

# Glucose Tolerance Testing and Anthropometric Comparisons Among Rural Residents of Kyiv Region: Investigating the Possible Effect of Childhood Starvation—A Community-Based Study

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**ABSTRACT:** A relationship between childhood starvation and type 2 diabetes mellitus (T2D) in adulthood was previously indicated. Ukraine suffered a series of artificial *famines* between 1921 and 1947. Famines of 1932 to 1933 and 1946 were most severe among them. Long-term health consequences of these famines remain insufficiently investigated. Type 2 diabetes mellitus screening was conducted between June 2013 and December 2014. A total of 198 rural residents of Kyiv region more than 44 years of age, not registered as patients with T2D, were randomly selected. In all, 159 persons answered the question about starvation of parental family, including 73 born before 1947. Among them, 62 persons answered positive. Anthropometric measurements and glucose tolerance tests were performed. A logistic regression model was used to evaluate results. Type 2 diabetes mellitus was detected in 7 of 62 persons (11.3%), who starved during childhood vs 6 of 11 (54.5%) who did not ( $P = .002$ ), age-adjusted and sex-adjusted odds ratio (OR) (95% confidence interval): 0.063 (0.007–0.557). Analysis of the anthropometric data revealed a negative connection between adulthood height and neck circumference (cm, continued variables) and childhood starvation: age-adjusted and sex-adjusted ORs 0.86 (0.76–0.97) and 0.73 (0.54–0.97), respectively. Individuals who starved during famines of 1932 to 1933 and 1946 in Ukraine had a decreased T2D prevalence several decades after the famine episodes.

**KEYWORDS:** Childhood starvation, screening-detected diabetes mellitus, Ukraine, Holodomor

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## Introduction

The scale and consequences of a 20th-century humanitarian catastrophe, known in Ukraine as Holodomor (meaning “killing by artificial famine”), have not yet been sufficiently investigated.<sup>1,2</sup> The possibility to conduct such studies is diminishing every year due to constant decrease in the number of famine’s survivors; therefore, any chance to perform such investigations must be used.

Some literature data<sup>3–7</sup> and our previous studies<sup>8–11</sup> suggest a possible positive correlation between starvation during childhood and the risk of developing impaired glucose tolerance, type 2 diabetes mellitus (T2D), and obesity. We also know about reduction in final height in those who failed to receive enough food during early stages of development.<sup>12</sup> However, in Ukraine, the above assumptions were made based only on the analysis of administrative databases of patients with diabetes, rather than on classic “field” epidemiologic studies, based on questionnaire and anthropometric data, whereas current prevalence of screening-detected type 2 diabetes (SDDM) among those who survived the famine during childhood and are still alive today remains unknown. We cannot overlook the possibility that carriers of atherogenic and diabetogenic genotype could have greater chances of surviving during famine but

lesser longevity chances due to early development of atherosclerosis and/or T2D.

The risk of SDDM associated with starvation at an early age, revealed by glucose tolerance testing many years later, is the subject of our investigation.

We have used the possibility to include glucose tolerance data from tests that began in 2013 among residents of Ukrainian rural areas<sup>13,14</sup> into the analysis of possible influence of starvation in 1932 to 1933 and/or 1946.

## Materials and Methods

This study contains test results of 198 residents of Andriivka and Kopyliv villages (Kyiv region, 50°32′56.0″N 29°50′12.2″E and 50°24′35.8″N 29°53′25.0″E; current population 1046 and 1170 persons, respectively), randomly selected from the general population older than 44 years between 5 June 2013 and December 3, 2014 who permanently live in the above communities and were not registered as patients with T2D. Relevant lists of residents from 2 towns, provided by family doctors were used for randomization. Patients were selected using random number tables and received an invitation to take part in the study. If the patients did not give consent to take



part, the invitation was forwarded to the next person in the randomized list.<sup>13</sup> During the “Great Famine” in the neighborhood many people died, what today resembles a memorial to fellow villagers died of starvation (Supplement Figure 1). After signing the informed consent forms, the participants filled out a form, providing information about current treatment and lifestyle, as well as about the fact of starvation in their family in 1930s and/or 1946.

