Type A Behavior and Risk of All-Cause Mortality, CAD, and CAD-Related Mortality in a Type 1 Diabetic Population

22 years of follow-up in the Pittsburgh Epidemiology of Diabetes Complications Study

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OBJECTIVE — To determine whether type A behavior predicts all-cause mortality and incident coronary artery disease (CAD) in a type 1 diabetic population.

RESEARCH DESIGN AND METHODS — Follow-up data (22 years) from the Pittsburgh Epidemiology of Diabetes Complications (EDC) study of childhood-onset type 1 diabetes were analyzed for the 506 participants who completed the Bortner Rating Scale (measuring type A behavior) and Beck Depression Inventory (BDI) at baseline (1986–1988). CAD comprised myocardial infarction as determined by hospital records/Q waves on electrocardiogram (ECG), CAD death (determined by a mortality classification committee), angiographic stenosis, ischemic ECG, and angina.

RESULTS — There were 128 deaths (25.3%) during follow-up. Univariate analysis showed an inverse relationship between Bortner scores and all-cause mortality ($P = 0.01$), which remained significant after allowing for age, sex, duration, HbA1c, education, smoking, BMI, and physical activity ($P = 0.03$). However, the addition of BDI scores attenuated the relationship ($P = 0.11$) with a significant interaction ($P = 0.03$) such that any protective effect against mortality was limited among individuals with lower BDI scores (bottom three quintiles) ($P = 0.07$), whereas no effect was seen in those with higher BDI ($P = 0.97$). Bortner scores showed only a borderline association with incident CAD ($P = 0.09$).

CONCLUSIONS — Those with higher type A behavior have lower all-cause mortality in our type 1 diabetic population, an effect that interacts with depressive symptomatology such that it is only operative in those with low BDI scores. Further research should focus on understanding this interaction.

The incidence of type 1 diabetes, which remains incurable, has continued to rise annually by ~3% (1). Unfortunately, prevention is not currently feasible. Therefore, the exploration of type 1 diabetes complications, untimely mortality and the associated risk factors, must continue. Type A behavior has been described as an action-emotion complex, meaning that the behavior is elicited by the outside environment (2). People characterized as having type A behavior tend to focus toward achieving and accomplishing more in less time than others. Because of these tendencies, these people tend to be competitive, aggressive, time urgent, and work oriented and can become annoyed if things are not achieved in a time frame they find sufficient (2). Therefore, it seems that type A behaviors are not a set of personality characteristics that come about due to the environment; rather, the behavior is a result of predispositions within a person that are exhibited due to specific situations (2). In an earlier review, Matthews et al. (2) noted that although type A behavior was linked to increased coronary heart disease (CHD) risk in the general population, findings were consistently negative in high-risk populations. For example, the prospective Western Collaborative Group Study (WCGS) found that those with type A behavior experienced an increased rate of CHD compared with type B behavior ($P = 0.001$) (3). However, in their high-risk population who had already undergone a CHD event, type A behavior had a lower CHD-associated mortality rate in those surviving 24 h or more than those characterized as having type B behavior ($P = 0.03$) (4). Therefore, it appears that type A behavior may have different effects on health depending on underlying chronic disease status.

Little is known about the psychosocial contribution to the increased coronary artery disease (CAD) risk seen in people with type 1 diabetes beyond depression (5,6), in particular, whether type 1 diabetes is an additional high-risk group in which the inverse association between type A behavior and CAD/mortality exists. Cross-sectional data from the Pittsburgh Epidemiology of Diabetes Complications (EDC) study have shown (using the Bortner Rating Scale) that participants with multiple complications, including CAD, retinopathy, neuropathy, and/or nephropathy, reported less type A behaviors than those without complications ($P < 0.05$) (5). The long length of follow-up now available in the EDC allowed a prospective analysis of the role of type A behavior in...
Type A behavior and mortality in type 1 diabetes

mortality and CAD, and we are unaware of
other investigations of this relationship.
We also investigated the association be-
tween type A behavior and CAD-related
mortality in those already diagnosed
with CAD and whether the established ef-
fect of depressive symptomatology on
CAD incidence interacted with or ex-
plained any effect of type A behavior.
Thus, the aims of the current study were
to investigate the relationships between
type A behavior and mortality, type A be-
behavior and incident CAD, and type A be-
behavior and mortality among those with
CAD during 22 years of follow-up.

