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Lipidemic Effects of Kissing are Mediated by Stress: Results from a National Probability Sample

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ABSTRACT

Previous studies have identified associations between affectionate communication and blood lipid levels but been limited by small, homogenous samples and failed replication attempts. Moreover, no study has tested the prediction derived from affection exchange theory that stress mediates the association between affectionate behavior and health. Using secondary analyses of data from the Midlife in the United States (MIDUS) Refresher study Biomarker Project, this paper remedies these limitations by testing the prediction that stress mediates the association between kissing and serum levels of triglycerides, high-density lipoproteins, and low-density lipoproteins using a large probability sample of U.S. American adults ($N = 863$). Results indicate significant indirect effects of kissing frequency on triglycerides and high-density lipoproteins for participants who reported kissing seven or more times in the previous month.

KEYWORDS

Kissing; lipids; health; affection exchange theory

Kissing is widely observed among human cultures (Eibl-Eibesfeldt, 2017), and although it is not ubiquitous (see Jankowiak et al., 2015), it has a prominent place in the cadre of nonverbal communication behaviors used to express interpersonal affection (see Floyd, 2019). As a behavior that is strongly associated with affectionate emotion, kissing also has the potential to effect improvements in physical health parameters that are exacerbated by stress. Among the outcomes studied with respect to kissing is serum cholesterol, and although some research has shown significant effects of kissing, and affectionate communication more broadly, on cholesterol levels, that research is plagued by multiple methodological and theoretic limitations that have drawn its conclusions into question (Hesse et al., 2020). This study uses secondary analyses of data from a large probability sample of U.S. American adults to remedy multiple limitations of previous work exploring the potential of kissing to affect lipid levels via its influence on stress.

This review situates kissing as a normatively positively valenced affectionate communication behavior with the potential to covary inversely with stress. Previous research on the health effects of kissing – both positive and negative – is then reviewed, including previous investigations of cholesterol. As described, however, much of that work is plagued by limitations that include implying but never directly testing the mediational model hypothesized from affection exchange theory (AET: Floyd, 2006a) linking kissing to cholesterol specifically via its influence on stress. Besides remedying methodological limitations, such as the use of small, non-representative samples, this study therefore provides the first direct test of AET's proposal that affectionate communication contributes to physical wellness in part through its negative association with stress.

Kissing as affectionate communication

Particularly in Asia, Europe, North America, and the Middle East (Jankowiak et al., 2015), kissing is a common nonverbal means of communicating affection. This is true in the United States as well. In an exploratory study with a representative national sample ($N = 1,121$) of U.S. American adults, Floyd et al. (2021) asked participants to describe each instance of affection they had communicated to someone in the previous 24 hours, and then coded the descriptions to identify discrete affectionate behaviors. Kissing was the third-most-commonly reported affectionate communication behavior, featuring in 27.2% of described expressions, behind only verbal expressions (41.5%) and hugging (32.8%).

Kissing also occurs in perfunctory social greetings, in non-romantic sexual interaction, and in religious rituals. Such instances aside, kissing is typically observed in relationships characterized by positive affect and is enacted to express and reinforce love, attraction, attachment, and affection, whether romantic or platonic (Floyd, 2006a). It is therefore logical to expect that kissing increases positive affect, at least on the part of the kisser (see Landau, 1989). Moreover, kissing behavior is included in measures relational closeness (Berscheid et al., 1989), intimacy (Waring, 1984), and affection (Floyd & Morman, 1998), suggesting consensus among social scientists that kissing reflects positive affect. Because it is normally a positive behavior, kissing may have the ability to lessen stress in the same way that other affection behaviors – such as hugging (Cohen et al., 2015), handholding (Coan et al., 2006), and affectionate writing (Floyd et al., 2007)—have been shown to. Several studies have documented health implications of kissing, for reasons including its influence on stress, as reviewed subsequently.

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Health effects of kissing

Most research on the health effects of kissing has addressed the implications of saliva and blood exchange. These effects include facilitating transmission of influenza (Schoch-Spana, 2000), infectious mononucleosis (Balfour et al., 2005), herpes simplex viruses (Cowan et al., 2002), and meningococcal meningitis (Tully et al., 2006), as well as allergies to food (Maloney et al., 2006) and drugs (Liccardi et al., 2002).

