Hemodynamic Reactivity to Psychological Stress in Young Adults with a Family History of Stroke

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Abstract

Cardiovascular disease (CVD) is currently the leading cause of death among Europeans and Americans. While many risk factors for CVD have been identified, their role in the etiology of CVD is not fully understood. One such risk factor is a family history of CVD. In addition to a positive family history, Type D personality has been established as an independent risk factor for CVD. The purpose of this study was to examine the hemodynamic reactivity to laboratory stress in young adults with a positive family history of stroke and/or Type D personality, and to look at its interaction. Thirty-nine healthy young adults completed two laboratory stressor tasks. Cardiovascular measures were recorded throughout the experiment. The results showed that there were no main effects of family history on cardiovascular reactivity. There was a significant effect of Type D personality on total peripheral resistance (TPR) during the speech recovery period, but not on systolic blood pressure (SBP), diastolic blood pressure (DBP) or cardiac output (CO). The interaction effect between family history and NA was significant. Lack of significant main effect of family history on reactivity might be due to too few participants.

Keywords: cardiovascular disease; Type D personality; negative affect; social inhibition; positive family history

Útdráttur

Hjarta- og æðasjúkdómar eru helstu dánarosök á meðal Evrópu- og Ameríkubúa. Þrátt fyrir að margir áhættuþættir þessara sjúkdóma eru orðnir vel þekktir er ekki mikið vitað um orsök þessara þátta. Á meðal áhættuþátta er fjölskyldusaga um hjarta- og æðasjúkdóma. Auk fjölskyldusögu, þá er Týpa D persónuleiki áhættuþáttur fyrir hjarta- og æðasjúkdóma. Tilgangur þessarar rannsóknar er að skoða lífeðlisleg viðbrögð við streitu í heilbrigðu, ungu fólki sem hefur jákvæða fjölskyldusögu um heilablóðfall og hefur Týpu D persónuleika. Samvirkni á milli fjölskyldusögu og/eða Týpu D verður einnig skoðuð. Alls voru 39 þátttakendur sem luku við tvenns konar streituvekjandi verkefni. Mælingar á lífeðslilegum viðbrögðum áttu sér stað yfir allt rannsóknartímabilið. Niðurstöður leiddu í ljós að jákvæð fjölskyldusaga um heilablóðfall hafði ekki áhrif á lífeðlisleg viðbrögð þátttakanda. Týpa D persónuleiki hafði áhrf á TPR en ekki aðrar lífeðlislegar breytur; SBP, DBP eða CO. Samvirkni á milli fjölskyldusögu og *NA* var marktæk. Ástæða þess að ekki fengust marktæk áhrif fjölskyldusögu á lífeðslilega virkni gæti verið vegna fárra þátttakanda.

Lykilhugtök: hjarta- og æðasjúkdómar; Týpu D persónuleiki; neikvæð áhrif; félagsleg hömlun; jákvæð fjölskyldusaga

Hemodynamic Reactivity to Psychological Stress in Young Adults with a Family History of Stroke

Cardiovascular disease and a family history

Cardiovascular disease (CVD) is currently the leading cause of death in Westernized countries and nearly half (47%) of all deaths are from CVD (Nichols et al., 2012). Diseases counted as CVD are, for example, hypertension, coronary heart disease and stroke (Lloyd-Jones et al., 2008). Coronary heart diseases are the single most common cause of death and stroke is the second. CVD is the main cause of death before the age of 75 in Europe, but the risk for CVD increases with age. Over all, CVD are responsible for 52% of deaths in women and 42% of deaths in men (Nichols et al., 2012). While many risk factors for CVD have been identified, their role in the etiology of CVD is not fully understood. One such risk factor is a family history of CVD (Lloyd-Jones et al., 2008). Williams and colleagues (2001) combined data from two large population-based studies for a total of over 130,000 families to investigate the relationship between family history of CVD and future onset. They concluded that most cardiovascular events with early onset are seen in people with a positive family history of CVD. Furthermore, although only 11% of families reported a family history of stroke, 86% of early strokes occurred in these families (Williams et al., 2001). Studies have been showing that individuals with a positive family history have an exaggerated reactivity to laboratory tasks (Manuck, Proietti, Rader, & Polefrone, 1985). Through a meta-analysis it has been shown that individuals with a positive family history of hypertension tend to have greater diastolic blood pressure (DBP) response to acute stressors (Fredrikson & Matthews, 1990).

Previous literature on family history has some limitations. For example, most studies only assess parental history and do not include other relatives such as grandparents. Another limitation to most previous family history studies is that typically, only family history of hypertension is assessed, and not a broader range of cardiovascular disease, such

as stroke. Also, the studies have failed to confirm the relationship between family history and hemodynamic profile.

Type D personality

Another risk factor for CVD are some personality traits (Denollet, 1998). Type D personality has been related to worsening CVD and cardiovascular mortality in patient population (Kupper & Denollet, 2007), and has been investigated as an independent risk factor for heart disease (Grande, Romppel, & Barth, 2012). Type D, or so-called "distressed" personality, is characterized as a tendency to experience high levels of negative emotions across time and situations, called negative affectivity (NA), without the ability to express them properly, called social inhibition (SI) (Habra, Linden, Anderson, & Weinberg, 2003; L. Williams, O'Carroll, & O'Connor, 2009). The prevalence of Type D personality ranges between 21-38% in general population (Denollet, 2005; Williams et al., 2008), and there appears to be no gender difference (Williams et al., 2008). As said, Type D personality is an independent risk factor for CVD, not because of exaggerated reactivity, but rather blunted reactivity (Grande et al., 2012).

Reactivity hypothesis

The negative influence of psychological stress on CVD has been known for a long time. Acute stressors, such as laboratory stressors, can trigger cardiac events and worsen heart disease (Rosengren et al., 2004; Treiber et al., 2003). One model that has directed much of the research on stress in general is the reactivity hypothesis (Krantz & Manuck, 1984). Reactivity refers to changes in cardiovascular levels in response to an acute stressor and is considered to be a trait characteristic, which indicates how an individual responds to daily stressors (Kamarck & Lovallo, 2003). The hypothesis was developed after several studies demonstrated that young participants with normal blood pressure, but at risk for future development of hypertension, had an exaggerated blood pressure response to a

variety of laboratory challenges (Stephen B. Manuck, Kasprowicz, & Muldoon, 1990). A number of physiological changes can be observed during an acute stressor, such as increased heart rate, increased blood pressure and increases in cortisol level. Previous studies have found that exaggerated cardiovascular reactivity to a laboratory stressor is an independent risk factor for CVD (Treiber et al., 2003). A major criticism of the reactivity hypothesis is that it does not address the duration of stress, chronic stress, or prolonged activation after stress (Schwartz et al., 2003). The duration for recovery and the magnitude of reactivity might have equally important roles in the development of cardiovascular disease (Christenfeld, Glynn, & Gerin, 2000).

