When do contrast sensitivity deficits (or enhancements) depend on spatial frequency? Two ways to avoid spurious interactions

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Abstract

Studies across a broad range of disciplines–from psychiatry to cognitive science to behavioral neuroscience–have reported on whether the magnitude of contrast sensitivity alterations in one group or condition varies with spatial frequency. Significant interactions have often gone unexplained or have been used to argue for impairments in specific processing streams. Here, we show that interactions with spatial frequency may need to be re-evaluated if the inherent skew/heteroscedasticity was not taken into account or if refractive error could plausibly differ across groups or conditions. By re-analyzing a publicly available data set, we show that–when using raw contrast sensitivity data–schizophrenia patients exhibit an apparent contrast sensitivity impairment at low, but not high, spatial frequencies, but that when using log-transformed data or when using generalized estimating equations, this interaction reversed. The reversed interaction, but not the overall contrast sensitivity deficit, would disappear if groups were matched on visual acuity. However, matching groups in this way is probably only defensible if acuity differences arise from optical blur. These analyses reconcile seemingly discrepant findings in the literature and demonstrate that properly reporting contrast sensitivity interactions with spatial frequency requires accounting for refraction error and skew/heteroscedasticity.

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Abstract

Studies across a broad range of disciplines–from psychiatry to cognitive science to behavioral neuroscience–have reported on whether the magnitude of contrast sensitivity alterations in one group or condition varies with spatial frequency. Significant interactions have often gone unexplained or have been used to argue for impairments in specific processing streams. Here, we show that interactions with spatial frequency may need to be re-evaluated if the inherent skew/heteroscedasticity was not taken into account or if refractive error could plausibly differ across groups or conditions. By re-analyzing a publicly available data set, we show that–when using raw contrast sensitivity data–schizophrenia patients exhibit an apparent contrast sensitivity impairment at low, but not high, spatial frequencies, but that when using log-transformed data or when using generalized estimating equations, this interaction reversed. The reversed interaction, but not the overall contrast sensitivity deficit, would disappear if groups were matched on visual acuity. However, matching groups in this way is probably only defensible if acuity differences arise from optical blur. These analyses reconcile seemingly discrepant findings in the literature and demonstrate that properly reporting contrast sensitivity interactions with spatial frequency requires accounting for refraction error and skew/heteroscedasticity.

Keywords: contrast sensitivity, spatial frequency, visual acuity, heteroscedasticity, schizophrenia

Abbreviations : cs, contrast sensitivity; cpd, cycles per degree; SZ, schizophrenia; HC, healthy control; GEE, generalized estimating equations; PANSS, positive and negative syndrome scale;

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Contrast sensitivity–the inverse of contrast threshold–corresponds to how much contrast energy is needed to identify a target reliably. Deficits have been reported in glaucoma, glare, ocular hypertension, amblyopia, macular degeneration, dry eye, multiple sclerosis, Parkinson's disease, and schizophrenia (Pelli & Bex, 2013). Contrast sensitivity enhancements have been reported in individuals with major depression, seasonal affective disorder, and among those at clinical high risk for psychosis (Keri & Benedek, 2007; Wesner & Tan, 2006). Contrast sensitivity is also adversely impacted by antipsychotic medication (Kelemen et al., 2013) and reductions in retinal dopamine synthesis (Jackson et al., 2012). Many of these studies report contrast sensitivity alterations that are *non-uniform* across the spatial frequency spectrum. Such interactions have either gone unexplained or have prompted authors to postulate specific biological differences. For example, in chronically ill schizophrenia patients, poor contrast sensitivity at the lower end of the spatial frequency spectrum has led some to hypothesize that cells in the magnocellular channel may be selectively impaired (Butler et al., 2005; Revheim et al., 2014; Martinez et al., 2012). Here, we leveraged a publicly available data set (Zemon et al., 2020) to consider whether illness-specific non-uniform reductions in contrast sensitivity could be explained by the heteroscedasticity or rightward skew inherent to contrast sensitivity data (i.e., with the variance being the highest for low-to-mid-range spatial frequencies and lowest for high spatial frequencies). We further considered whether potential group differences in visual acuity might generate more of a deficit at higher spatial frequencies. This second possibility was taken seriously since even small amounts of refractive error have been shown to worsen contrast sensitivity with high spatial frequency stimuli (Charman et al., 1979; Keane et al., 2014; Keane et al., 2022) and since visual acuity deficits are commonly observed in schizophrenia (Keane et al., 2016; Zemon et al., 2020). We show that both factors, taken together, strongly influence the direction and the statistical significance of reported interactions. Our results reconcile seemingly conflicting findings in the contrast sensitivity literature (Butler et al., 2005; Revheim et al., 2014; Zemon et al., 2020; Keri et al., 2002) and provide guidance on how to report such results.