RESEARCH DESIGN AND
METHODS—The EDC study is com-
prised of participants diagnosed with type
1 diabetes between 1950 and 1980 at
<17 years of age, seen within 1 year of
diagnosis at the Children's Hospital of
Pittsburgh. Biennial follow-up occurred
since baseline in 1986–1988, which in-
cluded questionnaires with physician ex-
aminations and laboratory analyses of
urine and blood for the first 10 years
and again at 18 years. Data up to the
22-year follow-up are now available. Par-
ticipants ≥18 years of age at study entry
completed the Bortner Rating Scale,
which measures aspects of type A behav-
ior (7) and has been shown to have good
test-retest reliability (2). Participants were
asked to circle the dot on the line that
represents where they believed they fell
between two different sentences. Some ex-
amples of the sentences included “never
late” versus “casual about appointments,”
“always rushed” versus “never rushed,
even under pressure,” and “take things
one at a time” versus “try to do many things
at once, thinking about what I am going to
do next.” 6 CAD was defined as myocardial
infarction confirmed by hospital records or
Q waves on electrocardiogram (Minnesota
Code 1.1 or 1.2); coronary artery stenosis,
defined as ≥50% blockage, or revascular-
ization; ischemic electrocardiogram, de-
efined using Minnesota Code 1.3, 4.1–4.3,
5.1–5.3, or 7.1; angina, diagnosed by an
EDC physician; or CAD death (determined
by a mortality classification committee).

Overall mortality, including CAD-
associated mortality and complication
status, was determined as of 25 February
2011. Searches were performed in both
the Social Security Death Index and the
National Death Index. In order to confirm
each death, death certificates were ob-
tained, plus, as appropriate, 1) hospital
records, 2) autopsy/coroner reports, and
3) interview with next of kin regarding the
death. The underlying causes of death,
and the hierarchical order for all contribut-
ing causes of death, were determined by a
Mortality Classification Committee con-
sisting of two or more physician epidemi-
ologists. This method is based on stan-
dardized procedures (8).

The following covariates were chosen
as potential predictors for our final model:
age, sex, duration, education, physical ac-

tivity, smoking, BMI, insulin dosage, HbA1c,
and depressive symptomatology. These covariates were chosen because
they are previously demonstrated risk fac-
ors for CAD and/or early mortality in type
1 diabetes (9). Education was assessed
using a five-point scale, i.e.: some high
school, high school graduate, some college,
bachelor's degree, and graduate education
beyond bachelor's. Physical activity was as-
essed using questions about current levels
of leisure activities (10), as well as by es-

timating the energy expenditure over the
past week (kcal/week) through use of ques-
tions asking about the daily number of
flights of stairs climbed, the number of
blocks walked daily, and all sports partici-
patation that had occurred over the past
week. Ever smoked was defined as having
had >100 cigarettes over their lifetime.

Insulin dosage was expressed as the num-
ber of units of insulin used per day divided
by the participant’s weight in kilograms. BMI
was calculated as participant’s weight in
kilograms divided by the square of their
height in meters. Fasting blood samples
were analyzed for HbA1c (microcolumn cat-

tion exchange; Isolab, Akron, OH), and
these original HbA1c values were converted
to Diabetes Complications and Control
Trial (DCCT)-aligned HbA1c, for all analyses
using a regression equation derived from
duplicate assays (DCCT HbA1c = 0.14 +
0.83[EDC HbA1c]). Finally, depressive symp-

tomatology was measured using the
Beck Depression Inventory (BDI) (11). The
BDI was a 21-item self-report scale that is
widely used in both healthy and ill popu-
lations. A score of 0–9 indicates minimal
depression, 10–18 indicates mild depres-
sion, 19–29 indicates moderate depres-
sion, and 30–63 indicates severe depres-
sion (11). BDI scores have been shown
to approximate clinically significant
symptoms of depression (11).