All participants were measured (body mass, height, waist circumference, neck circumference, arterial blood pressure [BP]). Body mass was measured using well-validated electronic scales, height—using standard portable stadiometer. Waist circumference and neck circumference were measured with a cloth-measuring tape at maximum transverse size in standing position. Body mass index (BMI) was determined as a relation of body mass in kilograms to squared height in meters. To measure arterial BP, we assessed the Korotkoff sounds using operational BP monitors from corresponding family medicine clinics. Blood pressure was measured twice, with an interval of 5 minutes. If there was a difference of more than 10 mm, we made a third measurement. The mean value of these 2/3 measurements was counted. High BP was determined as 140/90 mm Hg and above or by the fact of hypotensive drug treatment. Sufficient physical activity (30 min/d) and sufficient consumption of fruits and vegetables (500 g/d) were determined in accordance with current T2D prevention guidelines.<sup>15</sup>

The blood sampling was done on an empty stomach and 2 hours after taking a glucose solution (75 g of glucose in 200 mL of water). Blood plasma was quickly separated with a centrifuge (10 minutes; 1000g) and stored in a cold environment for further testing during 24 hours. Glucose and hemoglobin A<sub>1c</sub> (HbA<sub>1c</sub>) levels were determined by standard methods and in a certified lab: glucose oxidase method was used for glucose testing, and HbA<sub>1c</sub> levels were assessed using Clover A<sub>1c</sub> (Infortia Co., Ltd) system that uses boronate resin to bind HbA<sub>1c</sub>.

We also evaluated odds ratios (ORs) and corresponding 95% confidence intervals (CIs) to assess the risk of events in cross-sectional studies using the model of logistic regression. To evaluate the distribution of qualitative indicators, we calculated the manifestation frequency (%), whereas quantitative indicators, due to their nonparametric distribution in many cases, were given as medians and 1 to 3 quartiles. Frequency of events was compared using  $\chi^2$  test (Yates corrected). In all cases, differences of  $P < .05$  were considered probable. The 2-way Contingency Table Analysis (<http://statpages.org/ctab2x2.html>) and SPSS 11.0 software packages were used for statistical analysis. Shapiro-Wilk test was used to assess for the most adequate method of analysis for each case of comparing data rows. If one or both rows had a non-normal distribution of data, Wilcoxon test was used, and in case of a normal distribution, we used Student *t* test. In tables, the medians and 25th and 75th percentiles (quartiles 1 and 3) are given for comparison purposes.

## Results

Anthropometric and other measurements of rural residents (Kyiv region, Ukraine) belonging to different categories were created according to a response about starvation of parental family in 1932 to 1933 and/or 1946.

Of 198 studied individuals more than 44 years of age, 159 answered the question about starvation in the family either positively or negatively. Of them, 73 were born before 1947 and therefore may have personally suffered from the famine. From this number, 62 individuals (group 2) confirmed the fact of starvation in the family, and 11 persons (group 1) gave a negative answer. Those born after 1946 were added to groups 3 (answered negatively about starvation in parental family during 1932-1933 and/or 1946, 25 persons) and 4 (answered positively about starvation in parental family during 1932-1933 and/or 1946, 61 persons).

Quantitative data about lifestyle and anthropometric values for the 4 created groups are shown in Table 1. Women, who have not personally starved during childhood (group 1), had an age median greater by 4 years, compared with women from group 2 ( $P = .038$ ).

Among the studied persons of both sexes (men and women joined together), who replied negatively to the question about starvation in parental family and were born before 1947, there were a lot more individuals whose fasting glucose and/or glucose tolerance corresponded to diabetic level (fasting 7.0 and above and/or 11.1 after standard glucose loading). Comparing the number of individuals in these categories with a “diabetic” level of HbA<sub>1c</sub> demonstrates the same difference.

A separate analysis for women confirms the reduction in screening-detected diabetes mellitus, compared with those who replied negatively to the question about childhood starvation. In men, a similar difference is present regarding the fraction of diabetes, confirmed by HbA<sub>1c</sub> level in groups 1 and 2.

Men from group 1 had greater body weight, BMI, and waist circumference, compared with men from group 2. Analysis of other values for both sexes revealed only lesser physical activity of those from group 1, which is most likely related to them being older. There were no variations of values between individuals from groups 3 and 4.

Considering the age differences found in persons from groups 1 and 2, we conducted a regression analysis of the studied anthropometric indicators (as extended variables), taking into account age and sex.

The logistic regression analysis revealed standardized, according to age and sex, higher odds of belonging to group 2 (personal starvation in childhood) related to a decrease in height by 1 cm (OR = 0.857; 95% CI: 0.761–0.966;  $P = .011$ ).

A similar standardization by age and sex did not confirm any other anthropometric differences between the investigated groups, except neck circumference (Table 2). After adjusting for age and sex, the association between the reduction in this anthropometric indicator by 1 cm and childhood starvation becomes statistically significant: OR = 0.727 (0.543–0.974),  $P = .032$ .

**Table 1.** Anthropometric and other measurements of rural residents (Kyiv region, Ukraine) belonging to different categories, created according to a response about starvation of parental family in 1932 to 1933 and/or 1946.

