Can social interactions change the brain?

Social network effects on obesity and related co-morbidities

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Abstract

The aim of the present study was to examine to what extend different social network mechanisms are factors explaining the spread of obesity and obesity associated co-morbidities. Based on our theoretical framework we derive testable hypotheses regarding an indirect and direct impact of social networks on EGO's BMI and insulin resistance. To test our hypotheses we undertook a clinical and social survey including a sample of 1397 probands. Collected data include anthropometric and biochemical measures as well as health attitudes, behavioural and socio-economic variables and social network data. We used nonparametric and parametric regression models to analyse whether EGO's BMI and insulin resistance are determined by EGO's social network characteristics controlling for EGO's individual characteristics. We found significant PSM and GPS treatment effects for high sport activities, a frequent diet behaviour (p=0.000) of EGO's social peer group. Since our regression analyses results that obesity is the main determinant of the HOMA-index this established a significant indirect network effect on insulin resistance. We also found significant direct social network effects on EGO's insulin resistance, i.e. controlling for EGO's obesity status frequent diet behaviour (p=0.033) and sport activities (p=0.041) of EGO's peer group decreases EGO's HOMA index. Network phenomena appear not only to be relevant for the spread of obesity, but also for the spread of associated co-morbidities.

Introduction

Obesity is becoming a major health problem in many countries throughout the world with the increasing prevalence reaching almost epidemic proportions [1]. Especially the obesity associated co-morbidities like type 2 diabetes and cardiovascular disease are progressively causing biomedical and also socio-economic problems.

In the past, epidemiological studies revealed a significant correlation of the risk for a child to develop obesity with parental BMI, suggesting a genetic impact in the development of this important metabolic disease [2]. Subsequently, several studies identified risk alleles for obesity, with the most of them being involved in central appetite regulation in distinct brain areas in the hypothalamus. These risk alleles for example included SNPs in Proopiomelanocortin [3], Neuropeptide-Y [4], Leptin [5], Agouti-related Peptide (AgRP) [6] and, of particular importance, in the Melanocortin-4-receptor (MC4R) [7]. The identification of a central role of specific neurons within the hypothalamus in the pathophysiology of obesity lead to further experimental studies in affected human subjects in order to investigate if also distinct areas in the cerebral cortex are somehow involved in the abnormal regulation of eating behaviour. These studies for example identified regions in the medial frontal and middle frontal gyrus, which are important in the reward activity of the brain to be dysregulated in obese human subjects [8]. In contrast to the basal brain functions organised in the hypothalamus, these higher brain areas might be influenced not only by biological signals from the own organism, but also for example by behaviour and knowledge form different human individuals. Together, these findings suggest that effects of social networks might even be more important than genetic factors in the pathophysiology of human obesity. In this regard in 2007 Christakis and Fowler nicely have shown for the first time, that social networks are important in the spread of obesity [9]. In this analysis, as part of the Framingham

Heart Study, it has been reported that the risk for becoming obese for a human subject is increased by 57% if he or she had a friend who became obese in a given interval. This finding was particular interesting, since in the same analysis, in pairs of siblings the risk to develop obesity was only 40% increased if the other sibling was becoming obese. Interpreting their results Christakis and Fowler suggests that obesity is “contagious”, not as much by any sort of direct “passing on” but by changing weight–related behaviour (diet, exercise, lifestyle, etc.) [10]. Furthermore, the role of social networks as a determinant of the prevalence of obesity has been supported by other studies (Chohen-Cole and Fletcher 2008, Bahr et al. 2009, de la Haye et al. 2010). However, the specific mechanisms by which networks influence behaviour are not fully understood, yet. Moreover, existing empirical studies try to identify network effects using observational data are plagued by serious identification problems.

In this context, the aim of the present study was to test empirically the effect of social networks on the development of obesity whereas we deal with the identification problem by using an adequate econometric setting. We develop a theoretical framework identifying different mechanisms how social networks exert influence on EGO‘s obesity-status and associated co-morbidities. In particular, we distinguish social network effects influencing EGO’s health-related behaviour, i.e. imitation, social norms, social learning via up-dating of EGO’s health beliefs, respectively. Further, we argue that beyond direct influence on EGO’s behaviour, social network effects might also operate via EGO’s social context. Further, we argue that EGO’s lifestyle/ behaviour (nutrition and physical activities) has not only an indirect effect via obesity on associated co-morbidities, but also a direct effect. Based on our theory we derive testable hypotheses on the indirect and direct effect of different social network characteristics on obesity and obesity associated metabolic and immunological abnormalities like insulin resistance and low-grad inflammation.

A total of 1397 probands (EGO) and 7726 social network partners (ALTER) were included in the study. Data on nutrition and activity behaviour as well as relevant socio-economic characteristics of probands were collected. Socio-economic data included age, sex, education, household size, and household income. Behavioural and lifestyle data that were collected including frequency of undertaking diets, attitude towards food, nutritional knowledge, and frequency of physical activities. The data were collected for EGO as well as for all of EGO's social network contacts (ALTER). Moreover, ego-centric network data were applied to the name generator concept, the state-of-the-art methodology to collect social network data. We calculated different network multiplier measuring the field strength of different health-relevant behaviours and attitudes. We apply Propensity Score Matching (PSM) and generalized matching techniques (GPS) to identify the treatment effect of the different lifestyle attributes of EGO's social peer group on EGO's BMI and EGO's HOMA-index. The latter measures insulin resistance and is an indicator of type 2 diabetes a frequent co-morbidity of obesity. Matching allows the construction of a control group including other EGOs that are statistical siblings of the treated EGOs, but differ exactly regarding the considered treatment variable. Since, EGO's own characteristics are explicitly included in the statistical construction of the control group matching controls for a potential selection bias due to dynamic peer group selection such as homophily, but also other potential selection biases assuming all relevant determinants of selection into treatment are taken into account (Calliendo and Kopeinig 2008). The treatment effects of social networks on EGO's BMI are generated for the five different network characteristics. The matching results imply that the average BMI of EGO's social peer group has no significant influence neither on EGO's own BMI nor on EGO's HOMA-index, while we found significant network effects for the weight related behaviour and

attitude of social peer groups. In particular, we found significant treatment effects for sport activities of EGO's peer groups on EGO's BMI and HOMA-index.

A theoretical framework for the impact of social networks on obesity

To the extent that obesity is a choice or behavioural problem, the fact that people are embedded in social networks implies that obesity at least partly depends on social influence. To understand how social network effects operate on obesity and associated co-morbidities we derive a schematic theoretical choice framework presented in figure 1. In general individual outcomes like for example obesity or associated co-morbidities result jointly from individual behaviour of EGO and the interaction with different ALTER in her social network. The impact of the interaction within EGO's social network on EGO's outcome may result from an indirect effect, e.g. the fact that EGO's behaviour is determined by the behaviour of ALTER. Or it might result from a direct effect, i.e. EGO's outcome is directly determined by ALTER's behaviour.

