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Original Article

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Sleep efficiency and the metabolic risk score in very active older women and men

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Abstract

Study Objectives: Sleep disruption is a risk factor for obesity, diabetes, and cardiovascular disease in older adults. How physical activity (PA) interacts with the negative cardiometabolic effects of poor sleep is not known. We objectively measured sleep efficiency (SE) in very active older adults and examined the association between SE and a continuous Metabolic Syndrome Risk Score (cMSy).

Methods: Very active older adults (age ≥65 years) from a Master's Ski Team (Whistler, Canada) were recruited. Each participants wore an activity monitor (SenseWear Pro) continuously for 7 days to provide measures of both daily energy expenditure (metabolic equivalents, METs) and SE. All components of the metabolic syndrome were measured and a principal component analysis was used to compute a continuous metabolic risk score (cMSy, sum of eigenvalues ≥1.0).

Results: A total of 54 participants (mean age 71.4 years, SD 4.4 years, and 24 men and 30 women) were recruited and had very high PA levels (>2.5 h per day of exercise). Initially, there was no significant association between SE and cMSy ($p = 0.222$). When stratified by biological sex, only men showed a significant negative association between SE and cMSy (Standardized $\beta = -0.364 \pm 0.159$, $p = 0.032$).

Conclusions: Only older men show a significant negative association between poor SE and increased cardiometabolic risk, despite high levels of PA.

Key words: sleep quality; metabolic syndrome; physical activity; sex differences

Statement of Significance

Poor sleep is a known risk factor for high cardiometabolic risk and the development of metabolic syndrome in older adults. High physical activity (PA) is protective against developing metabolic syndrome but how PA interacts with the negative cardiometabolic effects of poor sleep is not known. We objectively measured sleep efficiency (SE) in very active older adults and examined the association between SE and a continuous Metabolic Syndrome Risk Score (cMSy). We showed that despite extremely high levels of PA, poor SE was associated with worsening cardiometabolic risk (as measured by cMSy) in older men only. This suggests that even very active older adult men might respond to sleep hygiene interventions to further reduce cardiometabolic risk.

Introduction

Metabolic syndrome (MS), a grouping of criteria associated with increased cardiometabolic risk is becoming increasingly common, affecting approximately 35% of all adults in the United States [1]. Increasing age elevates this risk, with approximately half of all US adults 60 years of age or older meeting current MS criteria1. MS has profound public health implications, increasing both cardiovascular morbidity, and overall mortality rates [2]. Poor sleep is a co-existing public health issue that has long been associated with increased mortality [3], with approximately one-quarter of people sleeping less than current recommendations [4]. These two public health issues have strong interactions, with a recent meta-analysis showing a strong association between poor sleep and MS [5].

Recent MS work has questioned viewing this condition as a binary dichotomy (a yes/no diagnosis), advocating the use of a single continuous score that incorporates all the values in this cluster of cardiometabolic risk factors (cMSy) [6]. This approach has been used in past epidemiological studies of MS showing low

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levels of cMSy at higher activity levels and high levels of cMSy in more sedentary participants [7, 8]. Both physical activity (PA) [9] and better quality sleep [10] have been shown to individually have strong inverse associations with the development of MS in older adults. The question of how protective high levels of PA are for the negative cardiometabolic effects of poor sleep is very much an open question.

We looked to address this knowledge deficit by examining the association of continuous metabolic risk and both sleep efficiency (SE) and total sleep time (TST) in an extremely active group of older adult Master's athletes. Our objective was to examine this population who have extremely high levels of PA and the association between cMSy and objective measures of SE and TST. We hypothesized that even in a group that greatly exceeded current PA guidelines, SE and TST would still show positive associations with cMSy.

Methods

Subjects

Study protocol was approved by the Human Subject's Committee of the University of British Columbia and all participants gave written consent. All participants were approached using posters and a series of information sessions given to the Whistler Master's Ski Club (WMSC, Whistler, British Columbia). This group consists of highly active older adults that have organized training events during the off-season. All participants were recruited in the context of previous activity studies [8, 11, 12]. The WMSC is an alpine, non-competitive ski team that trains together 3 times per week for approximately 1 to 2 h at the local gymnasium (a combination of strength and aerobic training) 3 times per week.