Blunted reactivity. Recent data suggest that blunted reactivity as well as exaggerated reactivity is associated with bad health outcomes (Phillips, 2011). Blunted reactivity refers to a cardiovascular response pattern, which is comparatively lower than typically seen during stress. It is unclear what determines the occurrence of blunting but one possibility is that it due behavioral factors, rather than biological (Phillips, Ginty, & Hughes, 2013). Some researchers have shown that blunted reactivity is associated with depression and obesity (Phillips, 2011). Blunted reactivity has been gaining more attention recently and especially in association with Type D personality (Howard, Hughes, & James, 2011; L. Williams et al., 2009). Studies have repeatedly shown that the distress personality is associated with bad CVD outcome (Denollet, 1998). Type D personality has shown to be significantly associated with worse disease outcome, even after controlling for traditional risk factors, symptoms of depression, anger, and anxiety. Studies on Type D personality have been showing that responses to stress are lower than normally is seen, so called blunted reactivity, but what makes it interesting is that Type D personality is a risk factor for CVD (Denollet, 1998; Grande et al., 2012; Kupper & Denollet, 2007).

Type D personality and hemodynamic profile.

Research that has been done in this area has looked at effects of Type D on hemodynamic profile that is the dynamic relationship between cardiac output (CO) and total peripheral resistance (TPR). CO provides information about the blood-volume ejecting by the heart per unit time. (Qim Y. Lee et al., 2013). TPR is a measure of vessel´s resistance (Lloyd-Jones et al., 2008). Together it regulates the blood pressure level. Changes in blood pressure can occur as a result of two different patterns of changes in CO and TPR. In the first pattern, an increase in CO is accompanied by an insufficient compensatory decrease in TPR, called myocardial response pattern, or, increase in TPR and an insufficient compensatory decrease in CO, called vascular response pattern (James, Gregg, Matyas, Hughes, & Howard, 2012). Decrease in blood pressure can be described in a similar way; where decrease in either CO or TPR is accompanied by smaller compensatory increase in the other variable. In the second patter, which is called mixed pattern, an increase (or decrease) in blood pressure is due to synergistic increases (or decreases) in both CO and TPR (James et al., 2012). Individuals that show vascular response to stress, rather than myocardial appear to be in more risk of CVD because of repeated or prolonged periods of increased vascular resistance (Gregg, Matyas, & James, 2002; Palatini & Julius, 2009). Studies in this area have been inconsistent and results have been mixed. In Howard et al. (2011) study there was reactivity difference between Type D and non-Type D where Type D individuals were showing less reactivity in both CO and TPR compared to non-Type D. Type D showed lower CO across time and higher TPR (Howard et al., 2011). This contradicts a study by Williams et al. (2009), where Type D males exhibited higher CO compared to non-Type D. In the two studies, Type D was associated with CO reactivity, although in opposite direction and for opposite sex. In Williams et al. (2009) study the effects were on male only, and on female only in Howard

et al. (2011) study. There was a different stressor task in the studies, which might help explain the difference in CO responses. Given that hemodynamic variables are particularly sensitive to task type, the fact that Type D personality predicted CO reactivity in a different way across the two studies might be explained by the qualitative differences in the two stressors (Howard et al., 2011; James et al., 2012). Also, research has shown that mental arithmetic, which was the stressor task in Howard et al. (2011) study, produces markedly different hemodynamic response profiles in males and females (Girdler, Turner, Sherwood, & Light, 1990). It is worth noting that there was no significant group effect of Type D on SBP or DBP (Howard et al., 2011; Williams et al., 2008).

The Type D literature has not yet reached a point where unresolved issues can be settled. As said before, Type D personality has been related to worsening CVD and especially to coronary heart disease (CHD), but little is known how it affects other types of CVD, e.g. stroke. The association between Type D and CO reactivity is inconsistent and further studies are needed.

Current study

The present study attempts to examine the association between Type D personality and laboratory measures of cardiovascular response, focusing on CO and TPR. The effects of positive family history of stroke on cardiovascular response will be examined.

Hypothesis:

1) Participants with a family history of cardiovascular disease will have greater reactivity to stress tasks than participants with a negative family history.
2) Participants high on Type D personality will have blunted reactivity to stress task compared to participants low on Type D.
3) The interaction effect of family history and Type D personality will be explored.

Method

Participants

Participants were 39, young healthy adults of both sexes (12 men and 27 women). The mean age was 27,4 years old. The researchers attended classes at Reykjavík University and approached students requesting their participation, which then spread by word of mouth to the researchers benefit. Participants were excluded if they reported a personal diagnosis of cardiovascular diseases or arrhythmias or were currently taking any medication that might affect the cardiovascular system. Further, only participants who knew the stroke history of biological parents and grandparents were included. Participation was voluntary and participants were free to withdraw at any time. All participants signed a consent form prior to participation.

Materials and apparatus

Family history questionnaire. Family history of cardiovascular diseases was assessed prior to the study. Participants were asked whether their parents or grandparents had ever been diagnosed with the following conditions: coronary heart disease, hypertension, high cholesterol, stroke, or diabetes. They were asked to further specify whether coronary heart disease occurred as early (before age 55) or late (after 55) onset, and whether diabetes was Type I or Type II. The family history questionnaire is presented in Appendix A.

Type D personality questionnaire. Participants underwent psychometric testing to establish Type D status using the 14-item Type D scale. The DS14 consist of two 7-item scales measuring negative affect (NA) and social inhibition (SI). Participants who score highly on both NA and SI using a cut-off point of ≥ 10 are classified as having a Type D personality. This is consistent with previous studies (L. Williams et al., 2009). The Type D questionnaire is presented in Appendix B.

Health questionnaire. Information was collected regarding biological and behavioral factors that might affect the cardiovascular system. This included questions on previous history of heart diseases, arrhythmias, and high blood pressure, as well as smoking, caffeine consumption, and exercise. The health questionnaire is presented in Appendix C.

Cardiovascular reactivity tasks

The cardiovascular reactivity tasks that were selected are mental arithmetic task, which Howard et al. (2011) used in their study, and a speech task, which was not used either in Howard et al. (2011) study or Williams et al. (2009) study. Both task are "active" task, and tend to stimulate myocardial responses where an increase in CO is accompanied by an insufficient compensatory decrease in TPR (James et al., 2012).