2. Methods

2.1. Participants

The patients and controls in this data set have already been fully characterized (Zemon et al., 2020; see also Table 1), but key details are repeated. Participants included 75 healthy controls and 68 patients with schizophrenia (n = 54) and schizoaffective disorder (n = 14). Participants were excluded if they met the criteria for alcohol or substance dependence within the last six months, abuse within the last month, or had any neurologic or ophthalmic disorders affecting contrast sensitivity. Participants had 20/32 or better corrected visual acuity at 4m based on the Logarithmic Visual Acuity Chart (Precision Vision, La Salle, IL). Patients were recruited from the Nathan Kline Institute for Psychiatric Research, and diagnoses were obtained using the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, 4th edition and available clinical information. Healthy controls were excluded if they had an Axis I psychiatric disorder. Figure 1 reproduces the demographic and clinical characteristics of the sample.

2.1. Measures and Procedures

The stimulus and procedural details have been described but are repeated for expository purposes. The mean background luminance was 100 cd/m^2, the viewing distance was 190 cm, and the presentation duration was either 33 or 500 ms. Horizontal sine-wave gratings with spatial frequencies of 0.5, 1, 4, and 7 cycles/degree (cpd) and a square-wave grating of 21 cpd were presented randomly on the left or right side of the screen. A two-down, one-up staircase procedure determined stimulus contrast (± .15 log-unit steps). Contrast sensitivity corresponded to the inverse of the contrast threshold, which itself was calculated as the mean of the contrast values of the last ten staircase reversals (Levitt, 1971).

	Patients (n=68)	Patients (n=68)	Controls (n=75)	Controls (n=75)	Group Comparison
Variable	Mean or percentage	*SD*	Mean or percentage	*SD*	*p*
Age at test (years)	39.1	10.1	36.2	11.3	.11
Gender (% Male)	79		65		.06
Age of First Hospitalization (years)	23.1	7.8			
Visual Acuity (logMAR)	.05	.09	-.06	.09	<.001
Participant Socioeconomic Status (SES)[1]	25.5	11.5	41.7	10.7	<.001
Parent Socioeconomic Status (SES)[1]	50.8	27.1	44.3	14.7	.11
Illness duration (years)	15.7	9.03			
Chlorpromazine equivalents (mg/day)	901.5	694.9			

PANSS, positive	19.6	6.0
PANSS, negative	17.9	4.2
PANSS, general psychopathology	35.4	6.2

TABLE 1 Demographic characteristics of patients (SZ) and controls (HCs), and illness characteristics of patients: age in years, visual acuity in logMAR, participant and parent socioeconomic status (SES), illness duration in years, chlorpromazine equivalents in mg, subscales of the Positive and Negative Syndrome Scale: positive, negative, general psychopathology (Data derive from Zemon et al., 2020).

[1]Instrument: Hollingshead Four-Factor Index of Social Status (1975).

2.2. Analysis

Contrast sensitivity data were submitted to a Type III sums-of-squares 2 (spatial frequency) x 2 (subject group) mixed-model analysis of variance (ANOVA); this was done once with the raw contrast sensitivity data and once again with the log-transformed data. In each case, a Greenhouse-Geisser correction was applied to account for violation of the sphericity assumption (Mauchly's test). In all cases, follow-up t-tests assumed equal variances unless a significant Levene's test required otherwise. Note that we simplified the analyses by examining only the two most extreme spatial frequency conditions–0.5 and 21 cycles per degree–since the former has been claimed to most clearly bias processing toward the magnocellular stream and the latter toward the parvocellular stream and since group interaction effects should be clearly observed at these endpoints (Butler et al., 2005). Note also that we confined our results only to the shortest stimulus duration (33 ms) since these stimulus types have been used in previous schizophrenia studies that sought to bias processing toward the magnocellular channel (Butler et al., 2005; Martinez et al., 2012; Revheim et al., 2014). Nevertheless, we also (and separately) analyzed the 500 ms stimulus duration data for completeness.

