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Article

Genetic and Environmental Influences on Perceived Social Support: Differences by Sex and Relationship

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Abstract

Previous research has shown that self-reports of the amount of social support are heritable. Using the Kessler perceived social support (KPSS) measure, we explored sex differences in the genetic and environmental contributions to individual differences. We did this separately for subscales that captured the perceived support from different members of the network (*spouse, twin, children, parents, relatives*, *friends* and *confidant*). Our sample comprised 7059 male, female and opposite-sex twin pairs aged 18–95 years from the Australian Twin Registry. We found tentative support for different genetic mechanisms in males and females for support from *friends* and the *average KPSS* score of all subscales, but otherwise, there are no sex differences. For each subscale alone, the additive genetic (A) and unique environment (E) effects were significant. By contrast, the covariation among the subscales was explained — in roughly equal parts — by A, E and the common environment, with effects of different support constellations plausibly accounting for the latter. A single genetic and common environment factor accounted for between half and three-quarters of the variance across the subscales in both males and females, suggesting little heterogeneity in the genetic and environmental etiology of the different support sources.

Keywords: Social support; twin; genetic; environment; sex

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Human beings evolved in groups and are, by nature, social animals (Caporael, 1997). Having social support leads to reduced mortality risk and better health according to recent meta-analyses (Chu et al., 2010; Jane-Llopis et al., 2003; Ozer et al., 2003; Prati & Pietrantoni, 2009; Robertson et al., 2004; Shor et al., 2013; Yarcheski et al., 2004) and reviews (Kessler & McLeod, 1985; Seeman, 1996; Uchino, 2006; Vaux, 1988). Cobb (1976, p. 300) defined social support as 'information leading the subject to believe that he is cared for and loved … esteemed and valued … and belongs to a network of communication and mutual obligations'. Finfgeld-Connett (2005) identified an array of processes such as comforting gestures, body language, attentive listening, sharing experiences, humor and knowing someone was available. In short, no single observable behavior has captured its entirety. We assessed perceived rather than received social support. Perceived support is the subjective assessment of whether members of a social network are available to provide support (Cohen & McKay, 1984), so it potentially involves all the processes outlined above. Moreover, it is more strongly related to health and wellbeing (Kessler & McLeod, 1985; Schwarzer & Leppin, 1991; Turner, 1992; Vaux, 1988; Wethington & Kessler, 1986). Received social support, by contrast, involves objective aspects such as the number in the social network, frequency of club or church attendance and marital status (Roberts & Gotlib, 1997), and so omits many of the subjective processes.

Humans differ substantially in their levels of social support, and in this article, we investigate the degree to which these differences are innate or shaped by the environment. Using twins, we partition the variance into additive genes (A), the common environment (C; effects shared by twins) and the unique environment (E; those unique to each twin). Earlier literature conceptualized social support as a property of the social environment where sources external to the individual determined levels of support (Pierce, 1997). There is merit in considering a model that incorporates etiologies internal to the individual, for two reasons. First, social support has trait-like stability over time (Coventry et al., 2004; Lakey & Cohen, 2000; Sarason et al., 1990) despite changes in the composition of the social network (I. G. Sarason et al., 1983; Solomon et al., 1988) and despite periods of developmental transition when elevated environmental change is expected (Sarason et al., 1986). Second, the twin studies of social support presented in Table 1 show genes account for approximately one-third of the variance in social support (Agrawal et al., 2002; Bergeman et al., 2001; Bergeman et al., 1990; Ji et al., 2008; Kendler, 1997; Kessler et al., 1992; Raynor et al., 2002), though this varies depending on the measure and source of support. For measures of perceived support, E is generally higher than for received support, given reports of perceived support are more subjective (Bergeman et al., 2001; Bergeman et al., 1990; Ji et al., 2008; Kendler, 1997). If both twins in the pair share the source of support (i.e., their parents or relatives), E is lower than

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Table 1. Previous heritability studies of social support and the percentages of variance explained. Where confidence intervals were reported they are presented in brackets below the estimate

Author	No. complete twin pair responses	Measure	Subscales	Additive genetic	Common environment	Dominant genetic	Unique environment
Bergeman et al. (1990)	424 pairs reared apart and together	Modified version of ISSI[a]; 18 items	Adequacy[f, #]	.30	.00	–	.70
			Quantity[f]	.00	.10	–	.63
Kessler et al. (1992)	821 female pairs	KPSS[b]; 19 items	Spouse[#]	*ns*	.24	*ns*	.76
			Other family/relatives[#]	.28	.18	*ns*	.54
			Friends[#]	.32	*ns*	*ns*	.68
			Confidant[e, #]	.00	*ns*	.50	.50
		Integration into affiliative networks; 4 items	Frequency of interaction with relatives	*ns*	.40	*ns*	.60
			Frequency of interaction with friends	*ns*	.27	*ns*	.73
			Frequency of church attendance	.36	.44	*ns*	.20
			Frequency of club attendance	.52	*ns*	*ns*	.48
Kendler (1997)	854 female pairs	Social Interaction Scale[d]; 16 items	Relative problem[#]	.49	.20	–	.30[-e]
			Friend problem[#]	.59	*ns*	–	.41[-e]
			Relative support[#]	.44	.28	–	.28[-e]
			Confidant[e]	.66	*ns*	–	.34[-e]
			Friend support[#]	.43	*ns*	–	.57[-e]
			Social integration	.75	*ns*	–	.25[-e]
Agrawal et al. (2002)	3074 pairs of males and females	Social Interaction Scale[d]; 16 items	Male				
			Relative problem[#]	.31 [.07, .46]	.10 [.00, .26]	–	.59 [.25, .93]
			Friend problem[#]	.06 [.00, .25]	.15 [.00, .26]	–	.79 [.25, .93]
			Relative support[#]	*ns*	.26 [.19, .31]	–	.74 [.69, .81]
			Confidant[e]	.27 [.17, .33]	*ns*	–	.73 [.60, .83]
			Friend support[#]	.28 [.10, .36]	*ns*	–	.72 [.49, .90]
			Social integration	.29 [.14, .36]	*ns*	–	.71 [.51, .86]
			Female				
			Relative problem[#]	.33 [.08, .46]	.06 [.00, .29]	–	.61 [.25, .93]
			Friend problem[#]	.25 [.00, .34]	*ns*	–	.75 [.25, .93]
			Relative support[#]	.20 [.09, .26]	.15 [.07, .25]	–	.65 [.49, .84]
			Confidant[e, g]	.02 [.00, .24]	.21 [.00, .37]	–	.77 [.39, 1.00]
			Friend support[#]	.28 [.10, .36]	*ns*	–	.72 [.49, .90]
			Social integration	.31 [.08, .40]	*ns*	–	.69 [.41, .96]
Raynor et al. (2002)	232 pairs of males and females	ISEL; 48 items		.00	–	.59	.41