For example, an indirect effect would be that EGO's obesity is solely determined by his nutritional behaviour and his time allocation on physical activities and other leisure activities, e.g. watching TV, while EGO's behaviour is partly determined by the behaviour of his social peer group via social norms or via EGO's beliefs or attitudes regarding the impact of eating on obesity or associated co-morbidities. An example, of a direct effect of ALTER's behaviour on EGO's outcome corresponds for example to the fact that the smoking behaviour of ALTER has an impact on the health status of EGO beyond EGO's own smoking behaviour. Such direct effect might also be conceivable for obesity and associated co-morbidities, e.g. the study of [9] implies that reward behaviour of the brain might not only respond to own but also to the behaviour of other people.

Finally, other external environmental factors (U) might directly influence the outcome of EGO or indirectly via determining EGO's or ALTER's behaviour. Of course, to the extent that EGO and ALTER operate in the same social context or external environment their outcomes are correlated.

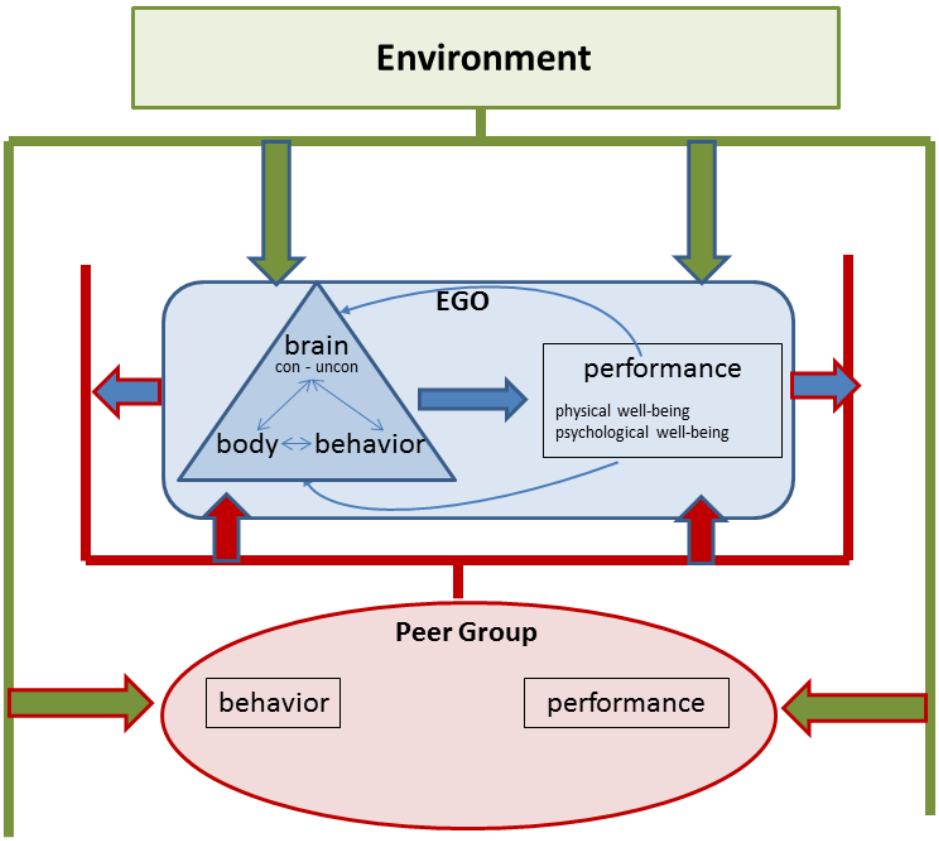

Figure 1: A theoretical framework for social network and environmental effects on obesity

Thus, to the extent that EGO and ALTER operate in the same social context or the same external environment their outcomes are correlated. Following the empirical literature on peer group effects a key challenge to identify what drives correlation between outcomes of individuals who interact together [Blume und Durlauf 2005, Manski 1993, Cohen and Fletcher 2008]. Basically the following problems arise:

1. The identification of a peer group effect can be complex if the behaviour of interacting agents mutually determinate each other. In this case the system of behaviour gives rise to a network multiplier effect [Bramoulle 2007].

2. Distinguishing between social context and external environmental factors based on observational data might be difficult. This follows from the fact that behaviour of a social group becomes similar or yields similar outcomes if a reference group operates under the same external environment or under the same social context.
3. Latent homophily: people interact with each other because they share similar attributes, e.g. similar preferences, attitudes or beliefs or similar outcomes. Hence, under homophily even without any social influence or common external factors, outcomes are correlated among members of the same peer group.

In this regard, Christakis and Fowler [9] tried to identify social influence via regressing EGO's outcome on the outcome of ALTER, where they used lagged outcomes of EGO and ALTER to control for latent homophily. However, lagged variables do not perfectly control for latent homophily [as has been demonstrated by [].

Second, lagged outcomes do not allow to distinguish the impact of a common social context from the impact of a common external environment [Cohen-Fletcher]. Therefore, Cohen and Fletcher used the lagged outcome specification of Christakis and Fowler [9], but in contrast to Christakis and Fowler [9] they additionally controlled for the external and social context using group specific fixed effects. Introducing group specific fixed effects they could nicely demonstrate that the impact of ALTER's obesity status on EGO's obesity status vanish. However, Cohen and Fletcher interpreted their results as empirical evidence that social influence on obesity can be excluded. However, this conclusion is based on their ad hoc interpretation that group specific fixed effects correspond to external environmental factors. These fixed effects, however, can also correspond to a specific social context, e.g. specific school norms regarding eating or sport oriented lifestyles. Obviously, the latter clearly corresponds to social influence.

In this context we suggest an alternative identification strategy. Following our theoretical framework as well as recent network models of social influence we assume that the environment (U) including basic socio-economic characteristics of EGO and ALTER (X^{EGO}, X^{ALTER}) as well as EGO's and ALTER's lifestyle attributes (Z^{EGO}, Z^{ALTER}) generate a socio-spatial field that operates on EGO and ALTER and determines their individual weight-related behaviour and hence obesity status. Assuming a simple linear approximation of the socio-spatial field implies

that the field strength operating on an EGO i (F^i) corresponds to a weighted sum of EGO's and ALTER's attributes (Z). The relative weight of EGO's attributes depends on the relative importance of her behaviour for her own outcome (λ_i), while the relative weight of different ALTERs in EGO i's peer group depend on the structures of the EGO's social network (T_i) [Henning et al. 2014].

$$\begin{aligned} Z_i^0 &= Z(x_i, u) \\ F_i &= \lambda_i Z_i^0 + (1-\lambda_i) H_i \\ H_i &= \sum_{j \in N^i} t_{ij} Z_j \\ O_i &= G(F_i, u) \end{aligned} \tag{1}$$

Hence, given the linearity of the model a stationary point of field-strength results as:

$$\begin{aligned} f &= M\, z^0 \\ M &= \left[I - (1-\lambda_{diag)} T_i\right]^{-1} \lambda_{diag} \end{aligned} \tag{2}$$

, where f corresponds to the vector of final field-strength operating on agents, while z^0 is the vector of initial attributes.