To corroborate the log-transformed results, we performed generalized estimating equations (GEE) analysis using the raw (non-transformed) contrast sensitivity data. The analysis incorporated a gamma distribution function with a log link (to reflect the relationship of mean and variance in contrast sensitivity data), robust estimators of the standard errors, a maximum likelihood estimation of the parameters, and an exchangeable correlation structure (since conditions were counterbalanced). GEE's advantage over generalized linear mixed models (GLMMs) is that it can handle heteroscedasticity, does not require correct specification of the covariance structure, makes fewer assumptions, and is more robust when only population-level (marginal) effects are desired (Pekár & Brabec, 2018).

To consider the role of visual acuity, we also conducted analyses that matched groups on acuity (Elliot, 2016) and included this variable as a covariate. We opted to remove high-acuity controls rather than lower-acuity patients since i) poor acuity may characterize the illness (Hayes et al., 2018; Shoham et al., 2021), ii) most healthy people with poor acuity never develop psychosis; and iii) it is inappropriate to add an illness-related covariate when the groups are not matched on the covariate (Miller & Chapman, 2001). Potential issues with interpretability of visual acuity confounds are further considered in the Discussion.

3. Results

Detailed side-by-side descriptions of the interaction effects for each analysis may be found in Supplementary Tables S1, S2, and S3. For the non-transformed short stimulus duration data (33 ms), there was a main effect of subject group (F (1,141)=22.4, p <.001,η_p^2=.14), spatial frequency (F (1,141)=637.2,p <.001 , η_p^2=.82), and an interaction (F (1,141)=12.7, p <.001, η_p^2=.083). Follow-up t-tests showed a larger contrast sensitivity

deficit at the low spatial frequency condition (t (141)=4.2,p <.001, *Hedges' g* =.70) versus the high spatial frequency condition (t (111.2)=3.7, p <.001,*Hedges' g* =.60), consistent with certain previous studies (Butler et al., 2005; Revheim et al., 2014).

Log transforms are often implemented because contrast sensitivity values are thought to vary according to a power law distribution and because such values can differ by order of magnitude across spatial frequencies. Log-transformations mitigate positive skew and heteroscedasticity and likely facilitate more accurate statistical inferences. We, therefore, re-ran the ANOVA on the log-transformed data. There was a main effect of spatial frequency and subject group, as before, but the direction of the significant interaction *reversed* (F (1,141)=41.9,p <.001, η_p^2 =.229; F (1,141)=1861.3,p <.001, η_p^2 = .930; F (1,141)=4.2,p =.04, η_p^2 =.029). Follow-up t-tests revealed more pronounced deficits at the high spatial frequency condition (t (141)=5.2, p <.001, Hedges' g=.86) versus the low spatial frequency condition (t (120.3)=4.6,p <.001, *Hedges' g* =.77).

Despite the nearly ubiquitous use of log-transforms throughout the life sciences, some have found fault with this practice because, for example, the magnitude of skew can be equal and opposite after the transformation and because parameter estimates can have more significant standard errors after the transformation (Feng et al., 2014). Generalized estimating equations (GEE) have been recommended as an alternative because they can flexibly account for differences in variance and rightward skew and are robust to the misspecification of the covariance structure (Feng et al., 2014; Pekár & Brabec, 2018). Our GEEs revealed results that were qualitatively the same as the ANOVAs with the log-transformed data: patients exhibited contrast sensitivity deficits that worsened from low to high spatial frequencies (Wald Chi-square (1) = 4.87, B =-.419, SE_B =.19, p =.027).

<Fig. 1. here>

Figure 1. *Contrast sensitivity results for controls (green) and patients (red) for briefly presented gratings (33 ms)* . (A, B) When using non-transformed data, patients exhibited worse contrast sensitivity deficits at low versus high spatial frequencies. (B) Such an interaction also emerged when using a subset of controls (n=34) that were approximately matched to patients on visual acuity; (C, D) When the data were log-transformed, the sign of the interaction reversed. However, this interaction disappeared when using the acuity-matched set of controls.