Mental arithmetic task. Participants were instructed to subtract the number seven from a four-digit number for three minutes. They were told to do this as quickly and accurately as possible, and to say each number aloud. If participants lost their place, another four-digit number was assigned to them. This task typically elicits large parasympathetic withdrawals and sympathetic activation (Berntson et al., 1994).

Speech task. Participants were asked to prepare and give a speech. Instructions were played over an audio speaker describing a scenario where the participant was pulled over by a police officer for an unfair traffic violation and is now in traffic court defending themselves. They were given a study card with several points to make during their speech and three minutes to quietly prepare. After the preparation period, the experimenter informed them that they would be videotaped and that the quality of their performance would be evaluated. They were then given three minutes to deliver their speech. This task typically elicits large parasympathetic withdrawals and sympathetic activation (Berntson et al., 1994).

Hemodynamic reactivity recording apparatus

Finometer Pro. Beat-to-beat blood pressure and HR were measured non-invasively using a Finometer hemodynamic cardiovascular monitor. The Finometer is based on the volume-clamp method first developed by Peňaz 1973 (as cited Howard et al., 2011). An appropriate-sized finger cuff is attached to the participant´s middle finger, which inflates to keep the arterial walls at a set diameter. Photo-plethysmograph is built-in into the finger cuff. It detects changes in the diameter of the arterial wall.

Design

The current study´s design is mixed. Firstly, participants were separated in two groups, positive or negative family history of stroke. The physiological responses to acute stressors were compared between these two groups. Secondly, the participants were divided in two groups depending on Type D personality status. These physiological responses were compared as well. To investigate the interaction effect of family history of stroke and Type D personality, participants were placed in one of four groups. Instead of using Type D personality as a requirement, we used negative affect, which is a subscale of Type D personality. The groups are as follows; NA and positive family history; NA and negative family history; non-NA and positive family history; non-NA and negative family history.

Procedure

All the procedures were approved by the Institutional Research Ethics Committee. Before arriving at the laboratory, the participant had completed the questionnaire assessing their family history of cardiovascular disease. Upon arrival, participants were greeted by the researchers and seated in a comfortable chair. The participant was asked to carefully read the informed consent form and sign it. The participant was told about its right to withdrawal any time. Participants completed the health questionnaire prior to the laboratory tasks and participant´s weight (kg) and height (cm) were measured. Next, the Finometer

cuff was attached to the participant´s middle finger of their non-dominant hand, as was the arm cuff. A Planet Earth video, which is a nature documentary, was used to facilitate relaxation and the establishment of cardiovascular baselines.

Following this 10 minutes relaxation period, participants were given instructions about the first task before completing a pre-task appraisal questionnaire. The stressor tasks were administered in counterbalanced order. After completing each stressor task, the participant was instructed to sit quietly for a five-minute recovery period before receiving a post-task appraisal questionnaire. Cardiovascular parameters were measured non-invasively using the Finometer throughout the procedure. The participant was thanked for their participation.

Statistical Analyses

Cardiovascular parameters were measured throughout the procedure. First, we calculated an average measure for every 60 seconds. Next, we calculated a four minutes baseline period and two, four minutes recovery period. The same was conducted with the stressor task, except it was a three minutes period. Three minutes for the mental arithmetic task, three minutes for the speech preparation and three minutes for the speech. This was done to CO, TPR, HR, DBP and SBP.

For Hypothesis 1, a series of one-way ANOVA´s were conducted with family history (FH+, FH-) as the between subjects.

For Hypothesis 2, a series of one-way ANOVA´s were conducted with Type D personality as the between subjects factor.

For Hypothesis 3, a series of two-way ANOVA´s were conducted for each task period with negative affect and family history as the between subjects.

For all Hypothesis, SBP, DBP, CO and TPR were used as the dependent variables, and age and BMI were entered as covariates. All analyses were run using a significance level of .05.

Results

Descriptive statistics

Family history. Family history was categorized into parental or grandparental history of stroke (n = 16). Participants were classified as positive family history if they responded "yes" for stroke. Participants were classified as negative family history if they responded "no" for stroke. If the responded "I don't know" for stroke, they were classified as "unknown" status and consequently dropped from further analysis.

Type D personality. Participants were classified as Type D personality if they scored ≥ 10 in both NA and SI (n = 9). See Table 1 for the number of participants in each category.

Table 1
Family history (FH) and Type D groups

FH group	Type D	non-Type D
FH+	5	11
FH-	4	19

Reactivity. The reactivity was assessed by subtracting the measures from the stressor task from baseline measures, which were assessed through resting period at the beginning of the experiment. See Table 2 for baseline measures.

Table 2
Baseline measures of SBP, DBP, CO and TPR

	n	Min	Max	Mean	Std. Deviation
SBP	39.00	101.00	163.50	123.38	12.57
DBP	39.00	58.00	90.00	71.79	8.08
CO	39.00	4.00	10.00	6.89	1.58
TPR	39.00	0.28	1.01	0.97	0.12

Hypothesis 1: Family history.

In order to test for main effects of family history on reactivity and recovery a series of one-way ANOVA´s were conducted with FH as the between subjects factor and SBP, DBP, CO and TPR as the dependent variables. Age and BMI were used as covariates for all analyses.

There were no main effects of family history on any of the physiological variables during any of the task reactivity or recovery periods (*p´*s > .10).

Hypothesis 2: Type D

In order to test the main effects of Type D personality on reactivity and recovery a series of one-way ANOVA´s were conducted with Type D as the between subjects factor for SBP, DBP, CO and TPR as the dependent variables. Age and BMI were used as covariates for all analyses.

There were no main effects of Type D personality on SBP, DBP, or CO (*p´*s > .10). During the recovery period following the speech task there was a significant effect of Type D personality on TPR, $F(1, 34) = 4.38$, $p=.04$. Participants with Type D personality had significantly higher TPR during the recovery period compared to their baseline states ($M = 0.035$, $SE = 0.012$) than participants without Type D personality ($M = 0.005$, $SE = 0.007$). Additionally there was a marginally significant effect of Type D personality on TPR during the math recovery period, $F(1, 34) = 2.97$, $p=.09$. Participants with Type D personality had slightly higher TPR during the recovery period compared to their baseline states ($M = 0.02$, $SE = 0.009$) than participants without Type D personality ($M = 0.002$, $SE = 0.005$).

Hypothesis 3: Interaction between family history and Type D

Upon examining the data we found that there was not sufficient power to conduct a two-way ANOVA between Type D personality and family history since there were only five participants with both Type D personality and a positive family history and only four

participants with Type D personality and a negative family history. As such, we decided to examine the interactive effects using the negative affect subscale of the Type D questionnaire in replacement of Type D personality. We conducted a series of two-way ANOVAs for each task period with negative affect and family history as the between subjects factors and SBP, DBP, CO and TPR as the dependent variables. Age and BMI were used as covariate.