Other studies have focused on the *benefits* of kissing. In study of 30 allergic rhinitis (AR) patients and 30 atopic dermatitis (AD) patients, Kimata (2003) reported that 30 minutes of romantic kissing significantly decreased skin wheal responses to Japanese cedar pollen and house dust mite, and reduced plasma levels of neurotrophin-3, neurotrophin-4, brain-derived neurotrophic factor, and nerve growth factor. Kimata (2006) later reported that 30 minutes of kissing reduced production of allergen-specific immunoglobulin E (IgE) in atopic patients, relative to non-clinical controls. These findings are relevant to the current study because allergic skin wheal responses and IgE production are exacerbated by stress in AR and AD patients. Kimata (2003, 2006) reasoned that if kissing a romantic partner is stress-alleviating, it should therefore precede significant reductions in these allergic responses. The same logic guides the present analyses: If kissing ameliorates the psychological and physiological experience of stress, it should therefore effect improvements in other health outcomes (besides allergic responses) that are exacerbated by stress.

The subsequent section details a theoretic and empirical argument for kissing as a stress-ameliorating communicative behavior. A review of physiological and psychological outcomes that are aggravated by stress follows, resulting in the hypothesis that kissing covaries with enhancements in these outcomes.

Kissing as a stress-alleviating behavior

A robust empirical literature demonstrates that physically affectionate communication – especially in the context of close relationships – can have stress-reduction effects. AET (Floyd, 2019) posits that expressing affection in close relationships initiates neuroendocrine processes that ameliorate stress and buffer individuals against its physiological effects, and that these benefits are independent of those associated with receiving affectionate behavior. Multiple studies have illustrated this pattern. For instance, Floyd (2006b) found that trait expressed affection was directly related ($\beta = .56$) to greater diurnal variation in the adrenal hormone cortisol, a pattern indicative of healthy hypothalamic-pituitary-adrenal axis regulation (Giese-Davis et al., 2004; see also Floyd & Riforgiate, 2008). Floyd et al. (2007) later demonstrated that during episodes of acute stress (in which cortisol levels are typically elevated), expressing affection in writing to a loved one accelerates the return of cortisol to normal levels, and Grewen et al. (2005) similarly reported that nonverbal affection reduced cortisol levels for both men and women. Floyd et al. (2007) even reported a strong inverse association ($\beta = -.85$) between affectionate communication and glycohemoglobin – an index of average blood glucose level, which is elevated by stress – after controlling for the effects of received affection, whereas van Raalte and Floyd (2021) demonstrated that hugging over a two-week period significantly reduced levels of interleuken1-β and tumor necrosis factor-α, two proinflammatory cytokines known to be elevated by stress.

Collectively, these studies reflect AET's proposition that affectionate behavior ameliorates the effects of stressors, which can account theoretically for its salutary effects on well-being. To the extent that stress exacerbates health indices such as glucocorticoids, glycohemoglobin, and proinflammatory cytokines, behaviors that ameliorate stress have the potential to effect improvements in these and similar outcomes. The present analyses apply AET's argument to an indicator of metabolic health, blood lipid levels. As detailed in subsequent sections, preliminary attempts to link affectionate behavior to lipid outcomes have been promising but have been fraught with both theoretic and methodological limitations that the present study aims to address.

Effects of affectionate behavior on blood lipids

If affectionate behavior can mitigate the effects of stress, then it is logical to predict that it can also effect improvements on physiological parameters that are exacerbated by stress, as Kimata's experiments demonstrated with allergic responses. The present paper performs secondary data analyses to test this proposition on blood lipids. Lipids are water-insoluble organic compounds that are present in the cell membranes of all body tissues and that perform numerous essential physiological functions, including maintaining membrane fluidity, producing bile, contributing to the metabolism of fat-soluble vitamins, and contributing to the production of steroid hormones, such as cortisol, progesterone, aldosterone, the estrogens, and testosterone (Welsh & Prentice-Craver, 2021). Lipids come in multiple forms, perhaps the most widely known of which is cholesterol.

Most cholesterol is produced in the liver, although the consumption of foods high in cholesterol, trans fat, and/or saturated fat (e.g., red meat, egg yolks, full-fat dairy foods, and fried foods) contributes to elevated cholesterol levels in the bloodstream (Longenbaker, 2017). Chronically elevated cholesterol – a condition known as *hypercholesterolemia*—can lead to the formation and accumulation of plaque deposits in the arteries, contributing to atherosclerosis or coronary heart disease. Multiple studies have documented that stress is associated with elevations in total cholesterol and changes in its constituent components: triglycerides, high-density lipoproteins (HDL, also known as "good cholesterol"), and low-density lipoproteins (LDL, also known as "bad cholesterol;" see, e.g., Bacon et al., 2004; Stoney et al., 1999). The specific mechanisms through which stress elevates cholesterol are as yet unknown, although they may reflect evolved processes through which stress-induced increases in energy (in the form of metabolic fuels such as glucose and fatty acids) initiate ancillary processes that elevate levels of LDL in the bloodstream (see Steptoe & Brydon, 2005). Other speculation implicates sympathetic nervous system activation and the rapid release of glucocorticoids, such as cortisol, and catecholamines, such as epinephrine and norepinephrine. Research shows that lipoprotein lipase activity is inhibited by both norepinephrine and cortisol (Jansen & Hülsmann, 1985; Miller et al., 1989),

which decreases the clearance of triglycerides, increases LDL concentrations, and decreases HDL concentrations (Huttunen et al., 1976).