Accordingly, to identify the impact of ALTER's lifestyle attributes on EGO's obesity status in an experimental design one would keep EGO's attributes constant and vary the attributes of ALTER. The measured change of EGO's obesity resulting from these experiments identifies the peer group effect. Trying to identify this impact using observational data we apply propensity score matching (PSM) as a non-parametric statistical methods to generate a quasi-experimental design, that is to identify a control and a treatment group, where both groups are identical regarding EGO's attributes and differ only regarding the attributes of the peer group as the treatment. Generalized matching….

The PSM-method is described in more detail below.

Please note that since we explicitly control for EGO's attributes when we estimate the treatment effect of EGO's social network we explicitly control for latent homophily. Further, to control for external environmental factors we use a set of relevant socio-economic characteristics of EGO to match treatment and control groups, while we assume that any other environmental factors, e.g. the distance of EGO's home to fast food restaurants, are uncorrelated with EGO's BMI[1].

[1] Of course applying PSM matching we exclude selection on unobservables, i.e. we assume that we control for all relevant lifestyle attributes. We argue that in our data unobserved environmental factors are uncorrelated

Data collection and measurement

We conducted a clinical and social survey [Food Chain Plus Study, funded by the federal ministry of education and research (BMBF), Number: 0315540A]. The survey started in September 2011 and was finalized in 2015. The total survey sample includes a subsample of 500 obese people with a BMI > 30 and a randomized control group of 897 probands. The study was approved by the local ethics committee (Number: 156/03) and written informed consent was obtained for every subject before inclusion into the study.

For each proband we collected anthropometic [weight, hight, blood pressure, waist circumference, sensoric testing, testing for muscular strength], and biochemical data [fasting insulin serum levels, fasting glucose serum levels, C-reactive protein serum levels, triglyceride serum levels] as well as behavioural data. The biochemical analysis was performed by routine measurements within the department of laboratory medicine at the University Medical Centre in Kiel. The Homeostasis Model Assessment Index for Insulin Resistance (HOMA-IR) was calculated as follows: fasting insulin (µU/ml)*fasting glucose (mg/dl)/405. Probands visited the CAU clinic where relevant anthropometic and biochemical data has been collected.

Further, data on nutritional and activity behaviour as well as relevant socio-economic characteristics of probands have been collected. For further details please see table A1 in the appendix. Socio-economic data included age, sex, education, household size, and household income. Behavioural and lifestyle data that were collected for ALTER including frequency of undertaking diets (DIET), attitude towards food (ATT), nutritional knowledge (KNOW), frequency of physical activities (SPORT). The data was collected for EGO as well as for all of EGO's social network contacts (ALTER). The detailed measurement concepts of collected data is reported in table A1 in the appendix.

Moreover, in a special social network survey we collected EGO-centric network data from each proband. Moreover, EGO-centric network data has been collected when probands visit CAU clinic using a specific computer based social network questionnaire. The collected ego-centric network data is applied to the name generator concept, the state-of-the-art methodology to

with EGO's BMI and hence matching correctly controls for any selection bias. However, as a robustness check we alternatively undertake IV-estimation controlling for selection on unobservables.

collect social network data [11]. To implement this, the following three name generator questions were asked:

G1: With whom do you regularly discuss personal problems?
G2: To whom can you turn for help if you have a problem?
G3: With whom do you regularly discuss health-related (especially weight-related) problems?

For all ALTER mentioned by EGO in response, we also asked for their *gender, age, education*, and *profession*. Further, we asked EGO to estimate for each ALTER the following characteristics: (1) ALTER-*BMI* measured in five categories (1-5) ranging from very slim to very fat, for details see table A1; (2) *Nutrition knowledge (ALTER-KNOW)*: 1=very low, 2=low, 3=average, 4=good, 5=excellent; (3) *Nutritional attitude (ALTER-AT)*: 1= food is mainly convenience; diet has to balance health and convenience aspects, 3=diet has to be mainly healthy; (4) *Frequency of sport activities* (ALTER-SPORT) longer than 30 minutes: 1=never; 2 = 1-2 per month, 3 = 1 per week, 4 = several times per week; 5 = every day; (5) *Diet behaviour (ALTER-DIET)*, we ask how often ALTER has made a specific diet to lose weight: 1=never, 2=1 time, 3=2-3 times; 4=4-5 times, 5= >5 times. At the end of the questionnaire, we also asked questions about the *strength, length*, and *importance* of the relation with the named individuals. Following the concept of Krackhardt (for further explanations see [11]), we asked interviewees to describe the pairwise relations of the ten most important individuals mentioned on a 3 point scale with 0 = do not know each other, 1 = know each other, 2 = know each other very well.

Based on our theory we calculated different network multiplier (NET-Z) measuring the field strength of different health-relevant behaviours and attitudes (Z= KNOW, DIET, BMI, AT, SPORT) prevalent in EGO's social network and operating on EGO (see eq. (1) and (2) above):

$$NET - Z_i = \overline{fr_i} \sum_{j \in N^i} t_{ij} X_j$$

, where t_{ij} is the relative strength of a network tie between EGO j and ALTER i and $\overline{fr_i}$ is the average absolute strength of a network tie. We measure the relative strength of EGO-ALTER relations (t_{ij}) using the relation frequency of ALTER's network contact with EGO. Please note that our network multiplier also corresponds to the *network force* a measure suggested by [14] as well as the position generator a EGO-centric network measure suggested by [12] [13].

Statistical analyses

First, we apply Propensity Score Matching (PSM) to identify the average treatment effect on treated (ATT) [Rosenbaum und Rubin 1983] of the different lifestyle attributes of EGO's social peer group (NET-Z).

To this end we define for each attribute NET-Z a binary treatment variable $D_{Net\text{-}Z}$ as follows:

$$D_{NET-Z} = \begin{cases} 1 & \text{if } \text{NET} - \text{Z} > \text{Mean}(\text{NET - Z}) \\ 0 & \text{if } \text{NET} - \text{Z} \leq \text{Mean}(\text{NET - Z}) \end{cases}$$

Further, we estimated separate probit function using each $D_{NET\text{-}Z}$ as endogenous variable and relevant socio-economic characteristics (X^{EGO}) and lifestyle indicators of EGO (Z^{EGO}) as well as all relevant lifestyle multiplicators calculated for EGO's social network except the one corresponding to the endogenous variable $D_{NET\text{-}Z}$. In particular, socio-economic variables include EGO's age, sex, household size (HS), education (EDUC), while EGO's lifestyle indicators include EGO-attitude towards food (EGO-AT), EGO's nutrition knowledge (EGO-KNOW), EGO's diet behaviour (EGO-DIET). Based on each estimated probit function we calculated corresponding PSM-scores for all EGO's and used calculated PSM-scores to match the treatment group (I_1) with a corresponding control group (I_0) applying a Kernel Matching operator [quote].