Cox proportional hazards models
were used to examine the univariate
and multivariable relationship between base-
line Bortner scores and overall mortality,
CAD incidence over 22 years of follow-
up, and CAD-related mortality among
those with CAD. To assess univariate
associations between baseline Bortner
score and potential covariates (i.e., age,
sex, duration, education, physical activ-
ity, smoking, BMI, insulin dosage, HbA1c,
and depressive symptomatology), Stu-
dent t test or Wilcoxon signed rank test
was used as appropriate. Cox propor-
tional hazards modeling was used to ex-
amine the independent association
between Bortner score and each out-
come (overall mortality, CAD incidence,
and incident CAD death among those
with CAD), adjusting for significant
baseline covariates. All statistics were
performed using SAS 9.3 (SAS Institute,
Cary, NC).

RESULTS—At the EDC baseline exam,
658 participants were seen. One hundred
and fifty two participants were excluded
from this analysis for having missing
covariate measures; however, 60 of these
participants were <18 years of age and
therefore not eligible to complete the
Bortner or the BDI, and an additional 92
participants were excluded, most com-
monly, for missing data on the BDI, Bortner,
or physical activity measures.

As of 25 February 2011, of EDC
participants who completed both the
Bortner and the BDI at baseline, and who
had complete covariate data (n = 506, 250
males and 256 females), there were 128
deaths (23.3%). Those excluded were
less likely to have a high school education
(P = 0.01) and were more likely to be
smokers (P < 0.01), but did not differ
significantly for age, duration, sex,
HbA1c, physical activity, BMI, or depres-
sive symptomatology (Supplementary
Table 1).

Significant covariate differences
existed between those with and without
incident CAD for age, duration, physical
activity, smoking, BDI, and insulin dosage
(Table 1). A significant trend was demon-
strated for both Bortner (P = 0.05) and BDI
score (P = 0.01) at baseline and CAD inci-
dence (Fig. 1A and C). A borderline unin-
variate relationship was seen between baseline
Bortner scores and CAD incidence (P =
0.09). No significant interaction was ob-
served between Bortner and BDI in rela-
tion to CAD incidence.

Differences existed by subsequent
mortality for most baseline covariates.
Deceased participants tended to be older,
with longer diabetes duration, male, less
physically active, and ever smokers and
had a higher HbA1c and BDI score and a
lower Bortner score compared with
interaction was significant (P = 0.01). Multivariable analyses (Table 3) of the association between type A behavior and all-cause mortality were performed with four models, progressively controlling for covariates. Model 1 was adjusted for age and sex, with Bortner score remaining significant (P = 0.01). Model 2 included age, sex, duration, HbA1c, education, smoking, BMI, and physical activity as covariates and demonstrated that Bortner score continued to significantly predict mortality (hazard ratio [HR] 0.99 [95% CI 0.98–1.00]; P = 0.03). For every one-point increase on the Bortner scale, there was a 1% lower mortality risk (Bortner Rating Scale range, 97–258). However, further adjustment for BMI (model 3) reduced the association between Bortner and mortality (P = 0.11).

Model 4 tested for an interaction between the Bortner Rating Scale and BMI for mortality (Table 3) as a significant inverse correlation was found between the two (r = −0.18, P < 0.001). This interaction was significant (P = 0.03), meaning type A behavior is only operative in those with lower BMI scores. Further BMI-stratified analyses were then performed. Based on the quintiles determined in our study population (BMI scores: 0–1, 2–3, 4–6, 7–12, and 13–32) (Fig. 1D), we compared the first three quintiles with the upper two quintiles, resulting in two categories, a BMI score ≤6 versus a BMI score >6. A borderline significant protective effect against mortality was seen with higher type A behavior score in the lower BMI quintiles (P = 0.07), but not with a BMI score ≥7 (P = 0.97).

Performing analyses by sex (mean Bortner score in men, 191.1, vs. women, 185.2; P < 0.01), a significant univariable relationship between Bortner and mortality (0.98 [0.97–0.99]; P < 0.001) was seen in men but not in women (0.99 [0.98–1.00]; P = 0.12). However, a greater proportion of men than women were type A within our population (P = 0.03). The significant relationship among men remained after multivariable adjustment (0.99 [0.98–0.99]; P = 0.03) but was attenuated after further adjusting for BMI (0.99 [0.98–1.00]; P = 0.10). A significant interaction between Bortner and BMI (P = 0.03) was noted, although stratification by the same cut points of BMI as above did not yield any significant differences. Stratifying by minimal to mild versus moderate to severe depressive symptoms, however, demonstrated that men with a minimal to mild BMI score (0.98 [0.97–0.99]; P = 0.02) were protected against mortality compared with those with a moderate to severe BMI score (1.00 [0.97–1.04]; P = 0.63) in multivariable analyses.