Because blood lipids are exacerbated by stress, and because affectionate behavior has stress-ameliorating physiological effects, it is logical to propose that increasing affectionate behavior can decrease cholesterol. Three studies grounded in AET have demonstrated such an effect. In two experiments, Floyd et al. (2007) modified the Pennebaker expressive writing paradigm (Pennebaker & Chung, 2011) to induce serial affectionate writing in a group of healthy adults. Positing that affectionate writing would reduce stress relative to controls, the authors hypothesized and found a reduction in total cholesterol. A later experiment by Floyd et al. (2009) conceptually replicated that finding by inducing more frequent romantic kissing in marital and cohabiting relationships, documenting a reduction in total cholesterol over the six-week trial. Finally, the proposed connection between affection and cholesterol is bolstered – albeit less directly – by a correlational study by Floyd et al. (2017) documenting that perceived social inclusion was significantly associated with LDL (but not HDL), as well as with blood glucose. Despite their promise in demonstrating an association between affectionate communication and health – via the assessment of lipid levels, in this case – these studies have been plagued by multiple limitations, as the next section describes.

Four limitations of previous lipid studies

Although they identified significant associations between affectionate behavior (including kissing) and blood lipid levels, the Floyd lipid studies (Floyd et al., 2007, 2009, 2017) were limited in at least four consequential ways. First, samples were small and homogenous demographically and geographically. Sample sizes in the Floyd studies ranged from 30 to 52 and largely comprised students from the same geographic area. Although sufficient to identify significant associations, these small and homogenous samples pose a threat to external validity. Second, as Floyd (2019) and Hesse et al. (2020) both pointed out, some attempts to replicate the Floyd lipid results have been unsuccessful (perhaps due to similarly limited samples). Third, with the exception of the Floyd et al. (2017) study, which focused specifically on HDL and LDL, the previous Floyd lipid studies measured only total cholesterol as an outcome variable. This approach is limiting in terms of specificity, insofar as some predictions may manifest for some components of cholesterol but not others, despite similar hypothesis; indeed, the hypothesis in the Floyd et al. (2017) study was supported for LDL but not for HDL. The ability to test AET's prediction separately on the constituent components of cholesterol adds empirical specificity to the prediction that affectionate behavior affects lipid levels via its effects on stress.

Finally, on the latter note, the prediction derived from affection exchange theory that affectionate behavior predicts healthier lipid profiles, such as lower levels of triglycerides, LDL, and/or total cholesterol and higher levels of HDL, is predicated on a mediating effect of stress, yet no previous study has tested that argument. Specifically, according to AET, affectionate behavior benefits health, in part, by modulating stress, which implies a mediated model in which affectionate communication is inversely associated with stress and stress is health suppressive, regardless of whether affectionate behavior exerts a direct effect on health or not. Although the Floyd et al. (2009) lipid study measured stress as an additional outcome of the kissing manipulation, it did not ascertain whether stress mediated the association between kissing and cholesterol – nor did the other Floyd lipid studies – even though that model is specifically implied by the argument derived from AET.

The present study addresses all four limitations by performing secondary analyses on data collected from a large probability sample of U.S. American adults and by explicitly testing the stress-mediated model implied by AET on constituent components of cholesterol. Specific hypotheses are articulated subsequently.

Hypotheses

The present study applies the logic of AET to the task of predicting how the frequency of kissing predicts three lipid outcomes: triglycerides, HDL, and LDL. The argument derived from AET implies that kissing exerts an indirect effect on health via an inverse association with stress.

H1: Kissing frequency is negatively associated with stress.

AET's argument next implies that stress is inversely associated with health, manifested here in positive correlations with triglycerides and LDL and a negative correlation with HDL.

H2: Stress is positively associated with triglycerides and LDL and negatively associated with HDL.

Finally, AET implies that affectionate behavior exerts a significant indirect effect on health via stress.

H3: Stress mediates the effect of kissing frequency on triglycerides, HDL, and LDL.