(*Continued*)

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Table 1. (*Continued*)

Author	No. complete twin pair responses	Measure	Subscales	Additive genetic	Common environment	Dominant genetic	Unique environment
Bergeman et al. (2001)	422 pairs reared apart and together	Modified version of ISSI[a]; 24 items	Quantity (friends): Time 1	.42	–	–	.58
			Time 2	.19	–	–	.81
			Time 3	.33	–	–	.67
			Adequacy (friends and family)[#]: Time 1	.22	–	–	.78
			Time 2	.24	–	–	.76
			Time 3	.41	–	–	.59
			Quantity (family): Time 2	.26	–	–	.74
			Time 3	.33	–	–	.67
Ji et al. (2008)	324 pairs of males and females	A scale from Xiao (1994); 10 items	Objective support	n.s.	.38 [.00, .48]	–	.62 [.51, .74]
			Subjective support	.30 [.00, .64]	.27 [.00, .56]	–	.43 [.34, .55]
			Utilizing support	.27 [.00, .53]	.15 [.00, .48]	–	.58 [.47, .70]
Wang et al. (2017)	1158 pairs of males and females	Lubben Social Network Scale – Revised	Support quantity	.49 [.43, .55]	–	–	.51 [.45, .57]
		Multidimensional Scale of Perceived Social Support	Support quality (total) [#]	.55 [.48, .60]	–	–	.45 [.40, .52]
			Support quality (significant other) [#]	.43 [.36, .50]	–	–	.57 [.50, .64]
			Support quality (family) [#]	.59 [.53, .64]	–	–	.41 [.36, .47]
			Support quality (friends) [#]	.37 [.29, .44]	–	–	.63 [.56, .71]

Notes (cont.). [#]Subscales measuring *perceived* social support.
ns, parameters were estimated but were nonsignificant so were fixed to zero; –, Parameter not estimated. ISSI, Interpersonal Support Evaluation List. [a]Henderson (1980). [b]With a different factor structure to the current study. [c]Includes co-twin, children, parents and relatives. [d]Same measure but factor analyses were unique to each study so subscales from each study are not always directly comparable. [e]Measures are not directly comparable. With regard to *confidant* (someone with whom you have a close relationship and can share your most private feelings), items were *Have a* (Kessler et al. 1992; identical to current study); *Have a* and *No. of* (Kendler, 1997) and *No. of* (Agrawal et al. 2002). [-e] Measurement error removed. [f]For quantity, the remaining 0.27 of variance was accounted for by the correlated environment; representing similarity between twins beyond heredity and the shared rearing environment. For Adequacy, the correlated environment did not account for any variance. [g]Here, the A and C parameters, but not both, could be dropped, so they used Akaike information criterion (AIK) to determine the best model fit, which was ACE.

if they do not (i.e., friends or confidant; Agrawal et al., 2002; Kendler, 1997; Kessler et al., 1992). The first aim of this paper is to replicate this previous research.

Some research suggests that men and women are fundamentally different in their interpersonal relations, and they represent different social cultures (see Henley & Kramarae, 1991; Johnson, 1989; Tannen, 1990). While women have a more communal orientation, with emphasis on sharing and discussing feelings, men have a more instrumental orientation, with emphasis on sharing and doing activities (Burleson et al., 1996). So, is the genetic architecture of social support different for men and women? To date, Agrawal et al. (2002) are the only authors to have addressed this question, and their findings are presented below. The second aim in this paper is to replicate their study with a sample twice the size. Within this second aim we address two questions:

Are the genes the same in males and females? Given Agrawal et al. (2002) found no such differences, and for complex traits generally differences are rare (Vink et al., 2012), we hypothesize no differences.

Is the extent of genetic or environmental influence the same in males and females? Previous research showed heritability to be generally higher in females than males for an array of psychological measures (Pilia et al., 2006). For social support, Agrawal et al. (2002) only observed differences on two of their six subscales (see Table 1), higher heredity in females for relatives and the reverse for confidants.

We consider seven separate members of the support network: spouse, twin, children, parents, relatives, friends and confidant. Previous research observed mean age and sex differences across these different members (Coventry et al., 2004; Olsen et al., 1991). In so doing, we extend the Agrawal et al. (2002) study of sex differences that distinguished just three sources: support from friends, relatives and confidants. We expect different results depending on who the twins are reporting on, as has been found previously (Agrawal et al., 2002; Kendler, 1997; Kessler et al., 1992): (a) For the twin subscale, where they report on each other, we expect higher C relative to the other subscales. (b) For the parents and relatives subscales, the twins are reporting on the same person, so we expect lower E. (c) For the spouse and children subscales, twins report on different people, so we expect higher E and thus lower C and A. (d) For friends and confidant, twins could be

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reporting on different people but could also report on the same person.

One of the 10 most replicated findings of behavior genetics is that the associations between environment measures and psychological traits are significantly mediated genetically (Plomin et al., 2016). Could the covariation among the subscales of social support be an exception to this rule, given social support has been traditionally a measure of the environment? The third aim of this paper is to use multivariate techniques to address a different question to that of the genetic architecture of each subscale: Is the covariation among the subscales genetic or environmental? Their correlations range from .19 to .79 and average .43 (Coventry et al., 2004). To date there have been no multivariate analyses of subscales of social support. We also explore sex differences on the genetic and environmental covariations of the subscales. Finally, we assess whether the genetic and environmental sources of covariation are best explained by one or multiple factors. As is the case for anxiety and depression, a single factor could account for all the genetic variance across the subscales. Alternatively, some subscales might be similar genetically but distinct genetically from other subscales.

Method

Participants

The participants comprise two twin cohorts from the Australian Twin Register (ATR): an older cohort born before 1964 and a younger cohort born 1964 to 1971. All provided written informed consent under study protocols approved by the Queensland Institute of Medical Research Human Research Ethics Committee.