Systolic blood pressure. There was a significant interaction effect between family history and NA on SBP reactivity during the speech preparation $F(1, 32) = 9.98$, $p=.003$, speech delivery $F(1,32) = 5.51$, $p=.025$, and a math task $F(1, 32) = 5.75$, $p=.023$. There was a significant simple effect of negative affect on reactivity, but only for participants who also had a positive family history of stroke (prep: $F(1, 32) = 9.64$, $p=.004$; speech: $F(1, 32) = 4.83$, $p=.035$; math: $F(1, 32) = 3.99$, $p=.054$). Participants who scored high on negative affect had significantly greater SBP reactivity to the preparation, speech, and math task (see Figure 1).

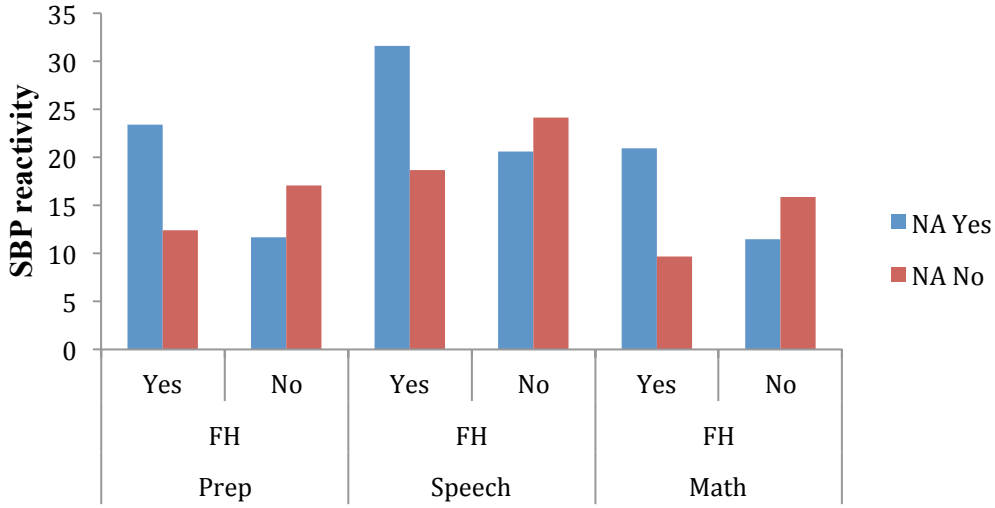

Figure 1. SBP reactivity to the preparation, speech, and math task.

Diastolic blood pressure. There was a marginal interaction effect between family history and NA on DBP reactivity during the speech preparation task $F(1, 32) = 3.12$, $p=.087$. A simple effects analysis showed that for participants with a positive family

history, those who scored high on negative affect had greater reactivity (M = 12.30, SE = 5.53) than those who scored low on negative affect (M = 7.89, SE = 4.70), $F(1, 32) = 4.35$, p=.045.

Cardiac output. There was a significant interaction effect between family history and NA on CO reactivity during the speech preparation $F(1, 32) = 5.58$, p=.024 and speech delivery tasks $F(1, 32) = 5.13$, p=.03, and a marginally significant effect for CO reactivity to the math task $F(1, 32) = 3.14$, p=.086. There was a significant simple effect of negative affect on reactivity, but only for participants who also had a positive family history of stroke, $F(1, 32) = 4.07$, p=.052. Participants with a positive family history who also scored high on negative affect had greater CO reactivity to the task compared to participants low on negative affect (see Figure 2). Interestingly, the opposite effect was seen for the speech delivery and math task; a simple effects analysis showed a significant effect of NA on CO reactivity to the speech task, but only for participants who did not have a family history, $F(1, 32) = 4.82$, p=.036. A similar trend was seen for the math task, $F(1, 32) = 3.34$, p=.077.

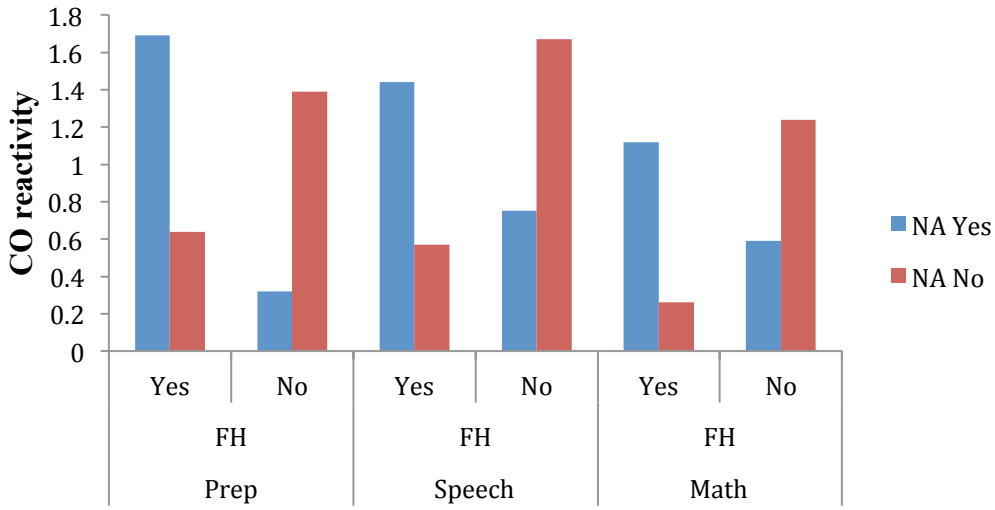

Figure 2. CO reactivity to the preparation, speech, and math task.

Total Peripheral Resistance. There was a significant interaction effect between family history and NA on TPR reactivity during the math task, $F(1, 32) = 6.67$, p=.015.

There was a significant simple effect of negative affect on reactivity, but only for participants who also had a positive family history of stroke, $F(1, 32) = 5.33$, $p=.028$. Participants with a positive family history who also scored high on negative affect had greater TPR reactivity to the task ($M = 12.30$, $SE = 5.53$) compared to participants low on negative affect ($M = 12.30$, $SE = 5.53$).