Out of 506 participants, there were 64 CAD-related deaths (12.6%). We found that Bortner significantly predicted CAD mortality (0.99 [0.98–1.00]; P = 0.04). The analyses were subsequently repeated excluding non-CAD deaths from the control group (essentially comparing survivors to CAD death [14.3%]), and a significant relationship between Bortner score and CAD death was found as well (0.98 [0.97–0.99]; P = 0.03).

We then examined the predictive value of the Bortner Rating Scale for CAD mortality in those with prevalent CAD. No univariate association was found with death among those with CAD within 22 years of follow-up (P = 0.35).

CONCLUSIONS—We observed a significant relationship between Bortner scores and all-cause mortality, which was attenuated after adjustment for BMI. We also noted the presence of significant effect modification of the relationship between the Bortner and mortality by BMI score. Thus, a borderline significant inverse association between type A behavior and mortality was only apparent among those in the bottom three BDI quintiles, whereas this relationship was lost in the top two quintiles. Analyses stratifying by sex suggested that only men were protected against mortality with a higher type A behavior score, even after adjustment for BMI. However, stratifying by BMI revealed a protective effect of type A behavior only in those with minimal to mild, but not moderate to severe, depressive symptoms. We found a borderline significant relationship between Bortner scores and incident CAD, which was attenuated after adjustment for duration.

To our knowledge, this is the first study to investigate the relationship between type A behavior and all-cause mortality in a type 1 diabetic population. The strengths of our study are the long follow-up and the completeness of data obtained for our population. In addition to demonstrating the importance of type A behavior and depressive symptomatology, our results affirm the role traditional, important covariates play on CAD development and early mortality in type 1 diabetes. Those with the highest type A behavior scores were at the lowest mortality risk, which is consistent with most
of the literature demonstrating that high-risk populations are protected with greater type A behavior (2, 3, 5). However, with the addition of BDI to our model, this relationship was attenuated. After determining that the Bortner scale and BDI were inversely correlated, we tested for an interaction between them to determine if the protective effect we observed from high type A behavior was really due to the low depressive symptomatology score in this group. The interaction term was significant, suggesting that type A behavior may be protective against mortality in the absence of depressive symptomatology (although this was only borderline significant). Any protection from type A behavior appears to be lost once the higher quintiles of depressive symptomatology are reached. This suggests that depressive symptomatology is a stronger predictor of mortality than type A behavior in type 1 diabetes. Indeed, the death rate was 17.8% in the bottom three quintiles, approximately two times lower than in the top two quintiles, at 34.5%.

The importance of depressive symptomatology in type 1 diabetes is expected, as it has been frequently demonstrated that those with high depressive symptomatology are at an increased mortality (12) and morbidity risk (including diabetes complications) (13). Comorbid depression and type 1 diabetes is also associated with poorer diabetes self-management and metabolic control, decreased quality of life, and higher healthcare usage (12). Our previous research showed that BDI score significantly predicted CHD even after controlling for hypertension, waist-to-hip ratio (WHR), white blood cell count, fibrinogen, smoking status, distal symmetric polyneuropathy, and overt nephropathy. However, this relationship became attenuated after the addition of all possible variables in the mediation analysis (6). Depressive symptomatology has also been found to increase WHR in both sexes (14) and appears to play an important role in the incidence and progression of type 1 diabetes–associated complications, as confirmed in our study.

We hypothesized that Bortner scores would continue to be predictive of mortality, even after controlling for BDI,
particularly because individuals with diabetes have to adopt regimented control along with other characteristic type A behaviors, such as having to do many things at once, thinking of what they might need to do next, becoming less casual about things, and feeling ambitious (7). Type A behaviors may increase the efficiency in which an individual cares for their type 1 diabetes, therefore preventing complications and early mortality. However, depressive symptoms may outweigh the significance of type A behavior, as demonstrated in our analysis. Adopting type A behaviors in order to better care for a chronic disease like type 1 diabetes may also partially explain why type A behavior is protective in high-risk, as opposed to the general, populations. Those with greater type A behavior in the presence of a chronic disease may treat symptoms and suspected complications more seriously and intensively than those characterized as having less type A behavior.