Method

Participants

The participants were originally recruited for the Midlife in the United States (MIDUS) Refresher sample (Ryff et al., 2010), a nationally representative sample of 3,577 U.S. American adults. The Refresher study was conducted to replenish the original MIDUS study, which aimed to explore the effects of social, biological, and psychological characteristics on age-related variations in wellness among a representative sample of U.S. Americans. From the original sample, a subsample (N = 863) completed the MIDUS Refresher Biomarker Project (Weinstein et al., 2017) study by filling out psychosocial measures and then having physiological measures taken at a clinical research unit.[1] The sample was 52% female and 48% male with an average age of 50.84 years (SD = 13.41 years; range 23–85 years). Most of the participants (58%) were married at the time of the study. Average annual household income was $52,636 ($SD$ = 50,446.77). Most of the sample (52%) had a college degree or higher, whereas 44% had a high school diploma and/or some college but no degree. With respect to racial

Table 1. Descriptive statistics and intercorrelations for study variables ($N = 863$).

Variable	Min	Max	*M*	*SD*	1	2	3	4	5	6
1. Kissing frequency	1.00	3.00	2.64	0.55	–					
2. Stress	10.00	44.00	22.49	6.36	−.14†	–				
3. Age	26.00	78.00	52.72	13.44	−.07*	−.23†	–			
4. BMI	17.08	77.58	30.40	7.65	−.15†	.11†	−.03	–		
5. Triglycerides	26.00	1071.00	116.89	73.77	−.03	.07*	.02	.18†	–	
6. HDL	20.00	137.00	59.04	19.59	.03	−.09†	.11†	−.35†	−.44†	–
7. LDL	3.00	323.00	98.56	34.43	−.03	−.01	−.06	−.03	.18†	−.07*

BMI = body mass index; HDL = high-density lipoproteins; LDL = low-density lipoproteins. Triglycerides, HDL, and LDL are measured in mg/dL. *$p < .05$ (two-tailed); †$p < .01$ (two-tailed). Exercise is not included in this table because it was a dichotomous variable.

background, 71% identified as white, 20% as Black or African American, 2% as Native American or Alaska Native/Aleutian Islander, 1.3% as Asian, and .2% as Native Hawaiian/Pacific Islander, whereas 5.9% claimed other racial backgrounds. Love et al. (2010) reported that the Biomarker Project participants were comparable to the full MIDUS sample on all demographic metrics except that the Biomarker sample was more educated. A sensitivity power analysis (Faul et al., 2009) indicated that the sample size of 863 provides in excess of 98% power to identify small ($f^2 = .018$) effect sizes in multiple regression, assuming $\alpha = .05$. The MIDUS study was reviewed and approved by the Education and Social/Behavioral Sciences and the Health Sciences IRBs at the University of Wisconsin-Madison.

Procedure

After providing informed consent and completing self-administered questionnaires common to all MIDUS participants, the Biomarker Project participants completed medical histories and clinical/physiological assessments during an overnight stay at a clinical research unit located either at University of California, Los Angeles (UCLA; 34.1%), University of Wisconsin (38.7%), or Georgetown University (27.2%). Biomarker data were collected between the years of 2012 and 2016. Participants chose the clinical research unit that would best minimize their travel burden. During the morning, before eating breakfast, participants had a fasting blood sample collected according to standardized procedures (Weinstein et al., 2017).

Measures

Kissing frequency was measured with a single item asking, "Over the past month, how often did you spend time kissing?" Response options were *never* (24.0%), *1–6 times* (32.4%), and *7 or more times* (43.1%, with .5% missing data). *Stress* was measured with the 10-item Perceived Stress Scale (Cohen et al., 1983; McDonald's $\omega = .86$). Items included "In the last month, how often have you been upset because of something that happened unexpectedly?" and "In the last month, how often have you felt nervous and stressed?" *Exercise*, used here as a covariate, was measured with a single item asking, "Do you engage in regular exercise or activity of any type for 20 minutes or more at least 3 times/week?" (with response options of yes, coded as 1, or no, coded as 0). Physiological outcomes were assessed during participants' overnight stays at the clinical research unit. A fasting blood draw was assessed for markers of lipid metabolism: *triglycerides, high-density lipoproteins (HDL)*, and *low-density lipoproteins (LDL)*. Lipids were assayed at Meriter Labs (Madison, WI) using a Roche Cobas Analyzer (Roche Diagnostics, Indianapolis, IN) to analyze frozen serum.[2] A physical examination during the overnight stay assessed *body mass index (BMI)*, used here as a covariate.

This paper's predictions and analytical strategy were pre-registered with AsPredicted.org on 2 July 2021.[3] Means, standard deviations, and intercorrelations for the study's measures appear in Table 1.

Results

Descriptive statistics

Average values were within or nearly within recommended ranges for all three lipid outcomes. According to the Centers for Disease Control and Prevention (CDC, 2021), healthy triglyceride levels are less than 150 mg/dL, whereas healthy HDL levels are 60 mg/dL and above and healthy LDL levels are less than 100 mg/dL. Mean values in the present sample were 116.89 mg/dL for triglyceride, 59.04 mg/dL for HDL, and 98.56 mg/dL for LDL. Given the range of values for each outcome, the hypotheses were tested both with the entire sample and after suppressing those with lipid values ±3SD from their respective means. Only 3% of the sample had out-of-range values, and there were no substantive differences in the results when outliers were removed, so the outliers were retained in the hypothesis tests.