Discussion

Previous research has shown that exaggerated reactivity is predictive of future development of cardiovascular disease (Manuck et al., 1985; Treiber et al., 2003). One of the aims of the current study was to examine the effect of positive family history of stroke on cardiovascular reactivity to psychological stress. There was no significant difference in baseline measures of SBP, DBP, CO, or TPR between participants with a positive and negative family history of stroke. We hypothesized that participants with a positive family history would exhibit an exaggerated response to the stress task compared to participants with a negative family history. Contrary to expectations, there was no significant effect of family history of stroke on parasympathetic or sympathetic reactivity to any of the tasks. Therefore, our hypothesis was not supported, which might be due to few participants. Previous studies have shown that having a positive family history of CVD results in exaggerated reactivity to laboratory tasks (Manuck et al., 1985). A meta-analysis found that DBP reactivity to stress is the most consistent reactivity measure in participants with a positive family history of hypertension (Fredrikson & Matthews, 1990). Although, no other studies have been conducted examining autonomic reactivity to an acute stressor in participants with a family history of stroke, previous studies have shown a relationship between reactivity and a family history of other CVD (Fredrikson & Matthews, 1990; Williams et al., 2001).

The present study attempted to overcome methodological limitation of previous family history studies in several ways. We assessed not only parental history of CVD, but also grandparents. Also, we got information about family history of stroke, not hypertension as previous research has focused on. In the end, we looked at reactivity in hemodynamic profile, but previous research has failed to confirm the relationship between family history and hemodynamic profile. However, it didn't result any significant.

The second aim of current study was to establish if Type D individuals exhibit a differential pattern of cardiovascular reactivity to stress compared to non-Type D´s. Previous research has shown that Type D personality affects cardiovascular response to psychological stress (Grande et al., 2012; Kupper & Denollet, 2007). We hypothesized that Type D individuals would have blunted reactivity to the stress task compared to participants low on Type D. Our hypothesis was not supported. However, no differences were found between Type D´s and non-Type D´s on measures of SBP, DBP and CO, there was a significant effect of Type D on TPR during the recovery period following the speech task. Also, there was a marginally significant effect of Type D on TPR during the math recovery period. Type D individuals had higher TPR during the recovery period compared to their baseline states than non-Type D participants. This is in contrast with previous research (Howard et al., 2011), which also identified higher TPR among Type D individuals. However, the current study did not demonstrate a relationship between Type D personality and CO, like previous research demonstrated (Howard et al., 2011; Williams et al., 2009). This kind of reactivity is called vascular response pattern. Those who show this kind of physiological response to stress, appear to be in more risk of CVD because of repeated or prolonged periods of increased vascular resistance (Gregg et al., 2002; Palatini & Julius, 2009).

The fact that Type D didn't effect actual blood pressure level, only the physiological determinants of blood pressure is consistent with previous research (Howard et al., 2011; Williams et al., 2009). In Habra et al. (2003) study, they looked at NA and SI as separated variables, SI was then associated with greater SBP reactivity and DBP reactivity to acute stress, but only in for men. NA was not associated with blood pressure reactivity rather heart rate reactivity. The current study failed to demonstrate any relationship between NA and SI on SBP, DBP and other physiological variables.

The third aim of current study was to explore at the interaction effect of family history and Type D personality. To our knowledge, no other studies have been conducted examining the interaction effect of family history and Type D personality. Because of insufficient power to conduct a two-way ANOVA between Type D personality and a family history, we decided to examine the interactive effects using the NA subscale of the Type D questionnaire in replacement of Type D personality. There was a significant interaction effect between family history and NA on SBP, CO and TPR.

The effects on SBP reactivity were seen during the speech preparation, speech delivery and math task. Those who scored high on NA had greater reactivity compared to those who were low in NA, but only if they had a positive family history of stroke. The same pattern was seen in CO reactivity to speech preparation. However, the effects were in opposite direction in speech preparation and in the math task. Those who were low in NA had a greater CO reactivity, but only if they didn't have a positive family history of stroke. This is interesting and was not expected. The effects on TPR reactivity were seen during the math task. Those who scored high on NA had greater reactivity, compared to those who were low on NA, but only if they had positive family history of stroke.

As has been known for a long time, a positive family history of CVD results in exaggerate reactivity to laboratory stressor (Fredrikson & Matthews, 1990; Manuck et al.,

1985) which is opposite with the Type D personality. Previous researches on Type D personality demonstrate that responses to stress are lower than typically is seen (e.g. Grande et al., 2012). When the two components of Type D personality are examined separately it can be seen that individuals high on SI response with greater blood pressure reactivity (Habra et al., 2003). No such association has been found between NA and blood pressure. However, individuals high on NA have responded with greater heart rate reactivity to laboratory stressors (Habra et al., 2003). Further studies are needed to demonstrate the relationship between NA and SI, and cardiovascular reactivity. The interaction effect between family history and Type D personality require further examination, as well.

Limitations

Several limitations of this study warrant further discussion. First, the generalizability of the current findings to a more at-risk population is limited by the fact that the sample consisted of young healthy adults. Second, the experiment was conducted in a laboratory, which may not be representative of stress experienced during daily life. Ambulatory blood pressure monitoring would be a logical next step in determining whether the relationship observed in current study still hold as individuals face daily stressors. Also, the sample size may have been too small to detect true differences in the effect of Type D personality and family history of stroke on reactivity. Finally, we failed to separate the components of Type D personality, NA and SI, and look at how it affects cardiovascular reactivity.

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Appendices

Appendix A

Vinsamlegast svarið þessum spurningum um líffræðilega móður þína:

1. Hvað er **móðir** þín gömul? _____ ☐ Ég veit ekki ☐ Látin
2. Hefur **móðir** þín verið greind með eftirfarandi sjúkdóma:

a.	Háþrýsting eða hár blóðþrýstingur?	☐ Já	☐ Nei	☐ Ég veit ekki
b.	Hátt kólesteról?	☐ Já	☐ Nei	☐ Ég veit ekki
c.	Týpu eitt (insulínháð) sykursýki?	☐ Já	☐ Nei	☐ Ég veit ekki
d.	Týpu tvö (insulínóháð) sykursýki?	☐ Já	☐ Nei	☐ Ég veit ekki
e.	Heilablóðfall eða slag?	☐ Já	☐ Nei	☐ Ég veit ekki
f.	Hjartaáfall?	☐ Já	☐ Nei	☐ Ég veit ekki
g.	Kransæðasjúkdóm?	☐ Já	☐ Nei	☐ Ég veit ekki
h.	Hjartabilun?	☐ Já	☐ Nei	☐ Ég veit ekki
i.	Aðra hjarta- eða æðasjúkdóma?	☐ Já	☐ Nei	☐ Ég veit ekki

3. Ef þú svaraðir "já" við spurningar **e**, **f**, **g**, eða **h**, átti atburðurinn sér stað *fyrir* 55 ára aldur?
   ☐ Já ☐ Nei ☐ Ég veit ekki
4. Ef þú svaraðir "já" við spurningu **e**, veistu hvernig heilablóðfall átti sér stað?
   ☐ Heilablæðing ☐ Heiladrep ☐ Ég veit ekki

