents the differences between groups. Comparison to two-parent family values was performed with χ2-test or Fisher Exact Test. Data are presented as lifetime prevalences at both time points, except for diabetes and thyroid diseases at the 31* and 46** years. Data collected from clinical examination at the age 46 *** years. aPrediabetes was defined in accordance with the Finnish Current Care Guidelines. B-Hp, Benjamini-Hochberg corrected p-value.

Field Code Changed