Triglyceride levels differed as a function of participant sex, with men ($M = 129.59$, $SD = 83.67$) having higher levels than women ($M = 105.21$, $SD = 61.14$), Welch's t (740.59) = 4.82, p (two-tailed) <.001, $d = .33$. Conversely, women had higher HDL levels ($M = 64.70$, $SD = 19.89$) than did men ($M = 52.85$, $SD = 17.26$), Welch's t (846.48) = −9.30, p (two-tailed) <.001, $d = .64$. Men's LDL ($M = 97.10$, $SD = 35.94$) was not significantly different from women's ($M = 99.89$, $SD = 32.98$), Welch's t (824.47) = −1.17, p (two-tailed) = .24, $d = .08$.

As noted in Table 1, the average body mass index (BMI) of the sample was 30.40, just above the CDC (2020) cutoff for obesity. BMI did not differ as a function of sex, with men ($M = 30.05$, $SD = 6.62$) having a nearly identical average BMI as women ($M = 30.72$, $SD = 8.48$), Welch's t (839.56) = −1.32, p (two-tailed) = .19, $d = .09$. According to CDC guidelines, .5% of the sample was underweight (BMI <18.5), whereas 23.8% of the sample had a healthy weight (BMI = 18.5–24.9), 29.4% were overweight (BMI = 25.0–29.9), and 45.2% of the sample was obese (BMI ≥30.0). With respect to exercise, nearly three-quarters of participants (73%) indicated that they engaged in exercise for 20 minutes or more at least 3 times per week, whereas the remainder (27%) indicated that they did not.

Hypotheses

The hypotheses predicted that H1) kissing frequency is negatively associated with stress; H2) stress is positively associated with triglycerides and LDL and negatively associated with HDL; and, H3) stress mediates the effect of kissing frequency on triglycerides, HDL, and LDL. The hypotheses were tested using model 4 of Hayes (2017) PROCESS. Bootstrapping procedures were used to generate regression weights for the direct and indirect effects, and confidence intervals around the indirect effects were used to infer statistical significance. Due to its limited response options, kissing frequency was treated as a categorical variable and the categories were compared using sequential contrasts that differentiated 1) the "never" kissing group to the "1–6 times" and "7+ times" groups, and 2) the "never" and "1-6" groups to the "7+" group. Based on previous research, participant sex, age, body mass index, and exercise were used as covariates (Gostynski et al., 2004; Kodama et al., 2007). The tests of H1 render identical results for all three lipid outcomes because the direct effect of kissing frequency on stress does not vary by lipid outcome. Tests of H2 and H3 vary by lipid outcome.

Table 2. Direct and indirect effects of kissing frequency on lipid outcomes (N = 863).

Predicting Stress			
X_1 contrast		−.37	
X_2 contrast		−1.49†	
Sex		1.18†	
Age		−.10†	
BMI		.05*	
Exercise		1.33†	
Predicting Lipid Outcome	*Triglycerides*	*HDL*	*LDL*
X_1 contrast	−.01	−1.53	−1.73
X_2 contrast	1.66	.69	−.85
Stress	.81*	−.21*	−.15
Sex	−26.54†	13.12†	2.36
Age	.12	.17†	−.16
BMI	1.62†	−.90†	−.19
Exercise	20.80†	−3.91†	4.47
Indirect Effects			
X_1 contrast	−.30 (−1.64, .71)	08 (−.18, .42)	06 (−.24, .50)
X_2 contrast	−1.21 (−2.70, −.12)	31 (.01, .72)	23 (−.36, .88)

Values in table are unstandardized regression coefficients from PROCESS model 4. X_1 contrast uses "never" kissing group as reference; X_2 contrast uses "7+" kissing group as reference. BMI = body mass index. Values in parentheses are 95% confidence intervals. *p < .05 ; †p < .01.