Vinsamlegast svarið þessum spurningum um líffræðilegan föður þinn:

5. Hvað er **faðir** þinn gamall? _____ ☐ Ég veit ekki ☐ Látinn
6. Hefur **faðir** þinn verið greindur með eftirfarandi sjúkdóma:

a.	Háþrýsting eða hár blóðþrýstingur?	☐ Já	☐ Nei	☐ Ég veit ekki
b.	Hátt kólesteról?	☐ Já	☐ Nei	☐ Ég veit ekki
c.	Týpu eitt (insulínháð) sykursýki?	☐ Já	☐ Nei	☐ Ég veit ekki
d.	Týpu tvö (insulínóháð) sykursýki?	☐ Já	☐ Nei	☐ Ég veit ekki
e.	Heilablóðfall eða slag?	☐ Já	☐ Nei	☐ Ég veit ekki
f.	Hjartaáfall?	☐ Já	☐ Nei	☐ Ég veit ekki
g.	Kransæðasjúkdóm?	☐ Já	☐ Nei	☐ Ég veit ekki
h.	Hjartabilun?	☐ Já	☐ Nei	☐ Ég veit ekki
i.	Aðra hjarta- eða æðasjúkdóma?	☐ Já	☐ Nei	☐ Ég veit ekki

7. Ef þú svaraðir "já" við spurningar **e, f, g, eða h**, átti atburðurinn sér stað *fyrir* 55 ára aldur?

☐ Já ☐ Nei ☐ Ég veit ekki

8. Ef þú svaraðir "já" við spurningu **e**, veistu hvernig heilablóðfall átti sér stað?
   ☐ Heilablæðing ☐ Heiladrep ☐ Ég veit ekki

Vinsamlegast svarið þessum spurningum um líffræðilega ömmu þína í móðurætt:

9. Hvað er **amma** þín gömul? _____ ☐ Ég veit ekki ☐ Látin

10. Hefur **amma** þín verið greind með eftirfarandi sjúkdóma:

a.	Háþrýsting eða hár blóðþrýstingur?	☐ Já	☐ Nei	☐ Ég veit ekki
b.	Hátt kólesteról?	☐ Já	☐ Nei	☐ Ég veit ekki
c.	Týpu eitt (insulínháð) sykursýki?	☐ Já	☐ Nei	☐ Ég veit ekki
d.	Týpu tvö (insulínóháð) sykursýki?	☐ Já	☐ Nei	☐ Ég veit ekki
e.	Heilablóðfall eða slag?	☐ Já	☐ Nei	☐ Ég veit ekki
f.	Hjartaáfall?	☐ Já	☐ Nei	☐ Ég veit ekki
g.	Kransæðasjúkdóm?	☐ Já	☐ Nei	☐ Ég veit ekki
h.	Hjartabilun?	☐ Já	☐ Nei	☐ Ég veit ekki
i.	Aðra hjarta- eða æðasjúkdóma?	☐ Já	☐ Nei	☐ Ég veit ekki

11. Ef þú svaraðir "já" við spurningar **e**, **f**, **g**, eða **h**, átti atburðurinn sér stað *fyrir* 55 ára aldur?
   ☐ Já ☐ Nei ☐ Ég veit ekki

12. Ef þú svaraðir "já" við spurningu **e**, veistu hvernig heilablóðfall átti sér stað?
   ☐ Heilablæðing ☐ Heiladrep ☐ Ég veit ekki

Vinsamlegast svarið þessum spurningum um líffræðilegan afa þinn í móðurætt:

13. Hvað er **afi** þinn gamall? _____ ☐ Ég veit ekki ☐ Látinn

14. Hefur **afi** þinn verið greindur með eftirfarandi sjúkdóma:

a.	Háþrýsting eða hár blóðþrýstingur?	☐ Já	☐ Nei	☐ Ég veit ekki
b.	Hátt kólesteról?	☐ Já	☐ Nei	☐ Ég veit ekki
c.	Týpu eitt (insulínháð) sykursýki?	☐ Já	☐ Nei	☐ Ég veit ekki
d.	Týpu tvö (insulínóháð) sykursýki?	☐ Já	☐ Nei	☐ Ég veit ekki
e.	Heilablóðfall eða slag?	☐ Já	☐ Nei	☐ Ég veit ekki
f.	Hjartaáfall?	☐ Já	☐ Nei	☐ Ég veit ekki
g.	Kransæðasjúkdóm?	☐ Já	☐ Nei	☐ Ég veit ekki
h.	Hjartabilun?	☐ Já	☐ Nei	☐ Ég veit ekki
i.	Aðra hjarta- eða æðasjúkdóma?	☐ Já	☐ Nei	☐ Ég veit ekki

15. Ef þú svaraðir "já" við spurningar **e, f, g, eða h**, átti atburðurinn sér stað *fyrir* 55 ára aldur?
☐ Já ☐ Nei ☐ Ég veit ekki

16. Ef þú svaraðir "já" við spurningu **e**, veistu hvernig heilablóðfall átti sér stað?
☐ Heilablæðing ☐ Heiladrep ☐ Ég veit ekki

Vinsamlegast svarið þessum spurningum um líffræðilega ömmu þína í föðurætt:

17. Hvað er **amma** þín gömul? _____ ☐ Ég veit ekki ☐ Látin

18. Hefur **amma** þín verið greind með eftirfarandi sjúkdóma:

a.	Háþrýsting eða hár blóðþrýstingur?	☐ Já	☐ Nei	☐ Ég veit ekki
b.	Hátt kólesteról?	☐ Já	☐ Nei	☐ Ég veit ekki
c.	Týpu eitt (insulínháð) sykursýki?	☐ Já	☐ Nei	☐ Ég veit ekki
d.	Týpu tvö (insulínóháð) sykursýki?	☐ Já	☐ Nei	☐ Ég veit ekki
e.	Heilablóðfall eða slag?	☐ Já	☐ Nei	☐ Ég veit ekki
f.	Hjartaáfall?	☐ Já	☐ Nei	☐ Ég veit ekki
g.	Kransæðasjúkdóm?	☐ Já	☐ Nei	☐ Ég veit ekki
h.	Hjartabilun?	☐ Já	☐ Nei	☐ Ég veit ekki
i.	Aðra hjarta- eða æðasjúkdóma?	☐ Já	☐ Nei	☐ Ég veit ekki

