

The Relationship Between Childhood BMI and Adult Serum Cholesterol, LDL, and Ankle Brachial Index

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Abstract

Objective: Effects of childhood overweight may persist into adulthood. We assessed the effect of childhood overweight on cardiovascular disease high risk factor levels in the same participants as adults, after controlling for adult BMI status.

Design: A subset of participants in an observational study (Heartwatch) were contacted approximately 26-27 years after initial enrollment to participate in a follow-up study on the long-term effects of childhood overweight. During follow-up, BMI, waist:hip circumference (WHC), blood pressure (BP), serum lipids, and ankle brachial index (ABI) were measured; additional BMI measures throughout childhood were obtained as available from the electronic medical record. Primary outcomes were ABI and serum LDL.

Setting: The 1982 Heartwatch study was conducted with children participants living in Marshfield, WI; follow-up included original participants who were re-contacted and agreed to be enrolled.

Participants: Participants were a stratified random sample of eligible participants in the original 1982 Heartwatch study. Of the original 3106 participants, 647 adult participants completed follow-up exams.

Results: Among males with 1982 BMI \geq 85th percentile, adult BMI, WHC, (both $p \leq 0.001$), ABI ($p = 0.001$), total cholesterol ($p = 0.01$), LDL ($p=0.003$) and BP ($p < 0.02$) were higher in 2008-09 as compared to males with 1982 BMI $<$ 85th percentile. Among females, BMI, BP and WHC (all $p < 0.001$) were higher in 2008-09. BMI in 1982 and 2008-09 were correlated [$r = 0.56$ (males); 0.58 (females), $p < 0.001$]. 2008-09 BMI was more strongly correlated with 2008-09 measures of ABI ($r = 0.16$, $p=0.006$, males) and high LDL [$r = 0.18$, $p=0.002$ (males); $r = 0.11$, $p=0.046$ (females)]. 1982 BMI was not independently associated with ABI or LDL after adjusting for adult BMI.

Conclusion: In a cohort studying childhood and adult overweight, childhood BMI was associated with health outcomes relating to cardiovascular disease in adulthood. However,

childhood BMI was not independently related to LDL-C or ABI levels in adulthood after accounting for adult BMI. Longitudinal measurements of BMI and other health risk factors were not found to improve accuracy of models for high CVD risk factor levels.

Keywords: Childhood overweight, BMI, Cardiovascular disease

Introduction

The prevalence of overweight and obesity among children, adolescents, and adults in the U.S. has increased dramatically over the past several decades (1) but may be reaching a plateau (2). Child and adolescent overweight tracks to adult obesity (3-5), a condition associated with an increased incidence of disease, including hypertension, cardiovascular disease (CVD), type 2 diabetes mellitus, gallstones, sleep apnea, and some cancers (6). However, it is not clear whether these conditions are specifically related to overweight during childhood, adolescence, or adulthood; or whether a cumulative risk burden exists where the longer an individual is overweight, the greater their risk of disease.

Many studies that have examined the impact of obesity occurring at different times during the lifespan have yielded conflicting results (4, 5, 7), and there is some discussion regarding whether certain periods of time contribute more risk later in life than other periods of time, or whether risk is contributed in a more linear fashion (8). Factors such as age when weight status was assessed during youth, availability of longitudinal data, confounding factors, and outcome measure (serum lipids, carotid intima media thickness (IMT) or ankle brachial index (ABI), for example), may contribute to disparate findings. An alternative to using IMT to predict pre-clinical CVD, ABI is a low-cost, non-invasive tool that acts as a surrogate for the presence of CVD risk by detecting arterial occlusions. It is inversely associated with the degree of sub-clinical atherosclerosis determined by IMT (9-11) and several large-scale longitudinal studies have shown that low ABI is associated with a marked increase in CVD risk (reviewed in (12)).

We conducted a follow-up study to determine the relationship between childhood weight status and CVD risk during middle adulthood among participants in a 1982 study on heart disease (Heartwatch). We tested the hypothesis that BMI \geq 85th percentile during youth is associated with increased CVD risk measured by ABI and serum lipids in adulthood, after controlling for the effect of adult weight status on disease.