Triglycerides

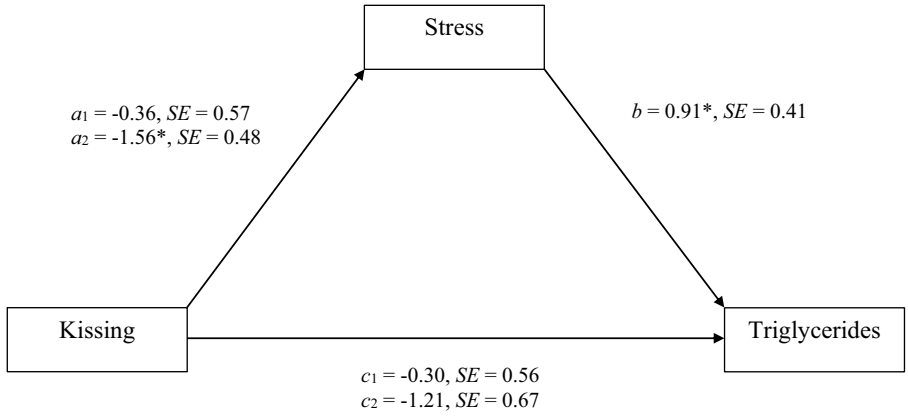
The hypothesized effects of kissing frequency were significant only for the comparison between those who kissed never/1–6 times and those who kissed 7+ times in the previous month. For that contrast, kissing was inversely associated with stress, β = −1.56, p = .001; stress was positively associated with triglycerides, β = .91 , p = .03; and kissing frequency exerted a significant indirect effect on triglycerides, β = −1.21, 95% CI: −.2.70, −.12. The direct effect of kissing on stress and the indirect effect of kissing on triglycerides were nonsignificant for the comparison between those who kissed never and those who kissed 1-6/7+ times in the previous month. Full regression results appear in Table 2, and the model appears in Figure 1 For triglycerides, H2 is supported and H1 and H3 are partially supported.

High-density lipoproteins (HDL)

As with triglycerides, the hypothesized effects of kissing frequency were significant only for the comparison between those who kissed never/1–6 times and those who kissed 7+ times in the previous month. As before, kissing was inversely associated with stress, β =−1.56, p = .001; stress was negatively associated with HDL, β =−.21, p = .03; and kissing frequency exerted a significant indirect effect on HDL, β = .31 , 95% CI: .01, .72. The direct effect of kissing on stress and the indirect effect of kissing on HDL were nonsignificant for the comparison between those who kissed never and those who kissed 1-6/7+ times in the previous month. Full regression results appear in Table 2, and the model appears in Figure 2 For HDL, H2 is supported and H1 and H3 are partially supported.

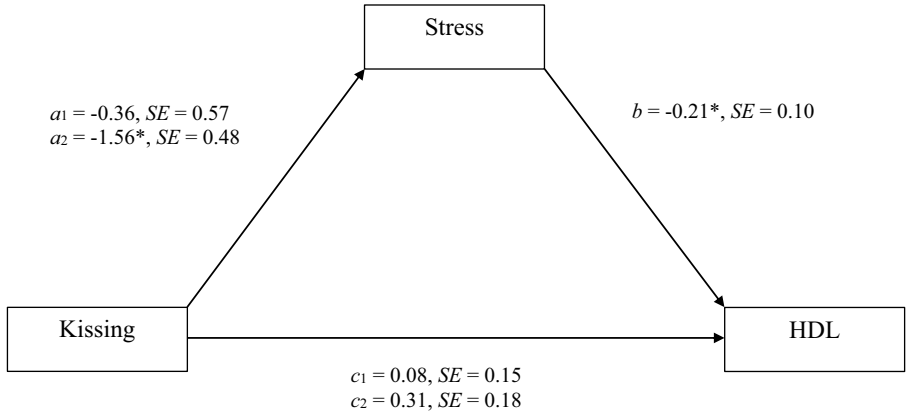
Low-density lipoproteins (LDL)

As with HDL and triglycerides, kissing was inversely associated with stress only for the comparison between those who kissed never/1–6 times and those who kissed 7+ times in the previous month. Stress was nonsignificantly associated with LDL, β = −.15, p = .44. Kissing frequency exerted nonsignificant indirect



*p < .05; paths with subscript 1 compare "never" kissing group to "1-6" and "7+" groups; paths with subscript 2 compare "never" and "1-6" kissing groups to "7+" group.

Figure 1. Direct and indirect effects of kissing frequency on triglycerides (N = 863).



*p < .05; paths with subscript 1 compare "never" kissing group to "1-6" and "7+" groups; paths with subscript 2 compare "never" and "1-6" kissing groups to "7+" group.

Figure 2. Direct and indirect effects of kissing frequency on HDL (N = 863).

effects on LDL for both contrasts. Full regression results appear in Table 2. For LDL, H1 is partially supported and H2 and H3 are unsupported.