A critical caveat is that patients' acuity differed from controls by about one line on an eye chart (t (141)=7.1,p <.001, *Hedges' g* =1.2, logMAR difference =.107). To consider whether acuity differences could explain the spatial frequency interaction, we simply removed healthy controls whose acuity was better than 20/20 (logMAR<0) so that the remaining controls (n =34) were approximately matched to patients on this variable (t (99.5)=1.9, p =.06, Hedges' g=.34, logMAR difference =.03). An ANOVA on the logged data revealed main effects of spatial frequency and subject group, but no interaction (spatial frequency: F (1,100)=1467, p <.001;η_p^2 =.94; group: F (1,100)=12.9,p <.001, η_p^2 =.11; interaction:F (1,100)<0.01, p =.99, η_p^2<.00). The GEE also revealed main effects of spatial frequency and group (B =-2.91, SE_B =.22, Wald Chi-Square(1)=177.15, p <.001; B =-.26,SE_B =.12, Wald Chi-Square(1)=4.48,p <.05), with no interaction (B =-.025,SE_B =.24, Wald Chi-square(1)=.01,p >.9). There was also no interaction when logMAR acuity was added as a covariate to the ANOVA (spatial frequency:F (1,99)=108.32, p <.001, η_p^2=.522; group: F (1,99)=11.00, p =.001, η_p^2=.100; interaction: F (1,99)=2.69, p= .10, η_p^2=.026) or to the GEE (B =-.059, SE_B =.26, Wald Chi-square(1)=.05, p >.8).

<Fig. 2. here>

Figure 2. *Contrast sensitivity results for controls (green) and patients (red) for longer presentation times (500 ms)* . Results were qualitatively the same as with the short presentation data (33 ms), suggesting, again, that spatial frequency interactions depend on whether the data were log transformed and whether groups differed in visual acuity.

To show that these results generalize to other viewing conditions, we also considered data from the longer presentation condition (500 ms). The interactions and main effects were similar to before (see Figure 2;

Tables S3). In particular, using all subjects, the interaction was significant in opposite ways depending on whether the log-transform was applied or not (raw data: F (1,141)=7.6, p =.006,η_p^2 =.05; log-transformed: F (1,141)=17.7,p <.001, η_p^2=.11). Using the matched subsample of controls, we found that the interaction showed reduced contrast sensitivity deficits at higher spatial frequencies before but not after the log-transform (raw data: F (1,100)=4.7,p =.03, η_p^2=.05; log-transformed:F (1,100)=2.6, p =.11, η_p^2=.03). In each of the above cases, there were main effects of group and spatial frequency (all$p$ <.001; all η_p^2>.14). GEEs yielded results that were qualitatively the same as the log-transformed results with one exception: using the subsample matched on acuity, there was now an interaction with spatial frequency such that patient deficits tended to worsen at the higher spatial frequency (B =-.436,SE_B =.19, Wald Chi-square(1)=5.26, p =.02). However, adding logMAR as a covariate would nudge the interaction out of significance territory (B =-.37, SE_B =.20, Wald Chi-square(1)=3.7, p =.06). Moreover, if we were to more closely match groups on acuity, then the interaction would likely be pushed further from significance by reducing the high spatial frequency group difference. To summarize, the results for the long presentation condition were largely the same as the short presentation condition.

Our focus in the study was on spatial frequency interactions. However, it is worth noting that–in all the foregoing analyses, including acuity matching–patients had worse contrast sensitivity than controls (all$p$ <.05). If we were to remove patients whose vision was worse than 20/25–so that the groups would become even more closely matched on logMAR acuity (M_{HC}=.02, M_{SZ}=.02, p =.96, Hedges' g=.01; n =34 SZ, 58 HC)–the log-transformed ANOVA results would again reveal a strong group deficit in the short duration condition (η_p^2>.10, p <.004) and the long duration condition (η_p^2>.11,p <.004) with or without a logMAR covariate.

4.0 Discussion

We have shown that apparent non-uniform reductions in contrast sensitivity can be explained by the heteroscedastic/skewed distribution of the contrast sensitivity data and by group differences in visual acuity. Our analysis gives reason to be cautious when interpreting interactions with spatial frequency in past studies; it also explains why the log-transformed data of Zemon et al. (2020) and Keri et al. (2002) yielded interactions that were opposite in sign to the interactions reported in certain previous studies (Butler et al., 2005; Martinez et al., 2012; Revheim et al., 2014) that did not transform the data.