Methods and Procedures

Study Design and Participants

We conducted a follow-up study of the original Heartwatch Study, conducted in 1982 in Marshfield, Wisconsin (13). Marshfield is a rural community with a relatively stable population, served by a single major medical center, the Marshfield Clinic. Briefly, all students in public and private schools in Marshfield, WI in 1982 were invited to participate in a cross-sectional cardiovascular risk factor screening study. Participating children/adolescents had height, weight, and fasting serum lipids measured at school; questionnaires were administered covering demographic characteristics, medical history, family history and diet. The original study included 3106 children aged 5-15 (13). Beginning before the original study, outpatient health care provided to Heartwatch study participants at Marshfield Clinic and inpatient data provided at St. Joseph's Hospital (the only hospital in the immediate area) have been recorded in a combined medical record which has followed participants consistently throughout the intervening period. Most of these historical data were available electronically for this study (diagnoses since the creation of the medical record to the present, laboratory results 1985 to present, height and weight 1994 to present). BMI results, for example, were obtained as available from the electronic medical record (EMR) from 1994-2010, with a median of 13 results available per subject (range 1-139).

The follow-up enrollment period occurred between April 2008 and December 2009. Based on a feasibility survey, researchers estimated that, of individuals still living and whose addresses were known, approximately 1030 subjects with baseline BMI < 85th percentile and 220 subjects with baseline BMI > 85th percentile would be available for participation. To improve statistical power for identifying associations with a minimum number of subjects, all individuals with BMI > 85th percentile were approached to be enrolled, and a stratified random sample

(initially matched 2:1 by gender and 1982 age groups 5-8, 9-11 and 12-16 years) of former participants whose BMI was \leq 85th percentile were also approached for enrollment. Numbers in the matched sample were subsequently increased to offset enrollment limitations in the BMI > 85th percentile group. The study procedures were reviewed and approved by the Marshfield Clinic Institutional Review Board. All participants provided written consent prior to enrollment.

Recruitment and Enrollment

A public affairs campaign with radio and newspaper press releases was used to promote regional public awareness of the follow-up study. Telephone numbers for 3075 Heartwatch participants (99%) were obtained from the Marshfield Clinic EMR. Up to five calls were made by trained personnel utilizing telephone scripts to locate the study participant and schedule an appointment for the follow-up examination. Weekend, evening and holiday visits were scheduled as needed. All visits took place at Marshfield Clinic.

Specimen Collection and Handling Procedures

Anthropometrics – Height was determined using a stadiometer and weight was measured using a beam scale, according to standard methods (14). Measurements were made in duplicate with the subject wearing light clothing (no shoes). Weight was recorded to the nearest ¼ pound; BMI was calculated (kg/m^2) and classified according to National Institutes of Health (NIH) and Centers for Disease Control and Prevention (CDC) recommendations (underweight <18.5, normal weight 18.5-24.9, overweight 25-29.9, obesity class 1, 30-34.9, and obesity class 2, 35-39.9) (15, 16). Study staff measured waist and hip circumferences according to standard procedures (17). The mean of two measurements was determined for each measurement site. Gender-specific cut-offs from the NIH were used to classify the point at which waist circumference was associated with increased disease risk: for males, > 40 inches

(102 cm) and for females, > 35 inches (88 cm) (15). Waist:hip ratio was calculated and used as a measure of fat distribution.

Blood pressure – Blood pressure (BP) was measured using standard procedures from the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) (18). Three BP measurements were recorded after the subject had been sitting at rest for five minutes. The average of the last two measurements was used for analysis. Hypertension was defined as BP > 140/90 (19).

Ankle-brachial index – After subjects rested supine for five minutes, a hand-held Doppler probe (Nicolet Pocket-Dop II, Nicolet Vascular, Golden CO) was used to measure systolic pressures in the right brachial artery, right dorsalis pedis and posterior tibial arteries, and the left brachial artery (18). Pressures were measured twice, and averaged for each limb. The ABI was calculated by dividing the minimum leg pressure by the maximum brachial pressure (20). An ABI value below 0.90 was considered abnormal (21). Normal ABI was defined as 1.10-1.29, low-normal as 1.00-1.09, and borderline normal as 0.90-0.99 (21). No participants had an ABI above 1.29.