The older cohort (Gillespie et al., 2003) participated in a mail survey in 1981 (Jardine & Martin, 1984) and comprised 7616 twin individuals (3808 twin pairs) aged 24–95 years ($M = 42.3$, $SD = 14.2$). In 1988–1989, they were mailed an extensive Health and Lifestyle Questionnaire. Follow-up of nonresponders was via telephone (up to five times), during which they completed an abbreviated interview obtaining basic demographic information. Twin pairs where one or both had died since participating in 1981 ($n = 139$) were excluded. A mailed questionnaire or telephone data were received from 6329 of the remaining 7338 participants, a response rate of 86%. Participants responding to at least one KPSS question totalled 5884, a response rate of 80%.

In 1989–1990, the Health and Lifestyle Questionnaire was mailed to the younger adult cohort (Gillespie et al., 1999; Heath et al., 2001), which included 8538 twin individuals (4269 twin pairs), aged 18–28 years ($M = 23.4$, $SD = 2.3$). Eighteen pairs were already in the older cohort, so were removed. The follow-up was the same. One thousand pairs were unable to be recontacted, which is understandable as they had been recruited 10 years earlier at school age. The response rate was 78% (5060/6502): lower because (a) it generally is for younger participants and (b) the older cohort had responded previously to the 1981 questionnaire. For participants responding to at least one KPSS question ($n = 3722$, the response rate was lower at 57%. This was because the last ~15% of responders were assessed with just an abbreviated telephone interview, which excluded the KPSS items because of the slightly lower return rate of the younger cohort.

Across both cohorts, 5821 males, mean age 30.4 ($SD = 12.6$) and 8297 females, mean age 33.5 ($SD = 14.2$), were able to be recontacted. There were 4100 pairs where both responded (58%) to one or more KPSS items (see Table 2).

Instruments

The Health and Lifestyle Questionnaire included the 19-item Kessler perceived social support (KPSS) measure (Coventry et al., 2004; Kessler et al., 1992, 1994). The first 18 items comprised three questions assessing respondents' belief that members of their social network would be willing to (a) listen to their problems, (b) understand the way they felt about things and (c) help if help were needed. The three questions were asked for six sources of support (spouse, twin, children, parents, relatives and friends) on a 4-point response scale; *Not at all* (0), *A little* (1), *Quite a bit* (2) and *A great deal* (3). The 19th item, referred to as *confidant*, asked 'Is there anyone in your life with whom you have a close relationship and can share your most private feelings?', with the response options Yes or No.

Coventry et al. (2004) report a factor analysis of the first 18 items of the KPSS with the current sample. The 19th item, *confidant*, was dropped from the analysis because of factorial complexity but is retained here as a stand-alone subscale, as used by others (Agrawal et al., 2002; Kendler, 1997; Kessler et al., 1992). The optimal factor solution in both males and females has seven factors: support from *spouse*, *twin*, *children*, *parents*, *relatives* and *friends* and *helping support* (i.e., perceived help from all six sources). Although Kessler et al. (1992) observes three factors (support from *spouse, other family/relatives* and *friends*) with the same KPSS and an all-female sample, a confirmatory factor analysis (with just females) shows their simpler factor structure is a significantly poorer fit ($\Delta\chi^2_{24} = 14{,}880$, $p < .001$). Possibly our larger sample allows us to detect the more elaborate factor structure (Coventry et al., 2004). Further, our factor structure is consistent with later research by Kendler et al. (2005) that also used the KPSS but with a similar factor structure to our own in males and females. The test-retest reliability, on a subsample of 879 twins who completed the questionnaire twice at a mean interval of 2.1 years, ranges from .55 to .72 (mean .64) with *confidant* an exception at .48 (Coventry et al., 2004).

Twins from the older and younger cohorts reported how close they were for: (1) sharing the same room, (2) having the same playmates, (3) dressing alike and (4) being in the same classes, with responses on a 4-point scale: *Never, Sometimes, Usually* and *Always*. Twins from the older cohort reported how often they had (1) seen and (2) contacted each other in the past nine years on a 7-point scale from *We live together* to *Not at all*. Twins from the younger cohort reported (a) if they were ever separated from their twin for more than a year (Yes or No) and (b) the age they started living apart. These eight items were used to assess the equal environments assumption (to be discussed).

Data Cleaning

To ensure responses referred to current members of the support network, responses were removed if participants reported (1) *spouse* support without being married (585),[1] (2) support from a deceased *twin* (17), (3) support from *children* without having any (163) and (4) support from *parents* when both were deceased (667). A support source was also deleted if participants had incomplete responses on any of the three items (help, listen and understand) comprising each source. Subscales were the mean of nonmissing responses on each factor and the KPSS score was the mean of nonmissing responses on all items (except the 19th item, which was dropped from the factor analysis).

[1]Individuals living with a partner were treated as married since it legitimately maximized the sample size for the genetic analyses of the *spouse* subscale.

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Table 2. Twin pair correlations (95% confidence intervals) by zygosity for each Kessler perceived social support (KPSS) subscale

Zygosity (*n* complete pairs)		Spouse	Twin	Children	Parents	Relatives	Friends	Help	Confidant	KPSS
MZ	*r*	.27	.62	.31	.51	.37	.32	.38	.38	.42
(2,009)	*c.i.*	(.20 to .34)	(.58 to .65)	(.23 To .38)	(.47 to .55)	(.32 to .41)	(.27 to .37)	(.33 to .42)	(.28 to .47)	(.38 to .46)
DZ	*r*	.15	.48	.14	.27	.19	.08	.24	.14	.29
(2,091)	*c.i.*	(.07 to .23)	(.45 to .52)	(.05 to .23)	(.22 to .32)	(.17 to .24)	(.03 to .13)	(.19 to .29)	(.03 to .24)	(.24 to .33)
MZff (1,377)	*r*	.27	.64	.26	.51	.35	.32	.36	.33	.42
	c.i.	(.19 to .35)	(.60 to .68)	(.17 to .35)	(.46 to .55)	(.29 to .40)	(.26 to .38)	(.30 to .41)	(.19 to .46)	(.37 to .47)
MZmm (632)	*r*	.27	.56	.46	.52	.43	.32	.42	.44	.43
	c.i.	(.13 to .41)	(.50 to .62)	(.32 to .57)	(.45 to .58)	(.35 to .51)	(.23 to .40)	(.33 to .50)	(.30 to .57)	(.35 to .50)
DZff (826)	*r*	.22	.53	.21	.31	.26	.12	.28	.15	.33
	c.i.	(.12 to .33)	(.48 to .59)	(.09 to .32)	(.23 to .38)	(.18 to .33)	(.04 to .19)	(.20 to .35)	(-.02 to .32)	(.26 to .39)
DZmm (381)	*r*	.12	.45	.11	.25	.15	.19	.23	.15	.32
	c.i.	(-.09 to .31)	(.35 to .54)	(-.16 to .36)	(.25 to .35)	(.03 to .27)	(.07 to .31)	(.09 to .36)	(-.05 to .35)	(.21 to .41)
DZfm (884)	*r*	.06	.47	.05	.25	.15	.00	.21	.10	.23
	c.i.	(-.07 to .19)	(.40 to .52)	(-.09 to .19)	(.17 to .32)	(.07 to .22)	(-.07 to .08)	(.12 to .29)	(-.06 to .27)	(.16 to .30)
n complete pairs		1,623	3,981	1,384	3,353	3,341	3,836	4,100	3,892	4,100
[a]Pairwise response rate		23%	56%	20%	47%	47%	54%	58%	55%	58%