Finally, we calculated for each treatment variable NET-Z the average treatment effect on treated as follows:

$$ATT = \frac{1}{N_1} \sum_{i \in I_1} \left[Y_i^1 - \sum_{j \in I_0} W_{N_0}(i, j) Y_j^0 \right]$$

Using the Kernel matching operator implies that all members of the control group are used to estimate the ATT, but with different weights, where the following weight for each observation in the control group are used:

$$W_{N_0}^{KM}(i,j) = \frac{G_{ij}}{\sum_{k \in I_0} G_{ik}}; \quad with: \quad G_{ik} = \frac{G(b_i - b_j)}{a_{N_0}}$$

G denotes the Kernel function [31] with a_{No} and b_j being specific parameters of the Kernel function. The kernel weights decreases with the distance of the propensity score of a member of the control group to the propensity score of the member of the treatment group. Thus, treated EGOs are compared with non-treated EGOs who have the same socio-economic characteristics as well as the same health related attitudes and behavior as treated EGO and who also have the same peer group characteristics despite from the treatment variable. PSM-matching is a statistical procedure that allows the construction of a control group including other EGOs that are statistical siblings of the treated EGOs, but differ exactly regarding the considered treatment variable. Since, EGO's own characteristics are explicitly included in the statistical construction of the control group PSM matching controls for a potential selection bias due to dynamic peer group selection such as homophily, but also other potential selection biases assuming all relevant determinants of selection into treatment are taken into account ('selection on observables' see [31]). However, the PSM method focus on the case where treatment is binary, while analysed peer group effects correspond to a continuous treatment. Hence, we follow Hirano and Imbens (2004) and apply the generalized propensity score method (GPS). GPS is an extension of the PSM method in a setting where the treatment is continuous.

Third to analyze to what extend social networks have an influence on insulin resistance and thus on EGO's probability to develop type 2 diabetes and cardiovascular diseases, we regress

EGO's HOMA-IR-index on EGO's obesity-status, socio-economic variables (X) and health related lifestyle attributes (EGO-Z) as well as on the corresponding social network multipliers (NET-Z) measuring the field strength of relevant lifestyle attributes of EGO's social network operating on EGO. EGO's obesity status is measured by a dummy variable, where OBS-EGO=1 indicates a BMI>30 and OBS-EGO=0 indicates a BMI<30. Regression models are estimated applying a two-stage IV-estimator to take potential endogeneity of EGO's BMI into account. We used EGO's lifestyle attributes (EGO-Z), the relevant network multipliers of EGO's social network (NET-Z) and EGO's socio-economic variables (X-EGO) as instruments for EGO's obesity status (OBS-EGO). Moreover, we use the second stage estimation to analyze the indirect impact of social networks on EGO's HOMA-IR-index via influencing EGO's obesity status (OBS-EGO), while the main regression at the first stage includes direct effects of social peer groups on EGO's insulin resistance, i.e. direct peer group effects correspond to effects operating via the direct influence of higher brain areas by the behaviour of other human individuals [8] that do not operate via a change in EGO's behaviour or obesity status.

However, since an average of 39% of EGO's network contacts are family ties estimations might be plagued by an endogeneity problem in the following sense. Insulin resistance is at least partly genetically determined [33]. Hence, assuming that health related behaviour and attitudes are also at least partly genetically determined might imply a spurious relationship between direct peer group effects induced by family ties and EGO's HOMA-index. Of course, we already control for this spurious relationship as well as for a potential spurious correlation due to homophily as a dynamic peer group selection since we explicitly include EGO's own health related behaviour and attitudes in our main regression equation. Nevertheless as an additional robustness check we undertake a three stage IV estimation where we instrumented EGO's family peer group behaviour and attitudes at a third stage using corresponding

behaviour and attitude of EGO's non-family ties as instruments. Moreover, we re-estimate our two-stage IV regression model excluding family ties completely. Further, we use results of the second stage of the IV-estimation as a robustness check of our PSM and GPS-estimations.

Results

Characteristics of the study cohort:

N= 677 subjects designated as EGOs were included. Basic descriptive statistics of our sample are reported in table 1. Any person to whom EGOs are linked serves as a social contact, and is designated "ALTER" in the following. A total of n=3033 ALTERS, observed family and social ties, were connected. This yields an average of 5.5 ties per EGO within the network. A total of 39.1% of the 3033 ALTERs were family contacts. The remainders 60.9% were connected through friendship to EGO. The average duration of relationship was 25.5 years with a standard deviation of 11.3. The minimum duration was 0.666 years and the maximum 61.5 years. The mean age of investigated EGOs was 51 years with a range from 19 to 84 years. The mean age of ALTERs was 48 years, ranging from 11 to 91. 34% of the EGOs were men, while 41% of ALTERS were male. The educational level was measured on a scale ranging from 1 to 10 (1 to 8 in case of ALTERs) with 1 indicating no formal education and a 10 (8) denoting a PhD-level. The average educational level of EGO's was 5.3 on a 10 point scale, while the mean education level of ALTERs was 4.1 on a 8 point scale. The frequency of EGO network contacts ranged from 28% who meet daily, over 39% who meet weekly to 29% who meet only on a monthly basis. Only 4% of ALTERs did meet less than one time per months by EGO.

Treatment effects of social network characteristics on obesity

PSM-method

In table 2 the treatment effects (ATT) of social networks on EGO's BMI are reported, generated from PSM-Matching analysis for the five different network characteristics. As can be seen from table 2 the PSM-matching results imply that the average BMI of EGO's social peer group has no significant influence on EGO's own BMI, while we found significant network effects for the weight related behaviour and attitude of social peer groups. In particular, we found significant treatment effects for diet behaviour and nutritional attitude, while nutritional knowledge and sport activities of peer groups had no significant impact on EGO's BMI. In quantitative terms the impact was the highest for diet behaviour resulting in an average treatment effect of 4.3, i.e. having a social network that frequently engaged in diets implies an increase in BMI by 4.3 kg/m^2. Given an average BMI of 31 in our sample this corresponds to a remarkable effect of more than 13%, which is highly significant with a t-value of 4.3 ($p < 0.001$). Analogously, we found a remarkably high effect on EGO's BMI of 3.5 kg/m^2 for health oriented attitude of EGO's social network corresponding to a reduction of over 10% of the average BMI. Furthermore, a high frequency of sport activities in EGO's social network reduces EGO's BMI by 0.75 kg/m^2, but this effect was statistically not significant.