Serum lipid and lipoproteins – A three-mL fasting (14-hour) blood sample was obtained for measurement of serum lipids and lipoproteins (total, HDL, LDL cholesterol; triglyceride level). Lipids were analyzed according to standard methods by Marshfield Laboratories (22).

Questionnaire – A self-administered questionnaire containing basic demographic and health status information was sent to subjects for completion prior to their clinical examination. This questionnaire included questions about smoking (“During your life, have you smoked at least 100 cigarettes?”) and self-reported diseases (“Do you have a history of any of the following health conditions: asthma, blood clots, cancer, depression, diabetes, gall bladder disease, heart disease, high cholesterol level, high blood pressure, obesity, osteoarthritis, polycystic ovary disease, rheumatoid arthritis, sleep apnea, other?”). Study staff reviewed the questionnaire for completeness during the follow-up visit.

Historical measures – Health history information, including height and weight, was abstracted from the electronic medical record for any participant who received at least some medical care at the Marshfield Clinic.

Analytic Approach

Standard descriptive statistics were used to characterize the follow-up study and to assess the extent to which the follow-up study participants were representative of the original study population. The primary exposure variable was BMI z-score; child and adult percentiles were analyzed as continuous variables by normalizing BMI results to age- and sex-specific z-scores, using data from the National Health and Nutrition Examination Survey. For children, the normalization used the United States Growth Charts smoothed percentile curves based on the May 30, 2000 release. For adults, tabled NHANES data from the same release were smoothed using the LMS method for creation of the z-scores (25, 26). Categorical variables for child and adult weight status were created based on standard definitions (27). Primary outcome variables used as measures of high cardiovascular disease risk were ABI and serum LDL. Thirty-seven subjects with medically treated dyslipidemia were excluded from analyses of LDL. Dichotomous variables were defined as abnormal ABI (< 0.9, yes/no) and dyslipidemia (as defined above, yes/no). The primary analyses were based on analysis of variance (ANOVA) for general linear models. Each primary outcome was analyzed for association with the baseline (child/adolescent) and follow-up (adult) BMI z-scores, with gender, age, and smoking (ever/never smoked 100 cigarettes) as covariates in the models. All analyses were performed using SAS® Version 9.2 (SAS Institute, Cary, NC), and a *P* value < 0.05 was considered statistically significant.

Results

Of 3062 still-living 1982 Heartwatch study participants, a total of 540 with BMI > 85th percentile were approached, and a total of 1567 with BMI < 85th percentile were approached to be enrolled. A total of 647 were enrolled in the follow-up study: 153 in the 1982 BMI ≥ 85th percentile group, and 494 in the 1982 BMI < 85th percentile group (**Figure 1**). In both the original Heartwatch study and the follow-up study, 44% of the participants were male in the BMI ≥ 85th group and 48% were male in the BMI < 85th group (**Table 1**). Overall age of participants was comparable between BMI groups in both study populations. BMI z-score, serum LDL, 2008-09 systolic and diastolic BP and waist:hip circumference ratio were all slightly lower on average in the BMI < 85th group compared to the BMI ≥ 85th group. There was a significant difference in calculated ABI between BMI groups for men, but not for women.

BMI z-score in 1982 was moderately correlated with BMI z-score in 2008-09 in both men ($r = 0.57$, $p < 0.001$) and women ($r = 0.58$, $p < 0.001$) (**Figure 2**). Children 9 years and younger when the original study was conducted ($n=238$) showed a correlation of original/current BMI z-scores of 0.55, while children 12 years and older ($n=165$) showed a correlation of 0.67 (p for difference in correlations = 0.05). Levels of LDL in 1982 were associated with LDL levels in 2008-09 ($r = 0.44$, $p < 0.001$).

Linear regression analysis revealed that neither the 1982 BMI z-score nor smoking at least 100 cigarettes over a participant's lifetime were significantly related to continuous ABI score or serum LDL in models also including gender, age, and 2008-09 BMI z-score (**Tables 2 and 3**). 2008-09 BMI z-score was positively associated with increased ABI score and serum LDL in these models.