Note that the pitfalls identified above have been discussed in other corners of science. For example, in the memory literature, it has been known that some "removable" interactions in two-way designs arise from non-linearities in the dependent variable (e.g., response probability) and can often be abolished by applying a transformation that conserves monotonicity (Wagenmakers et al., 2012; Loftus, 1978). In the vision literature, refractive errors have been shown to compromise contrast sensitivity at high but not low spatial frequencies, with smaller but still detectable effects in the mid-spatial frequency range (Keane et al., 2014; Keane et al., 2022; Charman & Heron, 1979; Johnson & Casson, 1995). Despite the long-standing nature of these problems, non-log-transformed data and their interactions continue to be discussed and interpreted in cognitive science, behavioral neuroscience, and vision science (Arnold et al., 2016; Jackson et al., 2012; Abrahamsson & Sjöstrand, 1986). Likewise, refractive error is often not regarded as a potential confound even when group differences increase with spatial frequency (Keri et al., 2002; Wesner & Tan, 2006).

While our focus was on spatial frequency interactions, our re-analysis also demonstrated that patient contrast sensitivity deficits could not be erased simply by matching groups on visual acuity. This shows that not matching groups on visual acuity can be a missed opportunity; when groups are well-matched in this regard, contrast sensitivity deficits (or enhancements) can be more definitively established.

Limitations and suggestions for reporting interactions with spatial frequency

A limitation is that we do not know why groups differed in acuity; thus, it is not yet clear whether it is

appropriate to match groups on acuity in the way we have done. On the one hand, at least one study has found that people with schizophrenia less often visit an optometrist (Viertiö et al., 2007; see also, Silverstein & Rosen, 2015), suggesting that poor contrast sensitivity at higher spatial frequencies may arise from not having appropriate eyewear (Zemon et al., 2020; Keri et al., 2002). This possibility should be taken seriously because optical blur within the "normal" 20/20 range can diminish sensitivity to Gabor elements with a frequency as low as four cycles/degree (Keane et al., 2022). On the other hand, people with anti-NMDA receptor encephalitis– a condition that symptomatically resembles schizophrenia and that attacks the same receptor that is commonly implicated in schizophrenia (Beck et al., 2020; Singh et al., 2022)–have worse acuity than matched controls, especially for more severe bouts of the infection (Brandt et al., 2016). Thus, either uncorrected refractive error, neural factors, or some combination could worsen contrast sensitivity deficits at higher spatial frequencies in schizophrenia. The same conclusion may hold for other special populations. For example, individuals of advanced age may have impaired acuity due to a combination of neural factors and optical under-correction (La Fleur & Salthouse, 2014; Liou et al., 1999).

Interactions with spatial frequency can be properly reported in a few ways. First, as may already be obvious, log-transforms can approximate homoscedastic, normal distributions, and generalized estimating equations may provide an even better way to model such data (Prekár & Brabec, 2018; Feng et al., 2014). Boxplots or histograms could reveal unexpected data distributions (such as those in Fig. 1B). To avoid confounds with optical blur, an optometrist could measure and correct acuity beforehand so that all subjects have their best corrected visual acuity at the time of testing (BCVA). Note that some investigators mistakenly use the term "BCVA" to refer to habitual acuity rather than optimal acuity (Elliot, 2016). However, only the latter can remove confounds associated with refractive error since many individuals with contacts or glasses will have out-of-date prescriptions. If groups cannot be matched on BCVA, this would be informative as it would indicate a neural origin to poor acuity and argue against any further matching based on acuity. If providing optimal correction to every subject is impractical, refractive error could instead be quantified with an auto-refractor. Portable auto-refractors generate spherical equivalent refractive error estimates that are similar to those of subjective refraction and retinoscopy with or without cycloplegia (Ciuffreda & Rosenfeld, 2015). In this approach, subjects with excessive refractive error could be excluded (e.g., >0.5 diopters, roughly equivalent to 20/30 vision), and subject groups could then be matched on refractive error in a post-hoc analysis, if not in the overall sample. Either way, refractive error must be considered before interpreting interactions with spatial frequency.

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