Discussion

A robust empirical literature attests to the health supportive nature of affectionate communication (Hesse et al., 2020). As Floyd (2019) himself has addressed, however, the finding that affectionate communication – including kissing, specifically – affects lipid levels has been scrutinized for its failure to replicate, which is likely attributable in part to samples that were small and offered poor generalizability. In a meta-analysis of research on affectionate communication and health, Hesse et al. (2020) even initially suppressed the Floyd cholesterol findings out of concern that they represented alpha errors, although the results of the meta-analysis were ultimately unaffected by whether the cholesterol studies were included or excluded. In addition, two of the Floyd cholesterol studies lacked specificity by measuring only total cholesterol, and – perhaps most important – none tested the mediational model directly implied by affection exchange theory, wherein affectionate communication influences health parameters such as lipids via its effect on stress.

The present data from the MIDUS Refresher study Biomarker Project offered the opportunity to test AET's mediational model on the constituent components of cholesterol (rather than simply on total cholesterol levels) using a large probability sample of U.S. adults. Results were significant only when comparing those who kissed never or 1–6 times in the previous month with those who kissed 7 or more times. As predicted, kissing was inversely associated with stress; stress was positively associated with triglycerides (but not LDL) and negatively associated with HDL; and stress significantly mediated kissing's association with triglycerides and HDL (but not LDL).

These findings offer substantially more empirical clarity regarding the relationship between affectionate behavior and lipids than any of the earlier Floyd studies. Most important, they support AET's contention that affectionate behavior influences health, in part, via its association with stress, a proposition not previously tested even in studies that were explicitly based on AET. At the same time, these findings clarify some limits of AET's prediction. Specifically, low-frequency kissing (six or fewer times over the course of a month) is not potent enough to affect the levels of those lipids via a negative influence on stress. This type of *threshold effect* for affectionate communication is only partially anticipated by AET, as discussed below. It is possible that this, along with sample limitations, also accounts for failed replications of the earlier cholesterol studies, insofar as experiments that failed to replicate an effect of kissing on lipid levels may have induced insufficient levels.

Another limit to AET's prediction is that the hypothesized effects manifested only for triglycerides and HDL, not also for LDL. Why kissing exerted the predicted effect on triglycerides and HDL but not on LDL is unclear. LDL had more variance than HDL, so a relative lack of variance is not to blame, nor is a relative lack of measurement reliability, given that all three indices were assayed in the same procedure. It is not the case that affectionate behavior influences only "desirable" lipids, such as HDL, commonly known as *good cholesterol*, because it also exerted a significant influence on triglycerides. Previous experiments testing the effect of affectionate behavior on lipids (e.g., Floyd et al., 2009) offer no empirical clarity because they measured only total cholesterol. The correlational study by Floyd et al. (2017) in fact found that social inclusion had a significant relationship with LDL but not with HDL. Social inclusion is a perception, however, whereas kissing is a specific behavior, and the Floyd et al. study used a small, nonrepresentative sample, unlike the current analyses. In sum, an explanation for the lack of a significant effect for LDL, given the significant effects on triglycerides and HDL, is elusory and must await further empirical clarification.

Theoretic and clinical implications

The presenting findings have at least two implications for AET, the first of which is that its assertion that affectionate communication influences health in part via a negative association with

stress has merit. This implication is frequently used to hypothesize associations between affectionate communication and well-being, yet no analyses have explicitly tested the mediational model. Importantly, AET does not identify stress amelioration as the *only* causal pathway via which affectionate behavior can enhance wellness, but it is a primary pathway articulated in the theory. Although the present analyses did not offer unequivocal support for the mediational model – insofar as the effect on LDL was nonsignificant – they are the first to demonstrate AET's claim, and further experimental tests of the effect of affectionate communication on wellness should likewise incorporate the mediational model upon which their hypotheses are based.

A second implication, however, is that AET may not adequately account for the effect of the frequency of affectionate behavior. As the present findings demonstrate, kissing exerted no salutary effects on lipids until it exceeded a particular threshold. It is unfortunate that the measurement of kissing frequency had too few response options (a point addressed below) to permit a more nuanced examination of the frequency above which kissing was beneficial, but even the rudimentary distinction between "never," "1–6 times," and "7+ times" within a month evidenced the existence of a threshold effect for kissing on lipid levels.

Can AET account for this threshold effect? In its fourth postulate, AET does specify that humans vary in their optimal tolerances for affectionate communication – which are "bounded on the lower end by *need*, or how much affectionate emotion or behavior are required, and on the upper end by *desire*, or how much affectionate emotion or behavior are wanted" (Floyd, 2019, p. 32, italics in original)—and that neither insufficient nor excessive affectionate communication is beneficial. Indeed, research has shown that receiving both *too little* affection (Floyd, 2016) and *too much* affection (Hesse & Mikkelson, 2021) not only fail to support health but are actually associated with health detriments.