GPS-method

As discussed above PSM method focus on the case where treatment us binary. However, analysed peer group effects in fact correspond to a continuous treatment. Hence, we follow Hirano and Imbens (2004) and apply the generalized propensity score method (GPS). GPS is an extension of the PSM method in a setting where the treatment is continuous.

In particular, applying the GPS-method we estimated the dose response and the treatment effect function, respectively testing for significant treatment effects on EGO's BMI as well as on EGO's HOMA-Index taking the five network characteristics as continuous treatment variables. Overall, our GPS-estimation results are summarized in table 2a. As can be seen from table 2 applying GPS implies significant linear treatment effects on EGO's BMI for the average BMI (Net-BMI), the level of sport activities (Net-sport) and the diet behaviour (NET-diet) of EGO's peer group, while applying GPS no significant treatment effect for the health oriented attitude towards eating and the nutrition knowledge of EGO's peer group results. In particular, the GPS-analysis results in a significant and positive impact of the obesity status and the diet behaviour of peer groups on EGO's BMI, respectively. Thus, while for diet behaviour GPS confirms the results of PSM, regarding the peer group BMI the GPS-analysis confirms in contrast to the PSM result the study of CF, that is even controlling for EGO's own health related behaviour and attitudes we find evidence for the influence of the BMI of social networks on EGO's obesity status, where obese peer groups increase EGO's obesity. Analogously, applying GPS we find a significant and negative treatment effect for the sport activities of EGO's peer groups on her BMI, i.e. more active peer groups imply a lower BMI of EGO. To save space we report the detailed GPS-results only for sport activities (Net-sport) as well as for the BMI of peer-groups (Net-BMI) in table 2a and 2b 8see also figure 2 and 3), while we refer to Henning and Zarnekow (2015) for the complete GPS estimation results.

Multiple regression analysis on social network effect on insulin resistance

Results of the second stage of the IV-estimations are reported in table 3 (Model A), while results of the probit estimation of the first stage of our IV-estimation are reported in table 4 (Model A). As can be seen from table 3 the main determinant of EGO's insulin resistance

corresponds to EGO's obesity status with a normalized partial impact of *0.258*. In absolute terms an increase of 1% in the probability of becoming obese implies an increase of *0.07* units of the HOMA-IR-index. However, beyond obesity status also sex has a significant effect on insulin resistance with a normalized coefficient of 0.101 (see table 3, model A). In particular, males generally have a higher HOMA-IR-index when compared to females, with an absolute difference of 1.44 between men and women.

The most remarkable finding of our analyses, however, was a significant and robust direct influence of social networks on EGO's insulin resistance, even correcting for EGO's BMI. In particular, a higher frequency of diet behaviour (p=0.052) as well as sport activities (p=0.038) in EGO's social network reduces significantly her/his HOMA-IR-index given normalized regression coefficient of -0.101 and -0.078, respectively. In quantitative terms a maximal difference in the diet behaviour of EGO's network changing from no diet to an average of more than 5 diets undertaken per network contact implies an absolute decrease of *1.16* units of the HOMA-IR-index. Taking the normal HOMA-IR-value of <2.0 as a reference this corresponds to a remarkable change of 58%, and even if we compare this with the average HOMA-IR-index in our sample of 4.8 it still corresponds to a remarkable change of 24%. Analogously, a maximal change of the frequency of sport activities in EGO's network from no activities to an average sport activity of at least once per day decreases EGO's HOMA-IR-index *by 1.06* units, which still corresponds to remarkable 53% and 22% compared to the critical HOMA-IR-value of <2.0 and the average HOMA-IR-value of *4.8* in our sample, respectively.

Interestingly, in contrast to the lifestyle of EGO's social peer group EGO's own lifestyle indicators have no significant direct impact on EGO's HOMA-IR-index. However, EGO's lifestyle significantly influences EGO's BMI-status (see table 4, model A), where especially EGO's health oriented nutritional attitude (EGO-AT) reduces significantly EGO's probability to

become obese (p=0.000) with a marginal effect of -0.096 (see table 4, model A). Moreover, a high education (p=0.000) and income level (p=0.041) reduce EGO's probability to become obese. Peer group effects on EGO's BMI, however, are less pronounced in the multiple regression analysis. Only for diet behaviour of EGO's peer network a highly significant positive effect on EGO's BMI-status was observed (p=0.000, see table 4). For all other network multipliers only an insignificant effect resulted from our multiple regression analysis.

Combining the strong and significant impact of the EGO's BMI on her/his HOMA-IR-index estimated at the second stage with the estimation results of the logit regression at the first stage implies a strong indirect social network effect on insulin resistance. We calculated the indirect network effect as the marginal effect of a network multiplier on EGO's probability of becoming obese multiplied by the marginal effect of EGO's obesity status on her HOMA-IR-index. Significant indirect and direct peer group effects were identified for diet behaviour, while sport behaviour and BMI-status of EGO's peer network impact only directly on EGO's insulin resistance (Model A, table 3, 4). In contrast, EGO's own health related lifestyle and nutritional knowledge impact only indirectly on his/her insulin resistance (Model A, table 3, 4). Please note that we essentially derived the same results, i.e. we observe significant direct and indirect peer group effects on EGO's HOMA-IR-index, undertaking a three-stage IV estimation instrumenting behaviour and attitude of EGO's family ties (Model B, table 3, 4). Furthermore, results of our preferred model A do not change if we include insignificant lifestyle variables of EGO and his/her peer network (see model A-0 in table 3). Moreover, these results remain also robust if we re-estimate the two-stage IV regression model excluding family ties (estimation results are not presented here, but are available from the authors upon request). Therefore, we are confident that our main estimation results correspond to robust findings.

Discussion and conclusion

In recent years biomedical research has been enormously extended in regard to the pathophysiology of obesity and its associated co-morbidities. In terms of body weight regulation, it was established early that genetic factors explain 30-50% of the obesity epidemic and that environmental factors are tremendously important [34]. While initial studies focused mainly on nutrition, eating behaviour and physical activity as important environmental factors, in 2007 Christakis and Fowler demonstrated that social network effects might even be more important than genetic polymorphisms in the development of obesity [11]. However, in contrast to what was found for obesity, until now no data exist on the impact of social network effects on insulin resistance, a key obesity-associated morbidity that is the mediator of obesity-associated type 2 diabetes, lipid disorders and atherosclerosis [35]. Therefore the aim of the present study was to (1) investigate the impact of social network effects on obesity development in an independent European cohort and (2) to examine potential direct and indirect social network effects on the development of insulin resistance.