Inclusion/exclusion criteria

All participants were members of the WMSC, and had to be 65 years or older. All participants with a previous cardiovascular illness (previous stroke, previous transient ischemic attack, angina, and myocardial infarction), those with a history of coronary revascularization, those with history of diabetes mellitus, current smokers, and those that used recreational drugs were excluded.

Data collection

Each participant attended a single laboratory visit to collect clinical, anthropomorphic, blood pressure, and blood work. As per current guidelines, we collected each component of the MS (waist circumference, blood pressure, triglycerides, high-density lipoprotein levels (HDL), and fasting blood glucose) [13]. We also measured a 2-h oral glucose tolerance test as per current American Diabetes Association guidelines [14]. All assessments were done in the off-season.

At this same laboratory visit, instructions were given on the use of the activity monitor, and the device was applied to each participant. A postage-paid envelope was provided to all for the return of the device. A member of the study team was available 24 h per day to answer questions about the use of the activity monitor. Our laboratory coordinator also recorded a medication list (including sedative use), caffeine use, and past medical history for each subject. Caffeine use was converted to the equivalent number of cups of coffee per day [15].

After 20 min of quiet rest, blood pressure was measured with each participant in the seated position using a digital sphygmomanometer (Welch Allyn, ABPM 7100). Blood pressure was averaged over three readings taken 5 min apart. Weight (by mechanical beam balance scale) was measured to the nearest 0.1 kg, along with height (Healthometer stadiometer) to the nearest 0.1 cm. As per current guidelines [16], waist circumference was measured using a plastic tape measure (at the level of the umbilicus). Triglycerides and high-density lipoprotein levels were measured in a private laboratory (Lifelabs).

At the end of the laboratory session, the activity monitor was fitted to each participant using an armband around the right upper triceps. The activity monitor (SenseWear Pro, BodyMedia, Sword Medical Limited, Blanchardstown, Dublin) was used to measure energy expenditure (EE) for 24 h per day, 7 days per week. Each participant was told to wear the accelerometer even during sleep, and to only remove the device while showering or bathing.

Energy expenditure and SE

The SenseWear Pro device calculates average energy expenditure (in Metabolic Equivalents, METS) and SE (percent time sleeping while lying down) through the incorporation of accelerometer data in 3-axes, gyroscope data, skin temperature, heat flux, and galvanic skin response (perspiration measure). This involves the use of proprietary algorithms that have been previously validated to measure both energy expenditure [17] and SE [18] in the adult population. The use of this device to measure energy expenditure has also been used in previous investigations [19], and has been validated against gold standard doubly labeled water techniques [20]. Each device was fitted to the right upper triceps and worn for 24 h per day, 7 days per week [12]. Proprietary software (Body Media InnerView Research Software, Version 5.1) was used to analyze data for both sleep and energy expenditure measures [21].

As per current guidelines for wearable activity devices [22], to be included in the analysis each participant had to wear the accelerometer for at least 5 valid days with which at least one of the valid days was a Saturday or a Sunday. 21 h of recorded activity on the accelerometer was defined as a valid day. Skin conductance measures provided by the SenseWear Pro allow the device to be able to detect when it is being worn, allowing a distinction to be made between an absence of PA and not wearing the device [21].

Statistical analysis

The continuous metabolic risk score (cMSy) [6] incorporated all risk factors found in the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults [23]; the components of MS consist of blood pressure, triglycerides, HDL cholesterol, waist circumference and fasting blood glucose [23]. A blood pressure index was calculated by averaging systolic and diastolic blood pressure [6]. Data are available on request (Kenneth.Madden@ubc.ca).

To create a single continuous cMSy score, a principal component analysis with varimax rotation was used on all components of the MS to derive principal components (PCs, eigenvalue 1.0) representing a large fraction of the variance observed in the five variables. As in previous work [6], cMSy was then computed by summing the first three PCs (weighted for the relative contribution of each to the variance). This creates a single continuous variable (cMSy) that represents an overall measure of metabolic risk.