Note: Twin pairs by zygosity; MZ, monozygotic males and females, DZ, dizygotic males and females, MZff, monozygotic females, MZmm, monozygotic males, DZff, dizygotic females, DZmm, dizygotic males, DZfm, opposite-sex dizygotic female-male.
[a]Computed as *n* per subscale/7,059. The 7059 includes all participants who were able to be recontacted but not those who were ineligible for a given item (e.g., participants who do not have children).

Table 2 presents pairwise response rates by subscale. Many participants did not have a spouse or children when surveyed so those subscales have lower pairwise response rates (23% and 20% respectively) compared to the other subscales (47–58%). Taking this into account, when the response rates for the spouse and children subscales were recomputed as a percentage of the participants who reported having a spouse or children, they were comparable to the other subscales.

Zygosity Diagnosis

Zygosity was diagnosed (a) by DNA for 316 pairs and (b) for all other participants using twins' answers to standard questions about similarity (95% accurate; Martin & Martin, 1975; Ooki et al., 1990), with inconsistencies resolved by (a) a phone call or, failing that, (b) a request for photos at different life stages.

Tests of the Equal Environment Assumption

The equal environment assumption (EEA) of twin studies requires the salient environment of monozygotic (MZ) pairs to be no more similar than for dizygotic (DZ) pairs. We emphasize saliency since we are only concerned with environments that affect outcome measures (i.e., social support). This paper tested for violations using the same method as Kendler et al. (1992), who observed no violations on the KPSS. On eight independent-group t tests, MZ pairs were significantly more likely ($p < .010$) to share environments than same-sex DZ pairs on six of eight environment variables (measured as the mean of Twins 1 and 2): MZ pairs reported more frequently sharing the same room and playmates, dressing alike and being in the same classroom, and they saw and contacted each other more frequently. The significant p values are, in part, a consequence of our large sample with effect sizes under 2% except for having the same playmates at 13%.

Saliency is assessed by testing whether the more similar environments of MZs are associated with more similar support in MZ pairs than DZ pairs. For the five zygosity groups shown in Table 2, each environment variable was correlated with the absolute value of within-pair differences on each KPSS subscale.[2] Only $^{8}/_{320}$ correlations were significant (using alpha =.001 to correct for multiple testing), all on the twin and parent subscales. On the parent subscale, the significant correlations were for environment variables explaining less than 2% of the MZ−DZ difference, so any inflation of the MZ correlation will be negligible. For the twin subscale, where MZs more frequently shared a playmate than DZs (effect size, 13%), the findings should be treated with caution as the violated EEA may inflate the genetic estimates.

Statistical Analysis

Skewness was severe for the subscales. For instance, for the spouse subscale (comprising three items each measured on a 4-point scale), the percentage of responses at each of the 10 levels from low (i.e. 0) through to high (i.e., 3) were 0.3, 0.5, 0.9, 1.7, 3.5, 6.1, 8.3, 14.2, 18.6, 46.0. Accordingly, the untransformed subscales were recoded into ordinal variables using the five thresholds that maximized equality between categories. By assuming an ordinal variable merely reflects thresholds on an underlying normal distribution of liability, it is possible to model the variance components of that continuous liability (Neale & Cardon, 1992). There are competing demands in determining the number of thresholds. Fewer thresholds reduce power (Neale et al., 1994), and more thresholds impede computational ability (Gillespie et al., 2000; Gillespie et al., 2003). Five thresholds best served these competing demands.[3]

The classical twin design (CTD) conceives variance as arising from four sources, additive (A) and dominant (D) genetic variation, common environment (C) and unique environment plus error (E). Using reared-together twin pairs, it is only possible to estimate three of these four parameters. But, when $r_{DZ}/r_{MZ} > {}^{1}/_{2}$, C > D and when $r_{DZ}/r_{MZ} < {}^{1}/_{2}$, D > C, we use this heuristic to fix the smaller of C or D to zero. This potentially biases the A, D and C estimates (Keller & Coventry, 2005) but, on average and for broad-sense heritability, the biases are not substantial (Coventry & Keller, 2005). In all models, the variance explained by age and sex (as necessary) was partitioned separately so their effects were removed.

Sex-limitation models[4] were fitted using standard methods (Neale & Cardon, 1992). The first estimated seven parameters (Model 1): A, C (or D) and E for both males and females, and r_g, which is the correlation of additive genetic effects shared by male–female DZ pairs. Twin modeling assumes DZ pairs share, on average, half their genes and if these are *same*, rather than *opposite*-sex pairs, r_g will approximately equal .5. However, for opposite-sex pairs, it is possible that the genetic effects that do exist are due to different genes in males than in females. If these genetic effects are completely different in males and females, r_g will be zero, and if they are the same r_g will be .5.[5]

The confidence intervals surrounding r_g indicated whether the same or different genes contributed to the genetic variation in males and females. If the upper confidence interval spanned .5, it suggested (a) the same genes acted in males and females (common effects sex-limitation model). If not, it suggested (b) a partially different genetic etiology, and if the lower interval spanned zero, (c) quite different. Confidence intervals spanning .5 and zero were diagnostic of limited power.

Males and females had the same magnitude of genetic and environmental influence if the fit of the model with A, C (or D) and E parameters equated for males and females (Model 2) was better than the fit with these parameters left free. All models were fitted to the raw data using Mx (Neale, 2004) with fits compared using the difference in −2log likelihood ($-2\Delta LL$), which has a chi-squared distribution with degrees of freedom equal to the difference in estimated parameters.