19. Ef þú svaraðir "já" við spurningar **e**, **f**, **g**, eða **h**, átti atburðurinn sér stað *fyrir* 55 ára aldur?
☐ Já ☐ Nei ☐ Ég veit ekki

20. Ef þú svaraðir "já" við spurningu **e**, veistu hvernig heilablóðfall átti sér stað?
☐ Heilablæðing ☐ Heiladrep ☐ Ég veit ekki

Vinsamlegast svarið þessum spurningum um líffræðilegan afa þinn í föðurætt:

21. Hvað er **afi** þinn gamall? _____ ☐ Ég veit ekki ☐ Látinn

22. Hefur **afi** þinn verið greindur með eftirfarandi sjúkdóma:

a.	Háþrýsting eða hár blóðþrýstingur?	☐ Já	☐ Nei	☐ Ég veit ekki
b.	Hátt kólesteról?	☐ Já	☐ Nei	☐ Ég veit ekki
c.	Týpu eitt (insulínháð) sykursýki?	☐ Já	☐ Nei	☐ Ég veit ekki
d.	Týpu tvö (insulínóháð) sykursýki?	☐ Já	☐ Nei	☐ Ég veit ekki
e.	Heilablóðfall eða slag?	☐ Já	☐ Nei	☐ Ég veit ekki
f.	Hjartaáfall?	☐ Já	☐ Nei	☐ Ég veit ekki
g.	Kransæðasjúkdóm?	☐ Já	☐ Nei	☐ Ég veit ekki
h.	Hjartabilun?	☐ Já	☐ Nei	☐ Ég veit ekki
i.	Aðra hjarta- eða æðasjúkdóma?	☐ Já	☐ Nei	☐ Ég veit ekki

23. Ef þú svaraðir “já” við spurningar **e, f, g, eða h**, átti atburðurinn sér stað *fyrir* 55 ára aldur?

☐ Já ☐ Nei ☐ Ég veit ekki

24. Ef þú svaraðir “já” við spurningu **e**, veistu hvernig heilablóðfall átti sér stað?

☐ Heilablæðing ☐ Heiladrep ☐ Ég veit ekki

Appendix B

Heilsutengdar Spurningar

1. Aldur: ___
2. Hefur þú einhvern tíman verið greind/greindur með eftirfarandi sjúkdóma:

Hjartasjúkdóma	Háþrýsting (of háan blóðþrýsting	Heilablóðfall
Hátt kólesteról	Hjartsláttartruflanir	Sykursýki

3. Vinsamlegast skráið öll lyf sem þú ert að taka, bæði lyfseðlisskyld og ekki. Vertu viss um að taka fram öll lyf sem þú hefur tekið á seinustu 48 klukkutímum, þó svo að það er lyf sem þú tekur ekki vanalega (t.d. sýklalyf eða verkjatöflur).

_____

_____

_____

_____

_____

4. Klukkan hvað fékkstu seinast að borða? _____
   a. Hvað borðaðir þú?

      _____
5. Drekkur þú drykki með koffín? □Já □Nei
   a. Ef þú svaraðir já, hvenær fékkstu seinast drykk með koffíni? Klukkan:

      _____
   b. Hversu marga drykki með koffíni hefur þú fengið í dag? _____
6. Reykir þú sígarettur? □Já □ Nei
   a. Ef þú svaraðir já, hvenær reyktirðu seinast? Klukkan: _____
   b. Hversu margar sígarettur hefur þú reykt í dag? _____
   c. Hversu margar sígarettur á dag reykir þú venjulega? _____
7. Hvenær stundaðir þú seinast líkamsrækt? Þ.e.a.s. hreyfing sem hækkaði hjartsláttinn hjá þér í 30 mínútur eða lengur.

   Dagsetning: _____ Mínútur: _____

   Tegund hreyfingar: _____

Appendix C

Dagsetning: _____ **Rannsóknarnúmer: _____**

DS[14] *spurningalisti*

Hér að neðan eru nokkrar fullyrðingar sem fólk notar stundum til að lýsa sjálfu sér. Við hverja fullyrðingu skaltu setja **hring** utan um þá **tölu** sem á best á við þig. Það eru engin rétt eða röng svör, það eina sem skiptir máli er þín eigin skoðun.

0 = RANGT 1 = FREKAR RANGT 2 = HVORUGT 3 = FREKAR RÉTT 4 = RÉTT

1. Ég á auðvelt með að ná sambandi við fólk sem ég hitti.....	0	1	2	3	4
2. Ég geri oft mikið úr smávægilegum hlutum.....	0	1	2	3	4
3. Ég spjalla oft við ókunnuga	0	1	2	3	4
4. Ég er oft vansæl/vansæll	0	1	2	3	4
5. Ég er oft pirruð/pirraður.....	0	1	2	3	4
6. Mér finnst ég oft vera þvinguð/þvingaður í samskiptum við annað fólk.....	0	1	2	3	4
7. Ég er almennt svartsýn/svartsýnn	0	1	2	3	4
8. Ég á erfitt með að hefja samræður við annað fólk	0	1	2	3	4
9. Ég er oft í slæmu skapi.....	0	1	2	3	4
10. Ég er lokuð persóna.....	0	1	2	3	4
11. Ég vil helst halda fólki í ákveðinni fjarlægð.....	0	1	2	3	4
12. Ég hef oft áhyggjur af einhverju.....	0	1	2	3	4
13. Ég er oft mjög niðurdregin/niðurdreginn.....	0	1	2	3	4
14. Í félagsskap veit ég ekki um hvað ég á að tala.....	0	1	2	3	4

Appendix D

REYKJAVÍK UNIVERSITY
HÁSKÓLINN Í REYKJAVÍK

Lífeðlisleg viðbrögð streitu hjá nemendum með fjölskyldusögu af heilablóðfalli.

Þér er boðið að taka þátt í rannsókn. Áður en þú ákveður að taka þátt, er mikilvægt að þú áttir þig á hver ástæða rannsóknarinnar er og hvað í henna fellst. Þetta upplýsingablað segir þér allt um tilgang rannsóknarinnar, áhættu og ávinning. Ef þú ákveður að taka þátt verður þú beðinn um að skrifa undir upplýst samþykki. Ef það er eitthvað sem er óljóst, þá er þér velkomið að hafa samband. Taktu þér eins mikinn tíma og þú vilt til þess að lesa yfir þessar upplýsingar. Þú ættir aðeins að samþykkja þátttöku í þessari rannsókn þegar þú veist að þú skilur hvers er ætlast af þér og þegar þú hefur haft nægan tíma til að ákveða þig.