The results reported here imply that specific lifestyles attributes of social peer groups, especially frequent diet and nutritional attitude in favour of healthy food, influence significantly EGO's BMI. These findings confirm the previously identified network effects on body weight gain in the US population [11]. Together, these findings provide evidence for the hypothesis that appetite regulation organised in the hypothalamus might be influenced not only by biological signals from the periphery (e. g. ghrelin and leptin [36]), but also via function of the cerebral cortex via knowledge and/or behaviour form different human individuals of the patients relatives and peer group [8, 11].

In addition to what was examined in earlier reports, in the present social network study we also calculated the HOMA-IR index for the first time for each of the 677 individual subjects in

order to obtain a measure for their insulin action. Applying our mathematical model, we identified for the first time that social networks can influence EGO's insulin resistance. This is in part explained by indirect effects of the peer group on EGO's BMI and the BMI determining EGO's insulin resistance. This finding is not unexpected since many clinical and experimental studies have shown insulin resistance of liver and skeletal muscle to be associated with obesity [37]. Ectopic lipid accumulation in liver and skeletal muscle in response to an excess of energy intake is postulated to explain this association, leading in turn to serine phosphorylation of insulin receptor substrate (IRS)-1 and thereby inhibition of intracellular insulin receptor signalling [35, 38]. Therefore, as shown in this report, if the peer group influences EGO's BMI then one would expect that EGO's insulin resistance should also be affected. Hence, from a mathematical point of view, the fact that peer group effects on EGO's BMI are in line with peer group effects on EGO's insulin resistance indicates, that the associations found in our cohort are true sociobiological effects rather than statistical artefacts.

The most remarkable finding of our study corresponds to the fact that we also have been able to identify direct peer group effects on EGO's insulin resistance. That means that our regression analyses yield these significant network effects even when we control for EGO's BMI and for EGO's own weight-related attitudes and behaviour, respectively. This is particular interesting, since it has been shown that for example physical activity is able to improve insulin sensitivity in overweight subjects independently of significant changes in BMI.[38] Therefore, the fact that the degree of physical activity of the peer group beneficially affects directly EGO's insulin resistance suggests the existence of a potent sociobiological mechanism in the pathogenesis of insulin resistance.

At a methodological level PSM-matching as well as two-stage multiple regression analysis are adequate methods avoiding the problem of latent homophily even if only cross-sectional data

can be used (no panel data). However these statistical analyses are based on certain assumptions that we could not test explicitly. In particular, PSM matching is based on the assumption of "selection on observables" [31], i.e. we have to assume that our analysis includes all relevant selection variables. Thus, to the extent that "selection on unobservables" occurs PSM would deliver biased results [31]. In contrast, our two-stage estimation is not plagued by the problem of latent homophily as it appears rather unrealistic to assume that peer group selection occurs on the basis of insulin resistance. However, since an average of 39% of EGO's network contacts are family ties estimations might be plagued by an endogeneity problem resulting from spurious relationship between direct peer group effects and EGO's HOMA-index induced by genetic relations among family ties and EGO. Given our estimation design this spurious relationship could only occur if weight related behaviour and attitudes are determined by the same genes as the HOMA-index which we consider as rather unrealistic. Nevertheless we undertook an additional robustness check, i.e. we conducted a three stage IV estimation where we instrumented EGO's family peer group behaviour and attitudes at a third stage using corresponding behaviour and attitude of EGO's non-family ties as instruments. Moreover, we re-estimated our two-stage IV regression excluding family ties. Both alternative estimation strategies delivered in essence the same results. Hence, beyond theoretical considerations also on pure statistical grounds we are confident that we can exclude spurious relationships and that our main results correspond to robust findings.

In summary our study indicates for the first time that social network phenomena appear not only to be relevant for the spread of obesity, but also for the spread of insulin resistance. Direct and indirect social network mechanisms have been identified as significant factors determining the risk for impaired insulin signalling. Weight-related attitudes and behaviour of peer groups exert particularly significant impact not only on EGO's obesity status, but also

directly on EGO's insulin resistance. These results might have important clinical implications for the design of future obesity therapy programs. Given the fact that many individual-level intervention strategies to prevent obesity, including nutritional education, behavioural therapy and physical activity [40, 41], achieve very few sustained effects [41] our results might be used to design novel weight loss programs. These programs should include not only patients (EGOs) treatment but also education of the patients peer group to achieve more sustained results of multimodal obesity therapy programs in the future. Moreover, beyond designing innovative obesity therapy programs including peer group effects based on external peer group compositions, understanding the dynamics of peer group formation might also enable the design of peer group structures that amplify identified positive peer group effects on EGO's obesity and related co-morbidities.

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Glossary

Term	Definition
EGO	The actor whose network and behavior choices are being modeled.
ALTER	A person connected to the ego who may influence the behavior of the ego. An actor who is named as a friend by the ego.
Actor	A respondent.
Homophily	The tendency for people to choose relationships with people who have similar attributes.
Peer influence	The effect of alters' behavior on ego's behavior.
Social Influences	Synonym for peer influence.
Tie	A connection between two individuals (nodes) that can be either one-way (directed) or two-way (bilateral)
Node	An object that may or may not be connected to other objects in a network.
EGO-centric network	Subset of social relations among all persons (ALTERS) to whom a specific individual person (EGO) has a social tie. The EGO-centric network is also called the neighborhood or peer group of EGO.
Network multiplier	The value of similar behaviors or attitudes, averaged across all of the EGO's ALTERs; network multiplier is used as a measure of peer influence.
PSM	Propensity Score Matching is a non-parametric econometric estimation method of treatment effects controlling for potential selection bias.
SABM	Stochastic Actor-Based Model.

Table 1: Summarized descriptive statistics of the FOCUS-sample

	EGO **(Std.deviation)**	**ALTERS**
	mean values	*mean values*
Age	51.05 (14.46)	48.89 (12.85)
Sex (1: male, 0: women)	1: 232, 0: 452	1: 1241 , 0:1759
Household-Size (HS)	2.35 (1.62)	
Income[2]	8.92 (4.01)	
Education[3] (EDUC)	5.32 (2.32)	[4]3.92 (1.84)
BMI	32.88 (10.98)	[5]2.80 (0.71)
HOMA-IR	4.93 (7.15)	
N	684	3033
Behavior		
Knowledge[6] (KNOW)	4.93 (1.85)	[7]3.64 (0.74)
Attitude[8] (AT)	4.14 (1.58)	[9]1.75 (0.54)
Diet[10]	4.93 (1.85)	[11]2.30 (1.08)
Sport		[12]2.55 (1.01)