A Cholesky decomposition assessed the source of covariation among the subscales. It was run separately in males and females as attempts at a multivariate sex-limitation model failed. When fitting the model to all eight variables using the raw data, numeric problems occurred due to greater missingness on some subscales (detailed below). Instead, models were fitted to correlation and asymptotic covariance matrices. These were estimated separately for each zygosity group using LISREL 8.72 (Jöreskog & Sörbom, 2005). Asymptotic covariance matrices were needed in addition to correlation matrices because the current paper used categorical data and polychoric correlations.

[2]Spearman correlations were used as the absolute values of the within-pair differences were skewed.

[3]The univariate models were initially run with a greater number of thresholds (between 6 and 9 depending on the subscale) but encountered computational problems, as is commonly found. When comparing the parameter estimates from these analyses (when they actually ran) against estimates derived using five thresholds, the differences were negligible.

[4]Models where an additional male (or female) genetic parameter is added, which is limited in that the parameter is *uncorrelated* with the genetic effects of the female parameter, unlike the other male genetic parameter, which is not.

[5]This parameter can also be modeled as the correlation of just the 'additive genetic variance *shared*' between DZ pairs: ranging from 1 (*completely the same genes in males and females*) to zero (*completely different genes*). As distinct from the correlation of 'additive genetic variance' for DZs modeled here (and ranging from .5 to 0).

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Table 3. Univariate model fitting results for the Kessler perceived social support (KPSS) subscales that had no evidence of sex-limited effects

	Model	A	95% CI	C/D	95% CI	E	95% CI	Test-retest r^a
Spouse	ACE	.25	[.04, .33]	.02	[.00, .19]	.73	[.67, .80]	.72
Children	ADE	.25	[.00, .38]	.07	[.00, .38]	.68	[.61, .76]	.66
Parents	ACE	.49	[.36, .55]	.03	[.00, .13]	.49	[.45, .52]	.66
Relatives	ACE	.35	[.21, .41]	.02	[.00, .10]	.63	[.59, .68]	.58
Help	ACE	.27	[.13, .41]	.10	[.00, .22]	.62	[.58, .67]	.61
Confidant	ADE	.16	[.00,.43]	.22	[.00, .47]	.62	[.53, .72]	.48

Note: [a]From Coventry et al. (2004).

When using data matrices for a Cholesky instead of raw data, the matrices are listwise deleted. Some subscales (spouse and children) had substantial missingness. Rather than this dictating a diminished sample, a sample size is increased if separate data matrices are created according to different levels of missingness. The matrices are used in combination to derive estimates. A cluster analysis on missingness created the separate data matrices. It identified three mutually exclusive subgroups with different constellations of social support:

A family constellation ($n = 715$): participants with high support from *spouse* and *children* and lower support from *friend*s. They had complete data on all eight subscales.

A premarriage constellation ($n = 1940$): participants with high support from *parents* and *friends* and no support from *spouses* or *children*, where they had missing data. There was no missingness on the other subscales.

A parentless constellation ($n = 874$): young and elderly participants who had lost their *parents* and had higher support from *children* where they had them. They had missing data for *parents* or *relatives* and sometimes also for *spouse* or *children*, but all other subscales were complete.

Separate data matrices were created for each. Few participants had a different pattern of missingness to the above. Four subscales had no missingness (*twin, friend, helping* and *confidant*), and when estimating their covariations the matrices of the three subgroups contributed ($n = 3529$). Six subscales (*twin, friend, helping, confidant, parent* and *relative*) had no missingness for the first and second subgroups and the matrices of these subgroups estimated their covariation. Finally, all eight subscales had no missingness in the first subgroup, and it alone estimated their covariation. The male estimates were not sensible due to missingness on the *children* and *spouse* subscales. We report the estimates of just the first six variables (i.e., not the *children* and *spouse* subscales) in males.

The factor structures of A, C and E were simplified by running a factor analysis on the A, C and E correlation matricies of the Cholesky using the statistics program 'R'.

Results

An earlier paper of ours (Coventry et al., 2004) describes trends in the means of age and sex for the KPSS subscales. Here, mean differences between zygosity groups were assessed by equating thresholds, controlling for sex and age.[6,7] For all modeling, thresholds were equated wherever they were homogeneous.

[6]For the children subscale, child's age rather than participant's age was entered as the covariate as child age showed a marginally stronger association with the children subscale.

[7]Details on the models used can be obtained from the first author upon request.

The twin correlations were assessed for homogeneity across the five zygosity groups (MZ females, MZ males, DZ females, DZ males and DZ opposite sex), controlling for age and sex. Where they suggested a different genetic architecture by sex, sex-limitation models were fitted. Table 2 presents the twin correlations. We could not equate the male and female MZs and male and female DZs for the twin subscale ($\Delta\chi^2_2 = 6.38$, $p = .041$) suggesting the magnitude of genetic or environmental effects may differ by sex, and we could not equate the DZ male, female and opposite sex pairs for the friend subscale ($\Delta\chi^2_1 = 6.89$, $p = .009$) and *KPSS* ($\Delta\chi^2_1 = 4.01$, $p = .045$), suggesting genes may differ by sex. Accordingly, for these subscales only we fitted sex-limitation models.

Univariate Analysis with No Apparent Sex Limitation

Univariate models were run for the spouse, children, parents, relatives, helping and confidant subscales since no sex differences were apparent from the correlations. The results are presented in Table 3. The confidence intervals showed that all estimated C and D parameters could be dropped, supporting an AE model for each subscale.

Univariate Analysis with Evidence of Sex Limitation

The results of sex-limitation models run on the twin, friends and KPSS subscales are presented in Table 4. The r_g estimates were .39, .15 and .07, respectively. The confidence intervals for all spanned .5 and zero, suggesting limited power. Rather than concluding nothing more, we instead contrasted the extremes by comparing models with $r_g = .5$ and $r_g =$ zero to one with r_g free. For the friends subscale, the Akaike information criterion (AIC) showed fitting $r_g =$ zero (AIC = −1.99) optimal to fitting $r_g = .5$ (AIC = −1.03). Likewise for *KPSS*, $r_g =$ zero (AIC = −1.96) fitted better than $r_g = .5$ (AIC = −0.13). While this supports different additive genes by sex the power is limited.

The A, C and E estimates for males and females were equated, where possible, with the r_g parameter free to vary where the AIC suggested different genes by sex. Where it did not (i.e., the twins subscale) $r_g = .5$, consistent with the univariate models. The friends subscale estimated D in females but C in males, but given D was twice C we used D for both sexes. As seen in Table 4, there were no sex differences on twin, friend or KPSS.