Without having measured the frequency of kissing that participants in the MIDUS study felt they required or desired in their lives, it is impossible to know whether their observed frequencies fell within their range of optimal tolerance. Absent that detail, it is equally impossible to know whether AET's postulate can account for the threshold effect observed here, wherein kissing demonstrated no effects below seven instances per month. That is because AET's assertion is relative rather than absolute, insofar as the effects of affectionate behavior are posited to be relative to an individual's range of optimal tolerance, such that a frequency of affectionate communication that is sufficient for one person may be insufficient or excessive for another. Whether the continued utility of AET ultimately requires it to postulate an absolute threshold for the benefits of affectionate behavior remains to be seen, but that is a viable question for future theoretic development.

To the extent that relational affection can improve lipid levels, these findings may have clinical import as well. Floyd's research program has already identified affectionate behaviors with potential therapeutic benefit, including kissing, hugging, and verbal affection (see Floyd et al., 2007, 2017; van Raalte & Floyd, 2021). Elevated cholesterol is a significant risk factor for cardiovascular disease (see Abdullah et al., 2018), currently the leading cause of death in the United States (CDC, 2019). The design of the present study does not allow the conclusion that *increasing* kissing necessarily improves lipid levels, but the present findings are consistent with that possibility. The suggestion to increase kissing is sometimes a component of marital therapy (Brezsnyak & Whisman, 2004), and could potentially be an adjunct to other common behavioral treatments for hypercholesterolemia, such as increasing exercise and modifying diet. Such a recommendation would certainly be premature without appropriate clinical research, especially given that kissing also carries multiple health risks, but this may be worth investigating with a clinical sample.

Strengths and limitations

A substantial strength of the present paper was its large probability sample. The largest sample in the Floyd cholesterol studies is 52 participants, who were relatively homogenous both demographically and geographically. Consequently, the present study's use of a large ($N = 863$) probability sample of U.S. American adults represents a considerable improvement over existing attempts to adjudicate the affection-lipid association.

Although the design of the data collection was technically nonexperimental, it nonetheless incorporated a temporal separation between psychosocial assessments (including kissing) and measurements of health parameters (including lipids). This bolsters the ability to investigate how kissing, with appropriate controls accounted for, *predicted* lipid levels, rather than simply covarying with them. Kissing and stress were measured at the same point in time, however, so the mediational model did not evidence complete temporal separation, only partial.

A significant limitation of the present study was its unsophisticated assessment of kissing, which was measured using an item from a longer scale of positive experiences. By asking participants how often they spent time kissing (as opposed to how many kisses they enacted during a specified period of time), the item was not unlike similar items on widely used affectionate communication scales, such as the affectionate communication index (ACI: Floyd & Morman, 1998). The consequential limitation of the item was its restricted response options of "never," "1–6 times," and "7+ times." In comparison to a Likert-type scale such as that employed by the ACI, which measures the frequency of affectionate behaviors on a 7-point scale, the current study's response options for the kissing item restricted the scores' variance, which may have limited its statistical power. In addition, it is unclear why the second and third response options were separated at 6 instances of kissing versus, say, 5, 10, or any other number. These limitations are understandable given that the MIDUS study was not designed to study kissing, or even affection, per se, but they do constitute an important psychometric limitation.

A second limitation is that the present results cannot be generalized beyond the U.S. American adult population, and it is conceivable that kissing may not have the same stress-ameliorating effects in cultures where it is less commonly observed, such as in Central America, South America, and Africa (Jankowiak et al., 2015). Even in such cultures, kissing can still influence health in other ways, such as through viral transmission, but it may not influence stress-exacerbated health parameters such as lipids if kissing itself has no appreciable stress-alleviating function. Similarly, the present findings cannot be generalized to children, for whom the connection between kissing behavior and stress is entirely untested.

Conclusion

When first considered, the idea that affectionate communication might influence a health outcome such as lipid levels may seem implausible. AET highlighted the association between affectionate communication and stress two decades ago (Floyd, 2002), however, and researchers have known for more than half a century that stress is related to cholesterol (Wertlake et al., 1958). These observations combine to support a mediated association between affectionate communication and cholesterol, and although some previous studies have demonstrated direct effects, they have been plagued by multiple limitations. By drawing on a substantially improved sample and testing AET's proposed mediational model explicitly, the present analyses offer markedly more trustworthy evidence of the affection-lipid association.

Notes

1. Data from the MIDUS Refresher Biomarker Project are available to researchers at https://www.icpsr.umich.edu/web/NACDA/studies/29282.
2. Although total serum cholesterol was also assayed, the analysis of its constituent components – triglycerides, LDL, and HDL – provided an opportunity to test the prediction of AET with greater specificity than in earlier studies. Total cholesterol was not analyzed here in addition to triglycerides, LDL, and HDL to avoid redundancy and elevated Type I error.
3. An anonymized version of the preregistration is viewable at https://aspredicted.org/blind.php?x=559tu2.

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