Little research exists on depressive symptomatology and subsequent risk of mortality in type 1 diabetes. The FinnDiane Study Group concluded that in women, baseline antidepressant agent purchase (their surrogate marker for depression) was associated with an increased mortality risk over 9 years of follow-up (2.15 [1.34–3.45]) (15). Although this association was only seen in women, our results demonstrate a similar relationship. Those with increased depressive symptomatology were not only at increased mortality risk, but the protection offered by type A behavior disappeared with increased BDI. Depressive symptoms, therefore, appear to play a very important role in predicting mortality in type 1 diabetes.

Investigating the association between type A behavior and mortality by sex showed that the protective effects of type A behavior are only significant in men. However, these findings may be partially attributable to a lack of power to detect the relationship in women as fewer women had a high type A score. The relationship among men remained until BDI adjustment. We compared those with minimal to mild versus moderate to severe depressive symptoms and found only those with high type A behavior and less than moderate depressive symptoms were protected against mortality.

It has been previously noted that because type A behavior questionnaires can be interpreted as geared toward work or competitive behaviors, men may respond differently than women (2). In other words, men may feel it is more socially acceptable, expected, and fitting of their traditional role to declare themselves “very competitive,” “hard driving,” and “ambitious” whereas women may not feel the pressure to fulfill that stereotype. Therefore, our male participants’ responses

<table>
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<tr>
<th>Table 2—Baseline characteristics (1986–1988) by subsequent mortality</th>
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<tr>
<td><strong>Living (n = 378)</strong></td>
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<tr>
<td>Age</td>
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<tr>
<td>Sex, % (n), males</td>
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<tr>
<td>Duration</td>
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<tr>
<td>Education, % (n), above high school</td>
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<tr>
<td>Total energy expenditure/week (kcal)</td>
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<td>Total sports expenditure/week</td>
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<tr>
<td>Smoke ever, % (n), yes</td>
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<tr>
<td>BMI</td>
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<tr>
<td>HbA1c, % (mmol/mol)</td>
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<tr>
<td>Insulin dosage (total units/kg body weight)</td>
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<td>Bortner Rating Scale</td>
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<td>BDI</td>
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†Median (interquartile range).

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<tr>
<th>Table 3—Association between type A behavior and all-cause mortality in type 1 diabetes</th>
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<td><strong>Covariate</strong></td>
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<tr>
<td>Model 1a</td>
</tr>
<tr>
<td>Age</td>
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<td>Sex</td>
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<td>Model 2b</td>
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<td>Age</td>
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<td>Sex</td>
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<td>Ever smoker</td>
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<td>HbA1c</td>
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<td>Model 3c</td>
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<td>BDI</td>
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<td>Age</td>
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<td>Ever smoker</td>
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<td>HbA1c</td>
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<td>Model 4d</td>
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<td>BDI</td>
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<td>Age</td>
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<tr>
<td>Ever smoker</td>
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<tr>
<td>HbA1c</td>
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<tr>
<td>Bortner × BDI</td>
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</tbody>
</table>

Cox regression (n = 506; 128 events). HR (95% CI) for all-cause mortality per 1 unit change in Bortner score.

on the Bortner scale may differ compared with women due to social norms, especially in the 1980s when the questionnaire was completed. At that time, if women were homemakers, perhaps they felt they were not facing the daily demands of a career and therefore had less type A responses. This is consistent with our data, as we saw a statistically higher mean type A behavior score in men compared with women (191.1 vs. 185.2; P < 0.01). A study that also administered the Bortner scale in the 1980s found that participants with no or minimal obstructive CAD had higher type A scores compared with those with obstructive disease. After further analysis by sex, the effect was only significant in men, consistent with our findings (16).