Discussion

Childhood BMI was not significantly associated with adult CVD risk factor levels measured by ABI and LDL after controlling for adult weight status in our population. However,

childhood BMI tracked closely into adulthood, and this association limited our ability to determine effects of childhood BMI independent of adult BMI. These findings suggest that regardless of whether childhood BMI is independently associated with adult CVD risk factor levels, it is strongly related to adult weight status.

Our findings are supported by others who have suggested that adult obesity modifies the association between childhood adiposity and sub-clinical CVD assessed by IMT in adulthood (28-30). Freedman et al. compared childhood and adult obesity as predictors of adult IMT in the Bogalusa Heart Study and found that adult obesity modified the association between childhood adiposity and IMT (7). Similarly, Li et al. found that higher childhood BMI predicted thicker adult IMT, but that the cumulative burden of risk factors from childhood to adulthood provided no additional predictive value for thicker adult IMT (5). Finally, both Lloyd et al. and Juonala et al. found that although obesity and overweight in childhood and adulthood are related, people who were overweight or obese during childhood but nonobese as adults had lowered risk compared to obese adults (29, 30). However, in the Cardiovascular Risk in Young Finns Study, Raitakari et al. provided evidence that cardiovascular disease risk factors in 12-18 year-olds, including increased BMI, were independently associated with adult IMT, even after adjusting for adult risk factors (4). However, risk factors for cardiovascular disease measured during childhood (ages 3-9 yrs) were either weakly (in adult men) or not at all (in adult women) associated with IMT.

The hypothesis that BMI tracks from childhood to adulthood is widely supported in the literature; Juhola et al. observed in the Cardiovascular Risk in Young Finns study (3) and Whitaker et al. observed in a large observational cohort in the United States (31) that adolescent BMI is closely related to adulthood BMI, and reviews by Lloyd et al. (29) and Serdula et al. (32) corroborate these findings. We were able to add to the existing knowledge base by examining the contribution of obesity over time to adult disease due to the extensive EMR system at the Marshfield Clinic, and to the stability of our cohort (33, 34). Our finding of non-significant additional predictive value related to the degree of overweight over time is supported

by Li et al. (5) who found that the cumulative burden of risk factors from childhood to adulthood provided no additional predictive value for disease.

This study used ABI to determine higher risk for cardiovascular disease. Ankle brachial index has been validated as a tool to determine CVD risk (35) and has been used widely to study CVD risk in other populations (20, 36-40). This study also used childhood BMI z-scores calculated from CDC growth charts (41) to stratify participants. Others have suggested that earlier CDC growth charts did not account for the rapid rise in childhood BMI during the 1980s and that confounders such as level of physical activity and quality of diet are apt to change the interpretation of this value (42). Finally, LDL was used as a marker of dyslipidemia in this study; others have reported a link between BMI and high LDL (43).

Our analysis did not account for several factors that may have influenced risk of adult CVD risk factor levels, including the wide range of ages in the original Heartwatch study (5 years-15 years) (13); and the years between the original Heartwatch study and the follow-up study, during which other risk factors or factors mitigating risk may have influenced or confounded the relationship between childhood BMI, adult BMI and risk of CVD. A review by Reilly et al. has found evidence to suggest that early childhood BMI does not track to adult BMI as closely as adolescent BMI does (6); this is supported by our finding that the BMI of children 12 years old and older had a tighter correlation to adult BMI compared to the BMI of children 9 years old and younger. However, the interval between key visits (1982 and 2008-2009) did not vary greatly among subjects, suggesting that the effects of time within our study population would have been relatively consistent across participants.

Our study is also limited by the lack of heterogeneity in racial or ethnic background of study participants. As such, we cannot generalize our findings to other populations and further work in this area is needed. Nonetheless, this study provided a rare opportunity to follow up on a cohort assembled in childhood with detailed data related to BMI and health indicators, and to examine the long-term CVD-related consequences of childhood overweight.

Our findings show strong associations for adult BMI with adult early CVD, and other factors including childhood BMI were not significantly associated with CVD risk factors when accounting for age, gender, and current BMI. Future research is needed to better understand the effect of timing of overweight on health outcomes throughout the lifespan.