[2] Income level: 1: <499 Euro to 16: >4000 Euro
[3] Education: 1 = means no educational achievement; 10 = PhD
[4] Education: 1 = means no educational achievement; 8 = PhD
[5] Nutritional status: 1 = very slim; 5 = very fat
[6] Reading of food information is important: 1 (not agree)-7 (agree completely)
[7] Knowledge: 1= no; 5 = excellent
[8] Low fat food is important: 1 (not agree)-7 (agree completely)
[9] Attitude: 1= food has to be tasty, 2 = food has to balance enjoyment of eating and health, 3= food has to be mainly healthy
[10] I always eat healthy and well balanced: 1 (not agree)-7 (agree completely)
[11] Diet: 1 = never; 5 = more than 5 times
[12] Sport: 1 = never; 5 = daily

Network		
Size	5.54 (2.93)	
Duration (years)	25.56 (11.33)	
Type (0: family, 1: friends)	0: 1186; 1: 1847	
Frequency[13]	3.11 (0.61)	
Intensity[14]	2.55 (0.41)	
Multiplier		
Knowldge (KNOW)	6.83 (2.39)	
Attitude (AT)	3.34 (1.39)	
Diet	4.04 (2.53)	
Sport	4.66 (2.33)	
BMI	5.07 (2.09)	

[13] Contact frequency: 1 = never to 5 = daily

[14] Intensity: 1= no talk about private issues; 3 = often

Table 2: Estimated PSM treatment effects (ATT) of social network characteristics on obesity (BMI)

Treatment Variable	Selection	Treated Group	Control Group	ATT	t-values*	p
$D_{NET\text{-}DIET}$	unmatched	35.900	31.050	4.849	5.7	0.000
	matched	35.900	31.613	4.287	4.3	0.000
$D_{NET\text{-}BMI}$	unmatched	33.357	32.543	0.814	0.96	0.567
	matched	33.357	33.203	0.154	0.14	0.779
$D_{NET\text{-}Sport}$	unmatched	31.830	33.652	-1.822	-2.13	0.005
	matched	31.830	32.582	-0.752	-0.71	0.200
$D_{NET\text{-}Know}$	unmatched	32.939	32.871	0.068	0.08	0.900
	matched	32.939	34.342	-1.403	-0.8	0.200
$D_{NET\text{-}AT}$	unmatched	32.210	33.520	-1.310	-1.54	0.050
	matched	32.210	35.753	-3.543	-2.95	0.000

- **t-values derived via bootstrapping**

Table 2a: GPS-results for NET-sport on EGO's BMI

The regression model is: Y = T + GPS + T*GPS

Source	SS	df	MS
Model	25910.579	3.000	8636.860
Residual	113396.889	1393.000	81.405
Total	139307.468	1396.000	99.790

Number of obs =	1397.000
F(3, 1393) =	106.100
Prob > F =	0.000
R-squared =	0.186
Adj R-squared =	0.184
Root MSE =	9.023

BMI	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
fraction	-44.431	14.628	-3.040	0.002	-73.126	-15.737
gps_flog	-106.283	15.759	-6.740	0.000	-137.197	-75.370
fraction_gps_flog	72.033	28.487	2.530	0.012	16.150	127.916
_cons	89.044	7.904	11.270	0.000	73.539	104.548

Figure 2: Estimated dose response and treatment effect function for NET-sport on EGO's BMI

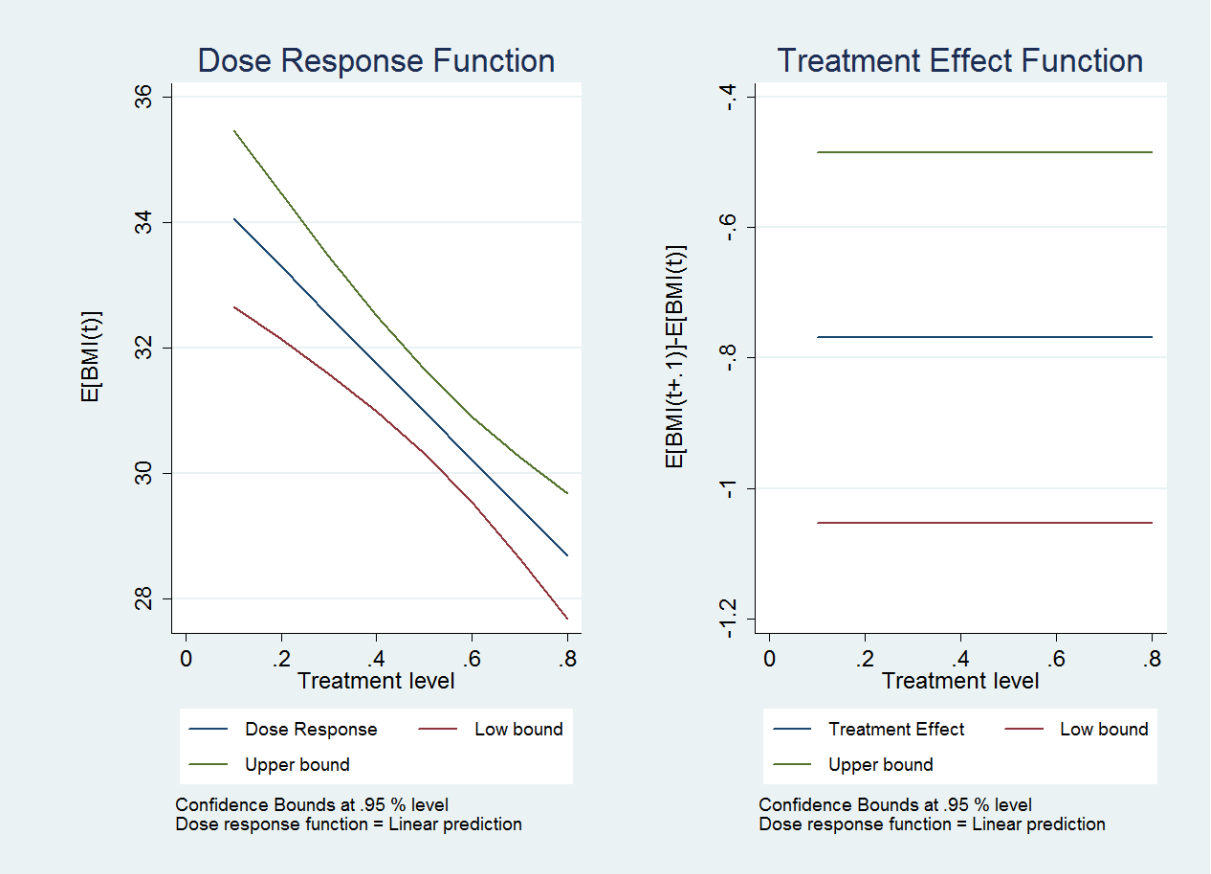

Table 2b: GPS-results for NET-BMI on EGO's BMI

The regression model is: Y = T + GPS + T*GPS

Source	SS	df	MS
Model	23662.914	3.000	7887.638
Residual	115644.554	1393.000	83.018
Total	139307.468	1396.000	99.790