MEASUREMENTS	GROUP 1	GROUP 2	P (1, 2)	GROUP 3	GROUP 4	P (3, 4)
Both sexes	11	62		25	61	
Fasting glucose, mmol/L	5.98 (5.52-7.44)	5.87 (5.368-6.56)	.805	6.18 (5.74-6.82)	6.41 (5.82-7.13)	.298
Glucose tolerance test, mmol/L	10.45 (5.96-12.49)	7.16 (5.52-9.24)	.173	6.07 (4.93-6.96)	6.36 (4.93-6.96)	.504
T2D according to glucose levels, No. (%)	8 (72.7)	14 (22.6)	.003	6 (24)	17 (28)	.920
T2D according to levels of glucose and HbA <sub>1c</sub> , No. (%)	6 (54.5)	7 (11.3)	.002	1 (4)	6 (9.8)	.642
High blood pressure, No. (%)	10 (91)	52 (84)	.881	14 (56)	43 (70)	.306
Sufficient physical activity, yes/no (%)	5/6 (45)	56/6 (90)	.007	21/4 (84)	57/3 (95)	.245
Sufficient consumption of fruits and vegetables, yes/no (%)	5/6 (45)	31/31 (50)	.96	17/6 (74)	31/30 (51)	.093
First-line relatives with diabetes, yes/no (%)	2/9 (18)	7/55 (11)	.889	7/17 (29)	16/44 (27)	.968
Infarction or stroke, yes/no (%)	4/6 (40)	7/55 (11)	.108	1/23 (4)	5/56 (8)	.85
Smoking, yes/no (%)	0/11 (0)	3/59 (5)	.936	3/22 (12)	5/56 (8)	.889
Alcohol consumption, yes/no (%)	6/2 (75)	31/28 (52)	.404	10/12 (45)	13/46 (22)	.084
Age, y	79 (76.0-81.0)	73 (71.0-77.3)	.015	59 (54.0-61.5)	59 (53.0-62.0)	.864
Men	4	13		7	13	
Fasting glucose, mmol/L	6.13 (5.56-7.59)	5.38 (4.95-5.85)	.209 <sup>a</sup>	6.12 (5.95-7.25)	6.11 (5.29-7.0)	.607 <sup>a</sup>
Glucose tolerance test, mmol/L	8.75 (6.03-13.19)	7.96 (5.54-10.94)	.592 <sup>a</sup>	5.18 (4.26-9.14)	5.61 (3.86-6.45)	1.0
T2D according to glucose levels, No. (%)	3 (75)	3 (23)	.193	2 (28.6)	3 (23.1)	1.00
T2D according to levels of glucose and HbA <sub>1c</sub> , No. (%)	3 (75)	1 (8)	.036	1 (14)	0 (0)	.747
Weight, kg	90.0 (72.8-108.0)	70.0 (57.4-82.5)	.034 <sup>a</sup>	81.0 (74.5-92.0)	92.0 (73.0-100.5)	.411 <sup>a</sup>
Height, cm	168.5 (162.8-180.3)	165.0 (162.8-180.3)	.206 <sup>a</sup>	168.0 (167-178)	173.7 (171.5-176)	.719
BMI, kg/m <sup>2</sup>	31.9 (25.7-35.3)	24.7 (22.7-29.5)	.044 <sup>a</sup>	27.5 (24.5-29.0)	31.0 (24.8-32.8)	.605 <sup>a</sup>
Waist circumference, cm	107 (98.75-112.25)	97 (94.5-100.5)	.016 <sup>a</sup>	103 (99-106)	101 (97-109.5)	.827 <sup>a</sup>
Neck circumference, cm	41 (37.75-43.50)	37 (36.0-41.0)	.154	41.5 (36.0-42.0)	40 (38.75-40.75)	.705 <sup>a</sup>
Age, y	80 (76.8-82.5)	78 (70.0-86.0)	.649	57 (53.0-59.0)	57 (51.0-62.0)	.842
Women	7	49		18	48	
Fasting glucose, mmol/L	5.98 (5.13-7.44)	6.09 (5.6-6.74)	.627	6.19 (5.71-6.78)	6.44 (5.84-7.16)	.156
Glucose tolerance test, mmol/L	10.45 (5.69-12.49)	7.16 (5.52-9.15)	.226	6.15 (5.48-7.0)	6.62 (5.42-7.66)	.469
T2D according to glucose levels, No. (%)	5 (71)	11 (22)	.025	4 (22)	14 (29)	.800
T2D according to levels of glucose and HbA <sub>1c</sub> , No. (%)	3 (43)	6 (12)	.13	0 (0)	6 (13)	.283
Weight, kg	63.0 (50.0-98.0)	72.0 (65.5-89.0)	.755 <sup>a</sup>	84.5 (66.8-104)	83.5 (76.0-93.0)	.989
Height, cm	162.0 (147.0-166.0)	155.0 (150.5-161.5)	.208 <sup>a</sup>	161 (157.5-162)	160 (156.1-164.8)	.692 <sup>a</sup>
BMI, kg/m <sup>2</sup>	29.2 (19.5-36.9)	30.7 (27.4-34.9)	.345 <sup>a</sup>	32.8 (27.1-40.2)	33.3 (29.2-36.6)	.734 <sup>a</sup>
Waist circumference, cm	106 (92-118)	108 (99-113.5)	.213 <sup>a</sup>	109 (102-125)	112 (107-119.75)	.681
Neck circumference, cm	36 (33.0-40.0)	36 (34.0-37.0)	.585 <sup>a</sup>	35.75 (33-38.25)	36 (34.63-38.0)	.739
Age, y	77 (75.0-81.0)	73 (71-75.5)	.038	60.5 (55.3-63.3)	59 (53.3-62.0)	.535