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Competing interests

LAC is currently an employee of Abbott Nutrition; at the time this research and analysis was completed, she was an employee of Marshfield Clinic.

Author contributions

LAC conducted research with assistance from RLB and other colleagues at Marshfield Clinic Research Foundation. RLB completed all statistical analysis. MS drafted and finalized the manuscript. CE provided scientific and statistical perspective and guidance throughout the research process. All authors read and approved the final manuscript.

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Table 1: Demographic characteristics of participants.

Characteristic	Male		Female	
	1982 BMI \geq 85 th percentile	1982 BMI < 85 th percentile	1982 BMI \geq 85 th percentile	1982 BMI < 85 th percentile
N	67	236	86	258
Age (years)	38 (36, 39)	38 (36, 40)	37 (35, 39)	37 (35, 39)
BMI (kg/m ²)	33 (29, 39)**	27 (24, 30)	34 (27, 39) †	25 (22, 29)
Systolic BP (mmHg)	120 (111, 128)**	116 (109, 123)	111 (104, 121) †	106 (99, 114)
Diastolic BP (mmHg)	82 (75, 87)**	78 (73, 83)	75 (69, 81) †	71 (66, 77)
Waist (cm)	110 (98, 125)**	94 (88, 102)	102 (91, 119) †	84 (75, 94)
Hip (cm)	113 (103, 124)**	103 (97, 107)	117 (106, 129) †	103 (96, 110)
ABI	1.14 (1.11, 1.18)**	1.12 (1.09, 1.16)	1.13 (1.09, 1.15)	1.11 (1.07, 1.16)
Total cholesterol (mg/dL)‡	203 (175, 223)*	186 (161, 209)	175 (161, 202)	175 (157, 194)
HDL cholesterol (mg/dL)	39 (32, 44)	40 (33, 47)	49 (42, 59)	49 (42, 61)
LDL cholesterol (mg/dL)	133 (114, 149)**	117 (98, 140)	103 (89, 121)	104 (88, 123)
Triglycerides (mg/dL)	122 (87, 191)	110 (71, 179)	89 (55, 142)	76 (53, 113)

Values shown are median (25th percentile, 75th percentile), as measured in 2008-2009. P-values indicated by asterisks are calculated from Wilcoxon rank-sum tests.

* $p < 0.05$, ** $p < 0.01$ among males with BMI \geq 85th percentile compared to males with BMI < 85th percentile

† $p < 0.01$ among females with BMI \geq 85th percentile compared to females with BMI < 85th percentile.

‡ For serum lipid data, $n = 59$ for BMI \geq 85th percentile and $n = 216$ for BMI < 85th percentile.

Table 2: Regression analysis of factors influencing ABI score.

Characteristic	Parameter estimate (β) ^a	Standard error	P-value
Male gender	0.35	0.50	0.48
Age	-0.10	0.09	0.25
1982 BMI z-score	0.33	0.33	0.33
2008-09 BMI z-score	0.65	0.33	0.047
Smoked at least 100 cigarettes in lifetime	0.54	0.51	0.28
Intercept	115.43	3.41	< 0.0001

^a Regression values are for a model including all covariates at once.

Table 3: Regression analysis of factors influencing serum LDL.

Characteristic	Parameter estimate (β) ^a	Standard error	P-value
Male gender	13.36	2.36	< 0.0001
Age	1.36	0.43	< 0.01
1982 BMI z-score	-2.63	1.57	0.09
2008-09 BMI z-score	5.87	1.55	< 0.001
Smoked at least 100 cigarettes in lifetime	-0.49	2.39	0.84
Intercept	57.01	16.03	< 0.001

^a Regression values are for a model including all covariates at once.

Figure Legends

Figure 1: Participant cohort diagram. Of the original 3106 Heartwatch participants, 44 were deceased and 32 had no 1982 BMI measurement on file during recruitment in 2008. A stratified random sample was taken of participants with 1982 BMI \geq and $<$ 85th percentile so as to match the BMI proportions of the original Heartwatch study.