For our initial statistical analysis, our outcome variable was cMSy, and our predictor variables were age, biological sex, EE, body mass index (BMI), and TST/SE. After this initial analysis, our analysis was then stratified by biological sex. Density plots were examined

Table 1. Demographics in subjects with high and low sleep efficiency

	Lower half of SE (n = 27)	Upper half of SE (n = 27)	All subjects (n = 54)	p Value
Age	71.8 (4.7)	71.1 (4.2)	71.4 (4.4)	0.585
Biological Sex	13 women, 14 men	17 women, 10 men	30 women, 24 men	0.438
Waist Circumference (cm)	88.4 (12.0)	84.7 (8.3)	86.5 (10.3)	0.190
BMI (kg/m^2)	24.9 (1.0)	23.6 (2.6)	24.2 (2.9)	0.100
Triglycerides	1.09 (1.04)	0.91 (0.36)	1.00 (0.59)	0.287
High-density lipoprotein	1.51 (0.36)	2.19 (0.31)	1.85 (0.51)	0.462
Systolic Blood Pressure (mm Hg)	117 (16)	117 (21)	117 (15)	0.995
Diastolic Blood Pressure	68 (5)	68 (10)	67 (7)	0.924
Mean Arterial Pressure	84 (10)	84 (10)	84 (7)	0.935
Fasting Blood Glucose (mmol/L)	5.2 (0.5)	5.0 (0.5)	5.1 (0.7)	0.410
Number of Medications	1.5 (1.6)	1.4 (1.0)	1.4 (1.5)	0.923
DEE (in METs)	1.49 (0.21)	1.48 (0.26)	1.48 (0.22)	0.887
cMSy	0.09 (0.68)	-0.39 (0.62)	-0.16 (0.59)	0.242
SE (percent)	74 (5)	89 (5)%	81 (7)%	<0.001*
Total Sleep Time (h)	5.8 (1.0)	7.4 (0.5)	6.6 (1.5)	<0.001*
OGGT (mmol/L)	7.7 (3.1)	6.4 (1.5)	7.0 (2.2)	0.049*
Caffeine intake (equivalent cups of coffee per day)	1.6 (1.6)	1.3 (1.5)	1.4 (1.5)	0.400
Alcohol intake (drinks per week)	6.7 (6.2)	6.0 (5.7)	6.3 (5.9)	0.666

Abbreviations: Mean (SD), BMI, body mass index (kg/m^2), SE, sleep efficiency (percent); *, DEE, daily energy expenditure (metabolic equivalents, METs); cMSy, continuous metabolic syndrome risk score; OGGT, oral glucose tolerance test; mmol/L (millimoles per liter); *, p-value < 0.05.

prior to statistical analysis for data skewing, and any variable that showed skewing issues was logarithmically transformed (base 10) prior to both the univariate and multivariable analyses. Variance inflation factors were examined for issues with multicollinearity (using a conservative threshold of two) to ensure that the assumption for multivariable regression was met [24]. After our initial models were created, a stepwise regression was performed with the least significant predictor removed sequentially until a final parsimonious model remained. Akaike's Information Criterion (AIC) was calculated after each predictor was removed from our models until the smallest AIC was obtained, indicating the best fit [24].

All statistical analysis was done using the R core software package 4.1.2 (2021-11-01) using a significance level of $p < 0.05$ [25]. Mean (standard deviation, SD) was used to express all descriptive statistics. Mean and confidence intervals were used to express all standardized beta (β) coefficients.

Results

Subject recruitment

A total of 60 participants from the Master's ski team were recruited and 55 participants met our inclusion and exclusion criteria. Our criteria for successful accelerometer data collection was not met in one participant (who removed the device during non-sleeping hours), which left a total number of 54 participants. On average, our accelerometers have worn an average of 99 (0)% of the study time, excluding the participant that did not reach criteria for data collection.

Subject characteristics (Table 1)

Demographic and baseline measures for all subjects, as well as those in the upper (above the median) and lower (below the median) halves of SE, are presented in Table 1. As seen in Table 1 our participants had quite a mean TST of about 6.5 h and a mean SE of 81%. Table 2 shows demographic and baseline measures for women and men. Our participants performed 3.9 (1.5) h of light activity and 2.6 (1.5) h of moderate/vigorous activity per day.