Abbreviations: BMI, body mass index; HbA<sub>1c</sub>, hemoglobin A<sub>1c</sub>; T2D, type 2 diabetes mellitus.

Data are given as number of persons (percentages) or medians (quartiles 1 and 3).

Group 1—born before 1947, answered negatively about starvation in parental family during 1932 to 1933 and/or 1946.

Group 2—born before 1947, answered positively about starvation in parental family during 1932 to 1933 and/or 1946.

Group 3—born after 1946, answered negatively about starvation in parental family during 1932 to 1933 and/or 1946.

Group 4—born after 1946, answered positively about starvation in parental family during 1932 to 1933 and/or 1946.

<sup>a</sup>Comparing using Student *t* test.

**Table 2.** Risks associated with childhood starvation, related to anthropometric measurements, as continued variables evaluated by a model of logistic regression.

MEASUREMENTS	CRUDE MODEL	P	SEX-ADJUSTED MODEL	P	AGE-ADJUSTED AND SEX-ADJUSTED MODEL	P
Height, cm	0.92 (0.849-0.997)	.041	0.919 (0.837-1.009)	.077	0.857 (0.761-0.966)	.011
Weight, kg	0.984 (0.95-1.02)	.382	0.983 (0.948-1.02)	.361	0.958 (0.917-1.002)	.059
BMI, kg/m <sup>2</sup>	1.013 (0.915-1.122)	.802	0.997 (0.894-1.112)	.957	0.957 (0.85-1.078)	.473
Neck circumference, cm	0.861 (0.712-1.041)	.123	0.879 (0.712-1.085)	.229	0.727 (0.543-0.974)	.032

Abbreviation: BMI, body mass index.

Given odds ratios and their 95% confidence intervals are associated with an increase in each measurement by 1 unit.

**Table 3.** Risk of developing diabetes<sup>a</sup> for those who starved during childhood.

	OR (95% CI)	P
Both sexes		
Crude risk	0.135 (0.027-0.679)	.015
Age-adjusted risk	0.059 (0.007-0.519)	.011
Sex-adjusted risk	0.131 (0.025-0.698)	.017
Age-adjusted and sex-adjusted risk	0.063 (0.007-0.557)	.013

<sup>a</sup>Plasma glucose levels 7.0/11.1 and above (fasting and/or after standard loading accordingly) and HbA<sub>1c</sub> 6.5% and above vs normal glucose tolerance.

Using the receiver operating characteristic curve to create categories of “reduced” and “nonreduced” height related to childhood starvation allowed to determine the following points of separation: for men—height greater than 167.5 cm (test sensitivity 75%, specificity 85%) and for women—159 cm (71% and 67% accordingly), which indicates a fair quality of the model.

We also analyzed the logistic regression model concerning the risk of having diabetes mellitus confirmed by HbA<sub>1c</sub> level, or normal glucose tolerance, associated with the fact of starvation during childhood (Table 3) and very low chances of having T2D, diagnosed by epidemiologic screening in group 2 (persons, whose families starved): OR without adjusting=0.135 (0.027-0.679) and when adjusting by age OR=0.059 (0.007-0.519). Simultaneous adjusting by age and sex does not alter statistical significance. Separate analysis for men and women also indicates only a statistical tendency toward reduction in T2D chances, influenced by starvation during childhood.