We also examined the relationship between Bortner scores and CAD as it is a major contributor to death. In those free of CAD at baseline, type A behavior predicted CAD during 22 years of follow-up, although this was of borderline statistical significance, and this relationship was attenuated after adjustment for duration. Thus, we did not find that in our population, increased type A behavior was protective against CAD or indeed CAD death among those with CAD. Other factors not measured in our study may play a role, and further research is needed to determine which other covariates may offer protection against CAD. Our results were not as hypothesized; however, it should be noted that type A behavior was also not detrimental to the development of CAD, which supports previous research in other high-risk groups (2,3). Because type A behavior was not related to CAD development, we evaluated whether mortality was predicted by Bortner scores based on whether the primary cause of death was CAD or non-CAD related. We found a significant difference between these two groups, with type A behavior protecting against CAD-related death, and again when comparing CAD death to survivors only.

A previous study by Lloyd et al. (5) concluded that lower type A behavior scores were associated with an increased macrovascular disease risk; however, the current study is the first that we are aware of to demonstrate that type A behavior in type 1 diabetes is protective against CAD death, specifically. In a 10-year follow-up study of middle-aged, employed men, specific personality traits that would be considered type A did not predict CAD death (17). The literature examining the relationship between type A behavior and CAD death is limited. Our remarkable finding that type A behavior is specifically protective against CAD death merits further investigation.

Thus far, study findings examining type A behavior in type 1 diabetes are conflicting and often focused on surrogate outcomes (such as glycemic control and complications) due to short length of follow-up time. The results of studies examining type A behavior and glycemic control were mixed, with some suggesting no association (18–20), others a detrimental association (21,22), and another a protective association of specific type A behaviors (i.e., neuroticism) (23). However, the majority of these studies were conducted three decades ago with large amounts of bias, which may explain why mixed results were demonstrated. The majority of the previously published studies were conducted cross-sectionally, using very small sample sizes, and used univariate methods of analysis only. Those that used multivariable analyses only controlled for a few relevant covariates. Lloyd et al. (14) found that those type 1 diabetic participants with multiple complications reported less type A behavior than those without any complications (P < 0.05) (5). In a separate study, it was also determined that in men, lower type A behavior score was predictive of an increased WHR. Because type A behavior was not shown to be detrimental in type 1 diabetes, and protective against complications as a whole and WHR, our hypothesis was generated that with longer follow-up, higher type A behavior may be protective against mortality. Future research needs to take place to examine this relationship in other high-risk populations.

Based on the literature that high-risk groups are protected by their type A behavior, we investigated the relationship between the Bortner Rating Scale and CAD case-fatality rate. We hypothesized that in this very high-risk group of people with both type 1 diabetes and CAD, type A behavior would be even more protective, but a relationship was not found. This may be due to several reasons, one being that we may have had an insufficient sample size to find a statistically significant result (28 deaths/125 with CAD). Another reason may be that these participants were too unhealthy to benefit from type A behavior at all, being that they have both type 1 diabetes and a serious complication. Another explanation may be that the type A behaviors were initiated at too late a time in life, and that type 1 diabetes and CAD had already done too much physical damage for any protective effect to take place against mortality. Perhaps behavior type is also a trait and can therefore be modified. If so, this has great implications for care as we can support behavior change to improve self-management, improving the health of those living with diabetes.

There were several strengths and limitations of our study. As mentioned previously, our long follow-up time allowed for us to use mortality as our outcome, as opposed to a surrogate end point such as complication status. Additionally, this was the first study to investigate the relationship between type A behavior and mortality in type 1 diabetes, providing data where there currently are none. Limitations of our study include our small sample size for detecting incident CAD death among those with CAD, which may have led to null results. Another limitation is the possibility of residual confounding; however, we feel we included predictors that are essential for investigating mortality in type 1 diabetes. Furthermore, there were up to 22 years of follow-up time between measuring type A behavior and mortality and/or the onset of CAD; however, we attempted to control for this in the analysis through use of Cox proportional hazards models.

In conclusion, future research is needed to investigate the interaction between BDI and type A behavior, as the latter was only protective in those with low depressive symptomatology. Further research is also needed to explore the protective relationship between type A behavior and CAD death. Understanding these relationships is an important next step in exploring the effects of psychosocial factors on mortality in type 1 diabetes.

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C.E.F. wrote the manuscript and performed the data analysis. C.E.L. reviewed and edited the manuscript. T.C. and R.G.M. contributed to the data analysis and reviewed and edited the manuscript. T.J.O. researched data, contributed to the data analysis, and reviewed and
edited the manuscript. All authors read and approved the final version of the manuscript. T.J.O. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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