To ensure no sex differences were overlooked in the univariate models of the spouse, children, parents, relatives, helping and confidant subscales of Table 3, sex-limitation models were also fitted (results not shown for parsimony). Consistent with the correlations, which were homogeneous by zygosity for these subscales, first, the AIC contrasts showed AICs were all more negative for

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Table 4. Results for Kessler perceived social support (KPSS) subscales that had sex-limitation models fitted

Model		Females			Males				$\Delta\chi^2$	Δdf	p
Twin		a^2_f	c^2_f	e^2_f	a^2_m	c^2_m	e^2_m	r_g			
1.	Free; A_f, C_f, E_f, A_m, C_m, E_m, r_g	.21	.43	.36	.23	.34	.44	.39			
1.	95% confidence intervals	.07 ~ .35	.30 ~ .55	.32 ~ .40	.01 ~ .42	.18 ~ .53	.38 ~ .50	.00 ~ .50			
2.	**$A_m = A_f$, $C_m = C_f$, $E_m = E_f$, r_g =.50**	**.24**	**.37**	**.39**	**.24**	**.37**	**.39**	.50	7.68	4	.104
2.	95% confidence intervals	.14 ~ .35	.28 ~ .45	.35 ~ .42	.14 ~ .35	.28 ~ .45	.35 ~ .42				
Friend		a^2_f	d^2_f	e^2_f	a^2_m	c^2_f/d^2_f	e^2_m	r_g			
1.	Free; A_f, D_f, E_f, A_m, C_m, E_m, r_g	.16	.16	.68	.25	.06	.68	.15			
1.	95% confidence intervals	.00 ~ .36	.00 ~ .37	.63 ~ .74	.00 ~ .40	.00 ~ .27	.60 ~ .75	.00 ~ .50			
2.	**$A_m = A_f$, $D_m = D_f$, $E_m = E_f$**	**.26**	**.05**	**.68**	**.26**	**.05**	**.68**	.00	1.04	3	.792
2.	95% confidence intervals	.07 ~ .36	.00 ~ .26	.64 ~ .73	.07 ~ .36	.00 ~ .26	.64 ~ .73	.00 ~ .26			
KPSS		a^2_f	c^2_f	e^2_f	a^2_m	c^2_m	e^2_m	r_g			
1.	Free; A_f, C_f, E_f, A_m, C_m, E_m, r_g	.19	.23	.58	.23	.20	.57	.07			
1.	95% confidence intervals	.03 ~ .35	.09 ~ .37	.53 ~ .63	.02 ~ .46	.08 ~ .38	.50 ~ .64	.00 ~ .50			
2.	**$A_m = A_f$, $C_m = C_f$, $E_m = E_f$**	**.20**	**.22**	**.58**	**.20**	**.22**	**.58**	.04	0.08	3	.995
2.	95% confidence intervals	.11 ~ .34	.10 ~ .29	.54 ~ .62	.11 ~ .34	.10 ~ .29	.54 ~ .62	.00 ~ .49			

Note: [a]Fixed. All models compared against Model 1. r_g additive genetic correlation between opposite sex dizygotic twin pairs. Bold type denotes best fitting model, determined by AIC where necessary (see results).

$r_g = .5$ than for $r_g = .0$, supporting the same additive genes by sex. Second, all the A, C or D and E estimates of males and females were able to be equated.

Multivariate Analyses

For the multivariate analysis, an ACE model was more applicable as no significant D was detected on any subscale. When estimated with multivariate modeling, the genetic and environmental architecture of each subscale (presented in Table 5 for the saturated models) differed from that of the univariate modeling in that, generally, for females, C was higher (by 12% on average) and E was lower (by 12% on average), while for males, C was again higher (by 11% on average) but A was lower (by 9% on average). We consider this further in the discussion.

The notable sex differences by subscale from the multivariate models were consistent with those at the univariate level (i.e., for twin and friend where sex-limitation models were fitted). The one exception was of confidant, where C was higher in females while A was higher in males. The multivariate estimates also allow us to compare males and females across all subscales. C was generally higher in females (range = 18 to 48%, mean = 33%; with the exception of friend at 7%) relative to males (range = 10 to 28%, mean = 19%). By contrast, the A estimates were similar in females (range = 6 to 34%, mean = 18%) and males (range = 14 to 31%, mean = 22%).

For the covariation among the subscales, the multivariate methods characterized a genetic and environmental architecture that was as follows: in females, when averaged across the eight subscales, additive genes accounted for 27% of the covariation, the common environment 34% and the unique environment 39%; an approximately even contribution from each. In males, however, slightly more of the commonality was attributable to the unique environment (48%), with less attributable to genetic variance (18%) but with the common environment still influential (35%).

Across the subscales, as expected, the variability in E was specific while for A and C it was more common; see Table 5. After extracting the main factors and rotating them, in females, just three A and three C factors explained 99% and 92% of the variability, respectively, and in males, just four A and two C factors explained 97% and 92% of the variability, respectively. However, beyond telling us that just a few A and C factors can account for almost all the variance, these factors turned out to be not meaningful in any theoretical or practical sense, so we consider them no further. A more parsimonious alternative was just a single factor for each. In males, these explained 52%, 78% and 51% of the variance for A, C and E, respectively, and in females, 77%, 59% and 46% of the variance, respectively. Thus, using a single A, C and E factor for all eight subscales allowed us to capture substantial portions of the variance in the KPSS.

Discussion

Our findings restate that social support should be no longer considered a purely environment variable. The broad-sense heritability estimates contributed significantly to variability on all eight subscales of the KPSS, explaining one-third (i.e. 32%) of the variability on average (range 21−49%, $SD = 9$) in the univariate models, consistent with earlier findings. (The multivariate estimates were generally lower and we discuss these later.) All the broad-sense heritability estimates comprised predominantly additive genes. While the MZ and DZ correlation suggested genetic dominance on some subscales, our univariate models failed to detect significant dominance, which is not surprising given the high additive/dominant correlation and the much larger samples required for adequate power (Martin et al., 1978; Neale et al., 1994). In any case, twin estimates of heritability from either ACE or ADE models are the least biased when interpreted as broad- rather than narrow-sense heritability (Coventry & Keller, 2005; Keller & Coventry, 2005).