## Discussion

This study shows that individuals who starved during famines of 1932 to 1933 and/or 1946 in Ukraine had a decreased screening-detected diabetes mellitus prevalence several decades after the famine episodes.

Besides, we have revealed a reduction in age and height in a group of individuals, born before 1947, whose families suffered from the famine. Decrease in age in a group of persons who starved during childhood may be a reflection of a higher mortality risk, or a decrease in life expectancy, compared with their nonstarving neighbors. This explanation totally contradicts the

conclusion made concerning a connection of Ukrainian Holodomor to mortality in later life. However, this conclusion was made by Vaiserman et al<sup>16</sup> based on one of the types of mathematical data modeling of a general government mortality register that did not consider neither individual questionnaires nor the deceased person's region of residence. Thus, the fact that 6 of 24 Ukrainian regions did not suffer from the famine is not considered, which we believe had a negative effect of the significance of this study.

Investigators of the Dutch famine showed that starvation during any period of gestation leads to an increase in T2D development in later life.<sup>17</sup> Our small study, most likely indicates an opposite result; however, it did not analyze the influence of starvation during gestation period. Our previous opinion about the effect of starvation on the development of T2D was not based on the results of personal questionnaires but rather on the analysis of population patient registers.<sup>8-11</sup>

We cannot exclude the factor of selection when explaining long-term consequences of childhood starvation. Survival during famine and/or further long life may be explained by certain genetic features.<sup>18,19</sup> However, we must note that survival due to one set of genes does not necessarily mean a further long life. On the contrary, it is possible that those who have these genes have worse chances for further long life due to their metabolic deviation. Therefore, a cross-sectional study performed after 70 years shows a decrease in SDDM and mean age in a group of individuals who have confirmed starvation of their parental family. A possible explanation that unites our recent results with previous ones could be that a speedup of T2D development induced by childhood



starvation could lead to a situation, where the carriers of a corresponding genes were already diagnosed with diabetes or diabetes may have even led to a reduction in life span, and therefore, they were not included into our screening (exclusion criteria—known T2D).

A recent genotyping of those who survived the Leningrad siege<sup>18</sup> also led to unexpected results: in case of UCP3 gene, “normal” allele C and C/C genotype prevailed in women, whereas “mutated” allele T, associated with atherosclerosis, T2D, and obesity was significantly less common. It would be interesting to conduct similar genetic studies of the inhabitants of Ukraine, who suffered from starvation in early childhood. It is clear that conducting epidemiologic studies in Eastern Europe using individual famine exposure data can clarify causal relation between undernutrition during postnatal development and T2D risk in adulthood.

So we believe that our results do not necessarily contradict the hypothesis of the acceleration of the development of T2D in persons exposed to starvation in the early period of individual development.

Despite a small number of individuals included in our study, we were able to show a decrease in height, proving the known effect of childhood starvation on final height of men and women,<sup>12</sup> whereas we did not find any epigenetic influence, ie, a relation between starvation of parents and anthropometric features of investigated persons.

Being possibly the first simultaneous interpretation of data concerning the fact of childhood starvation and anthropometric and biochemical measurements at the level of a Ukrainian rural community can be considered one of this studies’ strengths.

An apparent weakness of this study is its quantitative limitation, related to current external circumstances, which may have been the reason why we were unable to find some other expected associations. In case of the growing scale of this study, it will allow to confirm or refute the link between starvation in childhood and development T2D in later life.

Besides, this study has a methodological loss which is the lack of perinatal data. In particular, birth weight is missing along with gestational age. The Barker hypothesis or programming has its roots in what occurred during pregnancy and the first 4 to 5 weeks of life. Impaired fetal nutrition and excessive nutrition after being born are the main benchmarks.<sup>20</sup> However, we were not able to use birth weight data together with gestational age. The humanitarian disaster in Ukraine, known as the Holodomor, made it impossible for normal functioning of obstetric care in the villages, including weighing infants or recording gestational age. However, it is possible that Soviet medicine until the 1950s quite often did not provide for these data at all. Researchers of long-term effects of starvation of the inhabitants of besieged Leningrad could not obtain reliable data on birth weight. It is interesting that Stanner et al<sup>21</sup> stated that there was no difference between the subjects exposed to starvation in utero and those starved during infant life in glucose tolerance.

## Author Contributions

MDK contributed to the conception and design of the analysis. NVO and MDK were responsible of data acquisition and measurements. VAK and VGG performed the statistical analysis. MDK and VIK contributed to the intellectual content. The final approval of the manuscript was done by all authors.

## Supplement

Figure S1. Monument in memory of the villagers died of starvation. Village Andriivka, Makariv District, Kyiv region, Ukraine.

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