Comparing subjects in the lower versus upper halves of SE demonstrated significantly higher results from the oral glucose tolerance test in subjects with lower measures of SE (p = 0.049). Also, participants in the upper half of SE slept significantly longer ($p < 0.001$*). None of our participants used sedative medications.

With respect to biological sex differences, women showed higher high-density lipoprotein ($p < 0.001$), lower cMSy ($p < 0.001$), lower waist circumference ($p < 0.001$), lower body mass index (BMI, p = 0.002), and lower alcohol intake (p = 0.041). There was a trend for lower TST (p = 0.685) and higher OGGT responses (p = 0.181) in women, but this did not reach statistical significance (see Table 2).

Principal component analysis

In our initial analysis, a principal component analysis with varimax rotation was used on all components of the MS. When our correlation matrix was examined, there was no evidence of multicollinearity and our determinant value was 0.337 as per current standards [26]. Our Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy was 0.613 (>0.5) and our Bartlett's test of sphericity was statistically significant ($p < 0.001$). After examination of the scree plot, we extracted three factors that explained 82% of the variance. We then performed a confirmatory factor analysis using Oblimin with Kaiser Normalization, which did not increase the variance explained by our three factors [26].

Univariate analysis (Table 3)

None of our predictor variables showed skewing, eliminating the need for logarithmic transformation prior to statistical analysis. When all participants were analyzed together, only EE (negative association, $p = 0.007$) and BMI (positive association, $p < 0.001$) demonstrated significant associations with cMSy. Women showed significant associations between EE (negative association, $p = 0.003$) and BMI (positive association, $p = 0.029$) with cMSy, while men only showed a significant association between cMSy and BMI (positive association, $p = 0.003$).

Multivariable analysis (Table 4)

Initially, our multivariable regression model contained continuous predictor variables (SE, EE, age, and BMI) as well as a logistic (biological sex) predictor variable that was responsible for 47% of cMSy variance (see Table 4). Our highest variance inflation factor

Table 2. Demographics in women and men

	Women ($n = 30$)	Men ($n = 24$)	*p* Value
Age	70.5 (3.8)	72.6 (4.9)	0.084
Waist circumference (cm)	81.4 (8.2)	92.9 (8.8)	<0.001*
BMI (kg/m^2)	23.2 (2.7)	25.5 (2.9)	0.002*
Triglycerides	0.89 (0.38)	1.15 (0.78)	0.101
High-density lipoprotein	2.08 (0.38)	1.57 (0.44)	<0.001*
Systolic blood pressure (mm Hg)	116 (22)	118 (15)	0.510
Diastolic blood pressure	67 (11)	69 (10)	0.300
Mean arterial pressure	83 (5)	85 (10)	0.352
Fasting blood glucose (mmol/L)	5.0 (1.1)	5.2 (0.5)	0.461
Number of medications	1.2 (0.5)	1.7 (1.5)	0.164
EE (in METs)	1.49 (0.22)	1.48 (0.24)	0.919
cMSy	–0.21 (0.38)	0.27 (0.54)	<0.001*
SE (percent)	83 (11)%	80 (10)%	0.255
Total Sleep Time (h)	5.7 (1.1)	7.3 (0.5)	0.685
OGGT (mmol/L)	7.4± (2.7)	6.5 (2.0)	0.181
Caffeine intake (equivalent cups of coffee per day)	1.6 (1.6)	1.3 (1.5)	0.415
Alcohol intake (drinks per week)	4.9 (4.9)	8.1 (6.9)	0.041*

Abbreviations: Mean (SD), BMI, body mass index (kg/m^2), SE, sleep efficiency (percent); *, EE, energy expenditure (metabolic equivalents, METs); cMSy, continuous metabolic syndrome risk score; OGGT, oral glucose tolerance test; mmol/L (millimoles per liter); *, p-value < 0.05.