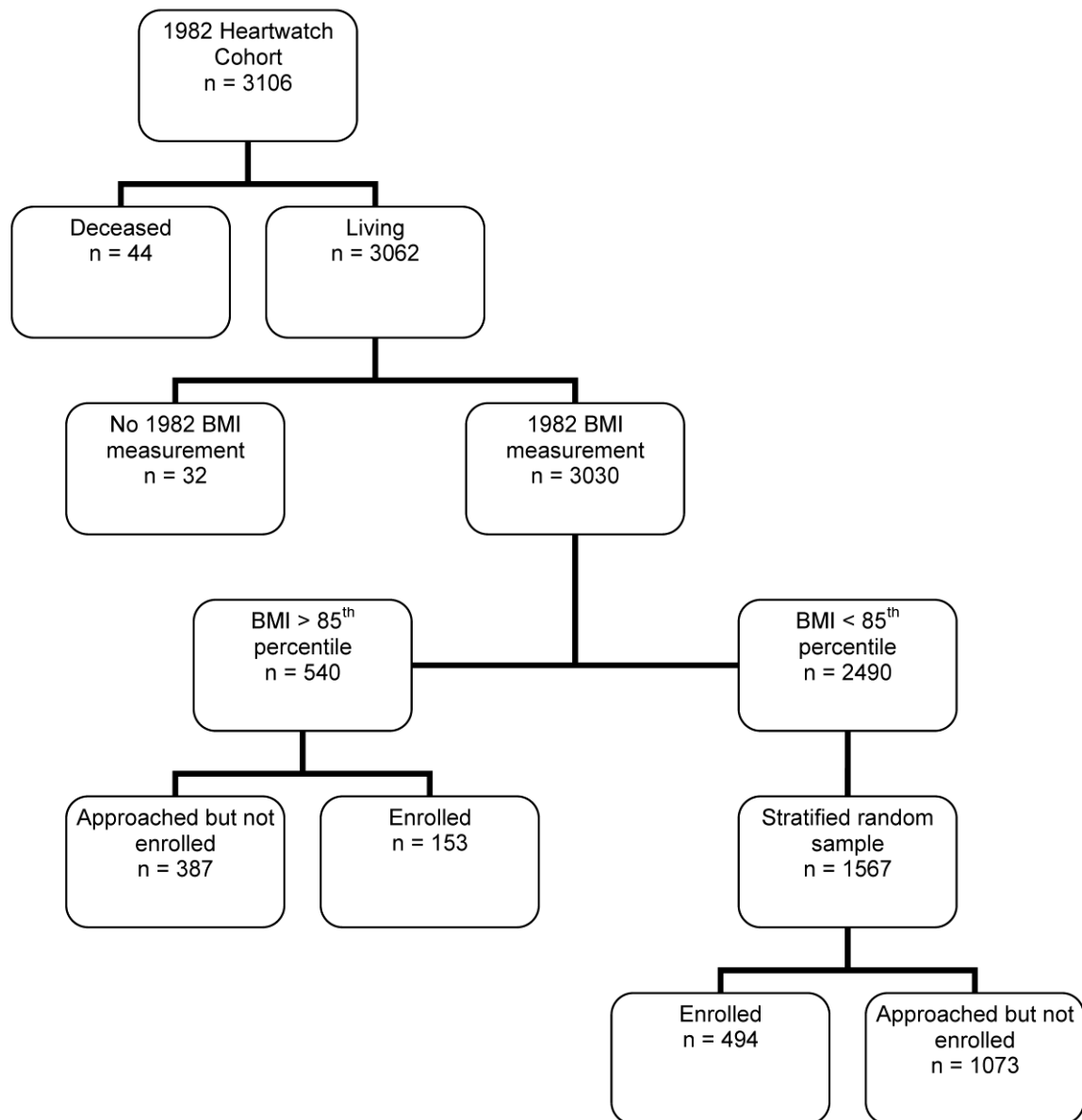


Figure 2: Correlation of childhood and adult BMI. After z-scores were assigned to each participant's childhood and adulthood BMI, these values were found to be tightly correlated with each other, both for men and for women. Pregnant women were not included in this analysis but are shown in the figure for reference. This model assumes a linear relationship between childhood and adult BMI. R = Spearman rank correlation coefficient.