Relative to behavior genetic studies generally, the unique environment explained substantial portions of variance, on average 64% (ranging from 49% to 73%, $SD = 8$) according to the univariate estimates (excluding here support from the *co-twin* since the

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Table 5. Path coefficients and percentages of variance for A, C and E from the saturated Cholesky decomposition of the Kessler perceived social support (KPSS) subscales. The subscales are presented in the order of entry

		Females									Males						
A	Factors	A_1	A_2	A_3	A_4	A_5	A_6	A_7	A_8	*% Var.*	A_1	A_2	A_3	A_4	A_5	A_6	*% Var.*
	Confidant	.24								*.06*	.45						*.21*
	Help	.21	.38							*.19*	.13	.35					*.14*
	Twin	.35	.29	.03						*.21*	-.02	.15	.46				*.24*
	Friends	.32	.33	-.21	-.01					*.26*	.24	.11	-.09	.26			*.14*
	Parents	.09	.53	.24	.04	.00				*.34*	.16	.34	.06	-.14	.39		*.31*
	Relatives	.36	.28	.03	.09	.00	.00			*.22*	.17	.46	.02	.16	-.03	.00	*.26*
	Children	.21	.13	-.06	.05	.00	.00	.00		*.07*							
	Spouse	.01	.29	.08	-.02	.00	.00	.00	.00	*.09*							
C	Factors	C_1	C_2	C_3	C_4	C_5	C_6	C_7	C_8		C_1	C_2	C_3	C_4	C_5	C_6	
	Confidant	.60								*.36*	.43						*.18*
	Help	.16	.49							*.27*	.23	.41					*.22*
	Twin	.13	.25	.59						*.43*	.49	.20	.06				*.28*
	Friends	.16	.06	-.12	.16					*.07*	.30	.23	-.17	.00			*.17*
	Parents	.15	.34	.03	-.26	.04				*.21*	.40	-.03	-.02	.00	.00		*.16*
	Relatives	.02	.39	-.01	-.11	-.11	.00			*.18*	.23	.11	.18	.00	.00	.00	*.10*
	Children	.41	.36	-.06	-.21	-.03	.00	.00		*.35*							
	Spouse	.36	.54	-.19	.16	-.01	.00	.00	.00	*.48*							
E	Factors	E_1	E_2	E_3	E_4	E_5	E_6	E_7	E_8		E_1	E_2	E_3	E_4	E_5	E_6	
	Confidant	.76								*.58*	.75						*.56*
	Help	.24	.69							*.54*	.17	.78					*.63*
	Twin	.30	.23	.45						*.35*	.07	.33	.00				*.48*
	Friends	.27	.42	.03	.65					*.67*	.17	.46	.10	.66			*.68*
	Parents	.12	.27	.23	.08	.53				*.43*	.08	.35	.26	.05	.57		*.52*
	Relatives	.38	.38	.08	.06	.19	.51			*.60*	.09	.52	.06	.09	.13	.57	*.63*
	Children	-.18	.36	.15	.11	-.01	.25	.42		*.44*							
	Spouse	.24	.10	.11	-.04	.05	-.11	.33	.47	*.42*							

Note: Age explained; for females, 14% of the variance on support from *children*, 2% on support from *parents* and 1% on support from a *twin*, *friends* and *relatives* and for males, 5% of the variance on *confidant* and 1% on *helping* support and support from *friends* and *parents*.

unique environment was low for reasons to be discussed). Importantly, when viewed alongside the test-retest reliabilities (rightmost column in Table 3), certainly no more than half this unique environment variance is measurement error. This suggests then that genuine (i.e., nonerror) unique environment effects, perhaps in the order of about one-third of the variance, do indeed account for differences in perceived social support. Thus, the results, in part, support the conventional view of perceived support as an environmental construct that can be manipulated for better health outcomes. Experimental work also supports this notion (i.e., Thorsteinsson & James, 1999).

The estimates of the unique environment varied depending on whether or not the twins shared the source of support, consistent with observations of the literature of Table 1 (Agrawal et al., 2002; Kendler, 1997; Kessler et al., 1992). Where twins unambiguously shared the shared the source of support, such as parents, the unique environment was lowest at .49% and successively increased for the partially shared sources of support (relatives, confidants and friends) to the sources of support that were unambiguously not shared, such as children at 68% and spouses at 73%. This is expected as the support twins receive from the same individual will be more similar than the support they receive from different people, where unique environment estimates are higher. In behavior genetics, twins often report on their own phenotype rather than a shared phenotype. In this regard, reporting on a shared source of support might be seen as an artefact (as discussed later for the twin subscale), with the consequence being higher familial aggregation.

The common environment effects from the univariate models were small (ranging from 0 to 10%) with the exception of the significant influences on support from the *co-twin* and *KPSS* (41% and 20%, respectively). However, with multivariate estimation, these common environment effects increased for most subscales. These increases meant that, surprisingly, C became greater than A for five of the eight female subscales and for three of the six male subscales. Overall, therefore, the multivariate approach presents a fairly different picture to that of the univariate models, as discussed later.

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Our findings were broadly in line with the comparable subscales from previous research. For instance, Kessler et al. (1992) observed (see Table 1), with just an all-female sample, CE, AE and ADE for support from spouse, friends and confidant, respectively. Similarly, for spouse, the MZ and DZ correlations suggested a CE model in females. For friend, the best fitting univariate model in females would have been an AE model had we chosen to fit this (we had some evidence of D). And for confidant, a univariate ADE model was clearly apparent from the point estimates.

The Effect of Different Constellations of Social Support

The broad aim of the multivariate modeling was to assess whether the covariation among the subscales was accounted for by genetic or environmental sources, a distinct question to that of the architecture of each individual subscale. However, the multivariate models also afforded a second pass at the architecture of the individual subscales, one arguably more accurate than the univariate estimates on account of the greater power afforded by the covariation among the subscales. Any discrepancies between the univariate and multivariate estimates of each subscale would warrant further scrutiny and would have implications for the architecture of the covariation among the subscales. We consider these discrepancies here.

For each subscale, the multivariate models generally estimated higher C (and lower A) than the univariate models. Several subanalyses (not shown) ensured these differences were not due to the multivariate analyses' use of (1) only same not opposite sex pairs, (2) a smaller sample due to listwise deletion and (3) asymptotic covariances rather than the raw data. Further, in all analyses, we partitioned the variability explained by age separately, so this did not contribute to any difference (see the footnote to Table 4). Finally, because we used categorical data rather than skewed continuous data we are comfortable that multivariate nonnormality was not responsible for any differences.