Table 3. Univariate regression analysis ($n = 54$)

Response variable	Predictors	R (CI 95%)	*p* Value
cMSy (all subjects)	SE	–0.170 (–0.421 to 0.105)	0.222
	D	–0.091 (–0.352 to 0.184)	0.518
	EE	–0.366 (–0.579 to 0.106)	0.007*
	Age	0.283 (0.016 to 0.512)	0.038
	BMI	0.597 (–0.746 to –0.392)	<0.001*
cMSy (Women)	SE	–0.178 (–0.202 to 0.511)	0.357
	D	0.036 (–0.335 to 0.397)	0.852
	EE	–0.532 (–0.752 to –0.206)	0.003*
	Age	–0.047 (–0.319 to 0.400)	0.805
	BMI	0.398 (–0.663 to –0.044)	0.029*
cMSy (Men)	SE	–0.317 (–0.638 to 0.100)	0.132
	D	–0.142 (–0.516 to 0.278)	0.509
	EE	–0.339 (–0.653 to 0.074)	0.105
	Age	0.299 (–0.118 to 0.627)	0.155
	BMI	0.586 (–0.800 to –0.239)	0.003*

Abbreviations: SE, sleep efficiency (percent); D, sleep duration per day (minutes); EE, energy expenditure (metabolic equivalents, METs); cMSy, continuous metabolic syndrome risk score; BMI, body mass index (kg/m^2); *, p-value < 0.05.

Table 4. Stepwise multivariate regression analysis (n = 54)

	R^2	Standardized β (Standard error)	p value
All subjects, Model 1 F = 8.48	0.47		<0.001*
SE		−0.096 (0.111)	0.390
EE		−0.172 (0.142)	0.230
Age		−0.030 (0.121)	0.808
Biological Sex (Woman)		−0.729 (0.244)	0.005*
BMI		0.364 (0.158)	0.025*
All subjects, MEM F = 20.36	0.44		<0.001*
Biological Sex (Woman)		−0.645 (0.228)	<0.007*
BMI		0.466 (0.114)	<0.001*
Women, Model 1 F(3,25) = 3.694	0.31		0.025*
SE		0.198 (0.177)	0.273
EE		−0.389 (0.242)	0.120
Age		−0.059 (0.186)	0.753
BMI		0.210 (0.255)	0.417
Women, MEM F = 10.67	0.28		0.003*
EE		−0.538 (0.164)	0.003*
Men, Model 1 F = 4.38	0.48		0.011*
SE		−0.380 (0.170)	0.038*
EE		−0.090 (0.231)	0.701
Age		−0.005 (0.209)	0.981
BMI		0.565 (0.210)	0.015*
Men, MEM F = 9.50	0.48		0.001*
SE		−0.364 (0.159)	0.032*
BMI		0.614 (0.159)	<0.001*

Abbreviations: SE, sleep efficiency (percent); EE, energy expenditure (metabolic equivalents, METs); MEM. Minimal Effective Model; R^2, coefficient of determination; β, beta-coefficient; BMI, body mass index (kg/m^2); p, p-value <0.05.

was 2.18 (BMI), indicating that multicollinearity was not an issue. Since our final parsimonious model showed a strong association with biological sex (p = 0.005) we stratified our analysis for women and men. Our second multivariable regression model (see Supplementary Table 5) showed no association between cMSy and TST for all participants (p = 0.355) and also when stratified by biological sex (women, p = 0.752; men, p = 0.244).

In our initial model containing women participants, there was no significant association between cMSy and any of our predictor variables (age, p = 0.753; SE, p = 0.273; EE, p = 0.120; BMI, p = 0.417). Our final model containing woman participants had only EE as a significant predictor variable with cMSy (p = 0.003).

When we analyzed only men, only SE (negative association, p = 0.038) and BMI (positive association, p = 0.015) showed significant associations with cMSy; none of our other predictor variables (EE, p = 0.701; age, p = 0.981) were significant. Our final most parsimonious model explained 48% of the variance in cMSy in men and both SE (negative association, p = 0.032) and BMI (positive association, p ≤0.001) showed significant associations with overall metabolic risk.

Discussion

Principal findings

The older adults recruited for this study vastly exceed PA guidelines [27], spending about 2.6 h per day in moderate or vigorous levels of PA. Our participants were quite an active subject pool; a previous study in Norwegian older adults showed an average of only 29 min per day (ages 70 to 74 years, similar to our participants ' mean age) [28].

Despite this very high level of energy expenditure, older men still showed a significant negative association between SE and cMSy, while no such association was seen in older women subjects. participants with low levels of SE had significantly higher OGGT levels. No association was found between TST and cMSy in our highly active population, in either women or men. Despite their high level of PA, our participants demonstrated a mean SE similar to that seen in normal older adults [29].