Tilgangur rannsóknarinnar
Tilgangur rannsóknarinnar er að kanna lífeðlisleg viðbrögð nemenda með fjölskyldusögu af heilablóðfalli. Þér ásamt u.þ.b. 60 öðrum er boðið að taka þátt í rannsókninni.

Hvað felur þátttakan í sér?
Rannsóknin felur í sér að þú mætir á tilraunarstofu okkar. Við þig verður tengt tæki sem mælir hjartslátt, blóðþrýsting og fleira. Lögð verða fyrir þig tvö, stutt verkefni. Rannsóknin tekur ca. 60 mínútur.

Þarf ég að taka þátt?
Það er alfarið undir þér komið að ákveða hvort þú viljir taka þátt. Ef þú ákveður að taka þátt, verður þú beðin(n) um að skrifa undir upplýst samþykki. Ef þú ákveður að taka þátt er þér frjáls að hætta þátttöku hvenær sem, án þess að gefa upp ástæðu fyrir því. Sú ákvörðun að hætta á meðan rannsókninni stendur eða ef þú ákveður að taka ekki þátt, mun ekki hafa neinar afleiðingar í för með sér.

Hvað felur það í sér fyrir mig ef ég tek þátt?
Það sem felst í þátttöku þinni er að mæta í eitt skipti á tilraunarstofu sem staðsett er í Háskólanum í Reykjavík. Mælitæki sem mælir hjartslátt, blóðþrýsting og fleira verður tengt við þig. Lögð verða fyrir þig tvenns konar verkefni sem þú verður beðin/n um að leysa eftir bestu getu. Þú verður einnig beðinn um að svara spurningalistum. Að því loknu er þátttöku þinni í rannsókn þassari lokið.

Hvað mun þátttaka mín standa lengi yfir?
Rannsóknin tekur um það bil 60 mínútur.

Hvaða ávinningur felst í þátttöku minni?
Það er í raun sú upplifun að fá að taka þátt í alvöru rannsókn. Vísindin öðlast ávinning þar sem við lærum meira um lífeðlisleg viðbrögð við streitu en enginn beinn ávinningur er fyrir þátttakanda rannsóknarinnar.

Hverjir eru mögulegir kostir og ókostir við þátttöku mina í rannsókninni?
Það felst engin áhætta í þátttökunni. Ef svo kemur upp að þú upplifir óþægindi þá er þér velkomið að hætta þátttöku hvenær sem er.

Hvað gerist í lok rannsóknarinnar?
Þegar allir þátttakendur hafa verið rannsakaðir (sem ætti að vera eftir 2 mánuðum frá þátttöku þinni), munt þú fá yfirlit yfir helstu niðurstöður, ef þú óskar eftir því. Þar munu helstu niðurstöður koma fram.

Hvað gerist ef ég skipti um skoðun á meðan á rannsókninni stendur?
Þér er frjálst að skipta um skoðun hvenær sem er, án afleiðinga.

Við hvern hef ég samband ef ég hef frekari spurningar?
Ef þú hefur frekari spurningar er þér velkomið að hafa samband við Fjólu Huld Sigurðardóttur í tölvupósti: fjolahs11@gmail.com. Einnig er hægt að hafa samband við ábyrgðarmann rannsóknarinnar sem er Jack James í tölvupóst jack@ru.is. Ef þú hefur einhverjar áhyggjur varðandi þessa rannsókn og vilt hafa samband við einhvern í trúnaði, getur þú haft samband við: **Sálfræðideild Háskólans í Reykjavík.**

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Upplýst samþykki

Heiti verkefnis: Lífeðlisleg viðbrögð streitu hjá nemendum með fjölskyldusögu af heilablóðfalli.

Nafn rannsakanda: Fjóla Huld Sigurðardóttir og Mardís Sara Karlsdóttir

Vinsamlegast merktu við

1. Ég staðfesti að ég hafi lesið upplýsingablaðið fyrir eftirfarandi rannsókn og að ég hafi haft tækifæri til þess að spyrja spurning. ☐
2. Ég staðfesti að ég hef skilið þær upplýsingarnar sem hafa verið gefnar og hef haft nægan tíma til þess að íhuga upplýsingarnar. ☐
3. Ég skil að þátttaka mín er valfrjáls og að mér er frjálst að hætta á hverri stundu, án þess að tilgreina ástæðu, og án þess að lagaleg réttindi mín verði fyrir áhrifum. ☐
4. Ég samþykki að taka þátt í ofangreindri rannsókn. ☐

Nafn þátttakanda	Dagsetning	Undirskrift
Rannsakanadi	Dagsetning	Undirskrift

Appendix E

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HÁSKÓLINN Í REYKJAVÍK

Samantekt rannsóknar

Heiti rannsóknar:	Lífeðlisleg viðbrögð við streitu hjá ungu fólki með Týpu D persónuleika og fjölskyldusögu um heilablóðfall.
Rannsakandi:	Fjóla Huld Sigurðardóttir
Leiðbeinendur:	Jack E. James Mardís S. Karlsdóttir

Markmið rannsóknar:

Markmið rannsóknarinnar var að skoða áhrif Týpu D persónuleika á lífeðlisleg viðbrögð við streitu en rannsóknir hafa sýnt að þeir einstaklingar sem hafa Týpu D persónuleika sýni oftast minni viðbrögð. Einnig var skoðað hvort þátttakendur með fjölskyldusögu af heilablóðfalli sýni sterkari sympatísk og parasympatísk viðbrögð í streitutengdum aðstæðum heldur en þeir sem ekki hafa fjölskyldusögu um heilablóðfall. Samvirkni á milli fjölskyldusögu um heilablóðfall og Týpu D persónuleika voru einnig skoðuð.

Niðurstöður:

Niðurstöður rannsóknarinnar leiddu í ljós að Týpu D persónuleiki orsakaði aukin TPR viðbrögð en hafði ekki önnur áhrif á lífeðlisleg viðbrögð. Fjölskyldusaga um heilablóðfall hafði ekki áhrif á lífeðlisleg viðbrögð þátttakenda.Þegar samvirkni á milli fjölskyldusögu og Týpu D persónuleik var skoðuð var Týpu D skipt í undirþættina neikvæð áhrif (NA) og félagslega hömlun, en hvoru tveggja er hluti af greiningu Týpu D. Í ljós kom samvirkniáhrif fjölskyldusögu og NA á SBP, CO and TPR.

Undirskrift

Rannsakandi:	Fjóla Huld Sigurðardóttir	**Leiðbeinandi:**	Jack E. James

Takk fyrir. Þátttaka þín í verkefninu var mikils metin. Ef þú hefur einhverjar athugasemdir varðandi verkefnið ekki hika við að hafa samband við Sálfræðideild Háskólans í Reykjavík