One plausible account of the discrepancy is that different constellations of social support inflated the multivariate but not univariate estimates of C. These constellations entail groups of participants with a different structure in their support network depending on their stage in life. The cluster analysis reported in the Methods section identified one such grouping set, comprised of three constellations (Family, Premarriage and Parentless). While correlated with age, these constellation effects would not be eliminated by entering age as a covariate since participants of the same age can be at different life stages.

The influence of these different constellations of social support would be via common environment effects specific to the different constellation within the sample used for the multivariate but not univariate analyses. To explain, take an example where different genes for a trait influence males and females: if we fit our model assuming the same genes influence males and females, the genetic estimates will be inflated. Like sex, specific constellations — such as the three mentioned above — represent differences across the sample. If common environment influences are specific to each constellation and we model these effects to be uniform across different constellations, the common environment effects will be inflated. We hypothesized that common environment effects specific to the different constellations inflated the multivariate estimates of the common environment. The univariate analyses would be impervious to these effects since these constellations only take form when considered in the context of all the sources of support.

Further analyses tested whether these effects of different support constellations could have inflated the estimates of C from the multivariate models. We reasoned that if we fitted the same multivariate model separately within each constellation, rather than for the whole sample, the estimates of C would be generally lower. This was because the C estimates within each support constellation would no longer be inflated by being collapsed with disparate C effects from other constellations. Accordingly, we estimated C separately for each of the three constellations then took the average C across these constellations weighted by sample size.[8] The estimates were indeed lower than those for the whole sample. As previously mentioned, for the whole sample the male and female estimates of C across each subscale were consistently higher (mean = .19, *SD* = .08; Table 5) than in the univariate analyses (mean = .04, *SD* = .04; Tables 3 and 4). By contrast, and as expected, in the by-constellation analyses, C estimates averaged 12% (*SD* = .06), and these were consistently (with one exception from 10) about midway between the univariate and whole-sample estimates. On balance, this provides support for our conjecture that constellation effects explain the higher C estimates from the multivariate (i.e., whole-sample) than univariate models. While this accounts for these findings, it is by no means firm evidence, and the topic warrants further scrutiny.

Given we suspect our multivariate estimates of the common environment were biased upwards by different support constellations, we placed greater weight on the univariate estimates, which show little variance explained by the common environment, a finding consistent with those found generally for behavioral genetics. What implications do these constellation effects have for the architecture of the covariation among the subscales? Had the multivariate estimates of each subscale been consistent with the univariate estimates, we could conceivably accept architecture for the covariation that was somewhat but not vastly different from the individual subscales (as the architecture of the covariation is a distinct question to that of the individual subscales). Since they were not consistent, we caution that the common environment effects influencing the covariation among the subscales could be similarly an artifact of the constellation effects.

Going forward, to more precisely address the contribution of the shared environment to social support there is now a pressing need to use family dyads beyond just the twin pairs used here. For instance, using the parents of these twins would allow dominant genetic and shared environment effects to be simultaneously estimated (Keller & Coventry, 2005), thereby reducing the possible bias in the estimates of the common environment we presented (Coventry & Keller, 2005). Further, this would establish whether the shared environment effects were a consequence of (a) familial transmission from parents to their children, as has been found for anxiety (Eley et al., 2015) and depression (Silberg et al., 2010) or (b) not transmitted and specific to the twin/sibling environment. This presents an important avenue for future research.

Sex Differences

In comparing males and females, the magnitudes of genetic and environmental influence were generally similar. Different genes in males and females were responsible for the genetic variation of some subscales (for the friends subscale and KPSS average) though the evidence for this was underpowered. If true, however,

[8]Based on five of the eight subscales since two (*spouse* and *children*) had data in just one of the three constellations, and a third (*twin*) had inflated estimates of C as detailed in the limitations.

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this sexually dimorphic additive gene expression is particularly noteworthy given that Vink et al. (2012) found such effects were rare for complex traits, and their methodology was identical to ours. However, our findings are plausible given (a) the evidence suggesting women are characteristically different from men in their social support (i.e., they share and discuss feelings, while men share activities; Burleson et al., 1996) and (b) studies using a different methodology that explore sex differences in specific genes routinely find sex differences (Ober et al., 2008; Trabzuni et al., 2013). How might such differences emerge? They are presumably a consequence of sexual selection toward adaptations that better aided just one sex in maximizing their reproductive success. For instance, the heterogeneous gene expression for thrill seeking (Vink et al., 2012) may have arisen with particular genes for this phenotype being more advantageous in men, thereby ensuring their expression in future generations of men but not women. Our evidence here suggests some loci for social support may act in a similar manner. Future studies, which have large samples for adequate power, will be well placed to further resolve this important question.

Limitations

These research findings should be evaluated in light of two limitations. First, for support from *co*-twin the correlations were higher than all other KPSS subscales. On all subscales, except twin, twins reported on the perceived behavior of a third party. The twin subscale was unique in that twin pairs reported on their perceived support from their co-twin, and vice-versa. This had the effect of inflating all twin pair correlations. Indeed, for the twin subscale, the mean correlation across zygosity was .53, relative to a lower range of mean correlations, from .19 to .37 on all the other subscales. This surely inflated familial aggregation; hence, the significance that we observed on the additive genetic and common environment estimates may be spurious and an artefact of 'reciprocal reporting' within the twin pair. Further, the twin subscale violated the EEA, and this might have increased the additive genetic effects and reduced common environmental influence. In sum, these effects would have both inflated the additive genetic influence.

Second, with a smaller sample of participants that reported support from children and spouses, for males, it was not possible to derive multivariate estimates for these subscales, which impeded any such comparisons.

Conclusion

On sex differences, in contrast to those generally found for complex traits, we find tentative support for heterogeneous gene expression in males and females for some subscales. This suggests future researchers would do well to include sex effects when considering the implications of perceived support for mental health. By the same token, our findings of a single A, C and E factor accounting for upwards of half of the variance suggest that while differences at the subscale level may emerge, the KPSS average scale alone will be appropriate for mental health research. The genetic influences reported here for what was once ostensibly an environmental variable suggest that associations with mental health are due, in part, to genetic commonality (i.e., Spotts et al., 2004; Wade & Kendler, 2000), and to the extent this is so, these associations will be noncausal. Finally, this research unveiled intriguing effects of different constellations of social support that biased upwards multivariate estimates of the shared environment. In instances where it is actually be possible to identify such constellations, behavior geneticists may benefit from knowing whether they have implications for their estimates.

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