Number of obs =	1397.000
F(3, 1393) =	95.010
Prob > F =	0.000
R-squared =	0.170
Adj R-squared =	0.168
Root MSE =	9.111

BMI	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
fractionZ	-254.433	67.189	-3.790	0.000	-386.235	-122.631
gps_flogZ	-65.310	67.046	-0.970	0.330	-196.831	66.211
fractionZ_gps_flogZ	430.261	109.506	3.930	0.000	215.447	645.075
_cons	65.614	41.052	1.600	0.110	-14.916	146.144

Figure 3: Estimated dose response and treatment effect function for NET-BMI on EGO's BMI

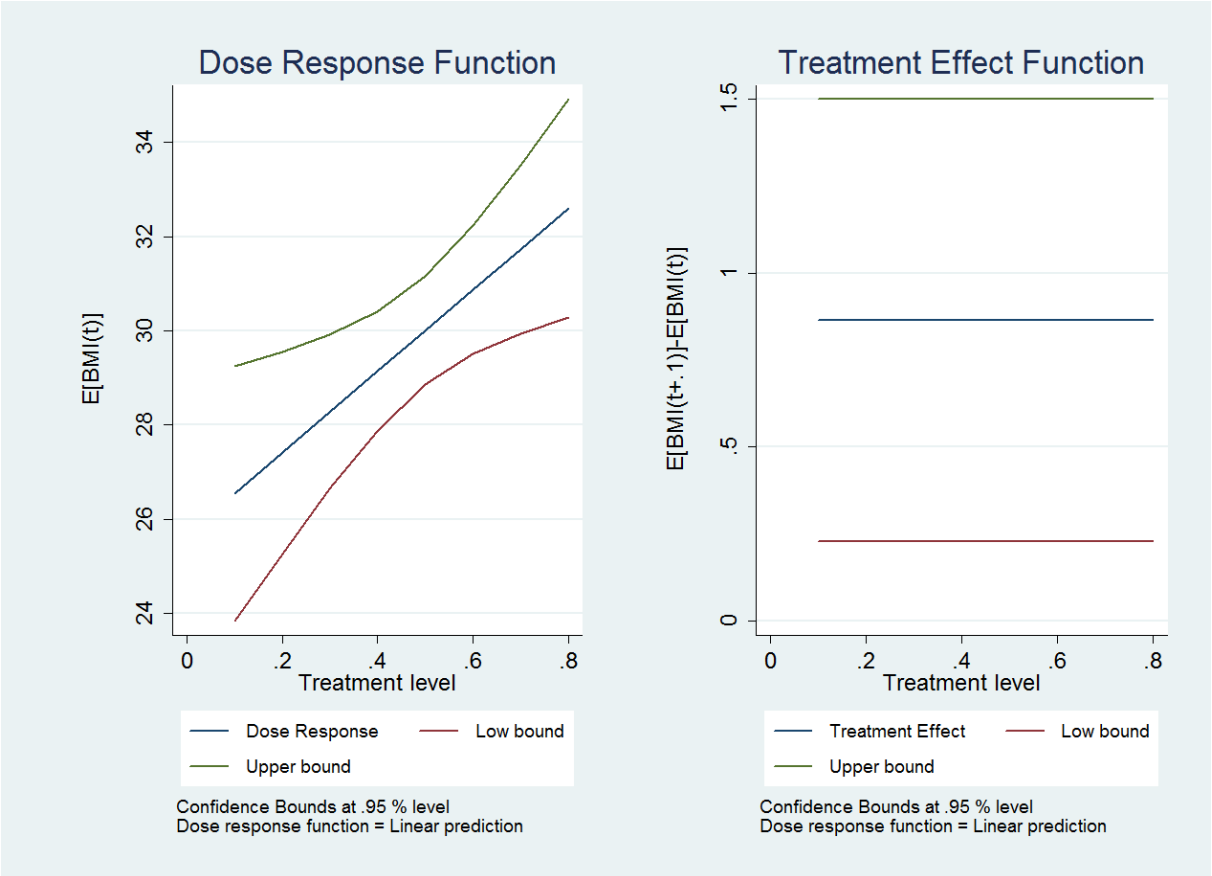

Table 3: Results of the IV-estimation: Dependent variable EGO's HOMA-IR

	Model A-0			**Model A**			**Model B**		
	Coef.	P>|	Beta	Coef.	P>|	Beta	Coef.	P>|	Beta
Male	1.388	0.0	0.098	1.439	0.0	0.101	1.459	0.0	0.103
EGO-DIET	0.140	0.4	0.038	0.140	0.4	0.038	0.184	0.0	0.051
EGO-AT	0.165	0.4	0.039	0.154	0.3	0.036	0.109	0.1	0.026
EGO-KNOW	-0.067	0.6	-0.020						
NET-AT	-0.051	0.8	-0.011						
NET-DIET	-0.291	0.0	-0.110	-0.267	0.0	-0.101	-0.082	0.6	-0.031
NET-BMI	0.284	0.0	0.088	0.332	0.0	0.103	0.245	0.0	0.076
NET-KNOW	0.126	0.4	0.045						
NET-SPORT	-0.264	0.0	-0.092	-0.225	0.0	-0.078	-0.202	0.0	-0.070
EGO-BMI	7.375	0.0	0.263	7.231	0.0	0.258	6.358	0.0	0.227
Prob > F =	0			0			0		
R-squared	0.386			0.385			0.374		
Adj R-squared	0.377			0.379			0.367		

- **t-values derived via bootstrapping**

Table 4: Results of Logit-Model: Dependent variable EGO's BMI-status (OBS-EGO) (IV-first stage)

	Model A			**Model B**		
OBS-EGO	coef	P>|z|	Marginal effect	coef	P>|z|	Marginal effect
NET-KNOW	0.031	0.613	0.008	-0.044	0.725	-0.011
NET-AT	-0.134	0.145	-0.033	-0.186	0.340	-0.046
NET-BMI	-0.034	0.536	-0.009	-0.232	0.048	-0.057
NET-DIET	0.238	0.000	0.059	0.504	0.000	0.124
NET-SPORT	-0.001	0.988	0.000	0.026	0.783	0.006
Age	-0.011	0.110	-0.003	-0.014	0.039	-0.004
Male	-0.053	0.789	-0.013	0.009	0.965	0.002
Income	-0.053	0.041	-0.013	-0.066	0.012	-0.016
Education	-0.268	0.000	-0.067	-0.265	0.000	-0.065
HS	0.093	0.252	0.023	0.069	0.333	0.017
EGO-DIET	0.294	0.000	0.073	0.280	0.000	0.069
EGO-AT	-0.384	0.000	-0.096	-0.378	0.000	-0.093
EGO-KNOW	0.144	0.002	0.036	0.149	0.002	0.037
Constant	1.170	0.030		1.883	0.014	

- **t-values derived via bootstrapping**