Previous work

A recent meta-analysis of observational studies of normal adults showed a clear association between having self-reported insomnia and also meeting the criteria for MS (and also having each of the individual factors of hyperglycemia, hypertension, obesity, or hyperlipidemia) [5]. Most meta-analyses of self-reported sleep durations have also shown an association between meeting MS criteria and short [30, 31] or long [32] sleep durations in normal adults. In an older adult population, similar findings with respect to TST and MS were found [10] although other studies have shown that only long (not short) sleep durations were associated with a higher likelihood of having MS [33]. To the best of our knowledge, our study is the first to demonstrate a lack of association between TST and cardiometabolic risk(cMSy) in extremely active older adult women and men, suggesting that PA might mitigate the risks of short TST in this population.

Our other study outcome measure (SE) is more established as the clinical gold standard for evaluating insomnia, as it is a direct measure of "spending too much time in bed trying to sleep." [34] A previous cross-sectional study of SE in 1499 older adults did not show any association between poor SE and meeting diagnostic criteria for MS, but it did show a negative association between SE and high triglyceride levels and a negative association between SE and hyperglycemia [10]. An observational study of older adults (with accelerometer measures of SE) showed a significant difference in the odds of obesity in those with low SE (less than 85%) [35]. Other studies of SE in middle-aged adults have shown an increased risk

of type 2 diabetes in subjects with poor SE [36] and a strong association between improved SE and glycemic control in older adults admitted to the hospital [37]. Although not exactly the same as SE,self-reported measures of poor sleep quality have also shown an association with an increased risk of MS [38] and obesity [39]. To the best of our knowledge, our findings are the first to demonstrate that in a population of very active older men, there is a negative association between SE and cardiometabolic risk (cMSy).

Potential mechanisms

Our study demonstrated that our participants with lower SE, in addition to increased cMSy, also had elevated oral glucose tolerance tests. This is in keeping with previous theories attributing the underpinnings of poor sleep and MS to an increase in insulin resistance [40, 41] although many have postulated an increase in inflammatory mediators (C-reactive protein and interleukin six) as an intermediate pathophysiologic step [42]. Poor sleep quality also has profound effects on diet, with a decrease in fruit and vegetable intake [43]. In addition, artificially restricting sleep in the laboratory setting has been shown to increase caloric intake [44] in men only [45], which may explain the divergence of our results when we stratified our results by biological sex. Although all our participants were either lean or on the lower boundary of overweight, the underlying presence of obstructive sleep apnea in our participants could partially explain the negative association between SE and cMSy seen in our study. Previous work has shown that even in young lean men, the presence of obstructive sleep apnea as measured by laboratory polysomnography is associated with increased insulin resistance [46], which would lead to both a decrease in SE and an increase in cMSy.

Clinical implications

There are clear clinical benefits of high levels of both sleep quality [47] and PA [48] in older adults. Our results show that in older adult men who on a *daily* basis met current *weekly* PA guidelines, there still is quite a significant negative association between poor SE and cMSy. It is possible that this population might respond to sleep hygiene interventions, although this has yet to be formally investigated.

Limitations and future research

Our study had a cross-sectional design, limiting our ability to make causal inferences. Our study did not formally measure either aerobic fitness or the presence of obstructive sleep apnea, which is a limitation. Although the SenseWear Pro device has been designed to be placed on the upper triceps (and has been validated by previous studies in this position [17, 18]) this placement is different from other commonly used devices. Any postulated mechanisms linking SE and cMSy are purely speculative and require further investigation. More prospective investigations need to be done to see if sleep hygiene interventions can improve cardiometabolic risk in active older men. As well, our participants were part of an extremely active homogeneous culture of PA (alpine skiing), which limits our ability to extrapolate our results to the broader, more sedentary older adult population.

Main conclusions

Despite extremely high levels of PA, poor SE was associated with worsening cardiometabolic risk (as measured by cMSy) only in active older men.

Supplementary Material

Supplementary material is available at *SLEEP Advances* online.

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Data Availability

Data will be made available on request.

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