First, while acknowledging that we excluded alternative explanations of our findings, such as the potential impact of depressive symptoms, Dr. Hubbeling rightly points out that the stimulus example used in our article involved a disgusting social norm violation and wonders whether all of the transgression behaviors used in the study involved themes of disgust. Dr. Hubbeling then considers whether such disgust content could explain the increased insula response observed in the patients with generalized social phobia (group main effect) across the three transgression types. This is an interesting point that we had failed to consider in our paper. Of the 26 embarrassing transgressions, four involved themes of disgust, while the remaining 22 involved non-disgust themes (e.g., forgetting to tip a waiter for good service or accidentally hanging up on a friend during a phone call). Thus, it seems unlikely that disgust content directly contributed to the observed group difference in the insula in our study, although we do not rule out a potential role of disgust in other generalized social phobia-related studies. In this regard, it is also worth noting that we did not observe any group-by-transgression interaction within the insula, a result we might have expected if disgust had differentially affected the two groups, given that the behaviors categorized as no transgression, as indeed the naming implies, never involved any disgust behaviors.

A second point made by Dr. Hubbeling is that the stimuli used in fMRI studies should be made publicly available. We completely agree with this point and confirm that the stimuli used in the study are available by request from the corresponding author (K.S.B.).

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The authors' disclosures accompany the original article.

This letter (doi: 10.1176/appi.ajp.2011.10121800r) was accepted for publication in February 2011.

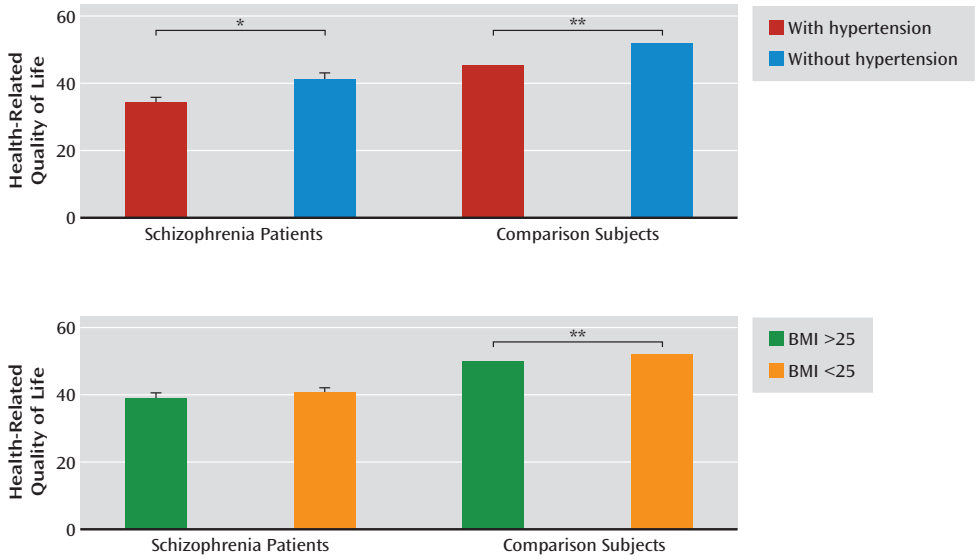
Impact of Hypertension and Body Mass Index on Quality of Life in Schizophrenia

TO THE EDITOR: We were pleased to read the article by Joseph I. Friedman, M.D., and colleagues in the October 2010 issue of the *Journal* (1). The article highlights the negative impact of individual cardiovascular risk factors, particularly hypertension, on memory performance in patients with schizophrenia, and suggests that proper treatment of these risk factors may improve cognitive outcomes in these patients.

Pronounced memory deficits have been linked to low quality of life in patients with schizophrenia (2). In recent years, the importance of predictors of quality of life on treatment outcomes among these patients is being recognized (3). We analyzed quality of life among patients with schizophrenia who had hypertension and a body mass index (BMI) >25 as reported in the National Epidemiologic Survey on Alcohol and Related Conditions (N=43,093), a large representative U.S. survey. This survey assessed substance use and mental and physical disorders, including history of schizophrenia, as well as self-reported hypertension and a high BMI in the past 12 months.

We measured quality of life using the 12-item Short-Form Health Survey, a norm-based general health scale, for eight subgroups: participants with schizophrenia with and without hypertension (N=134 and N=252, respectively) or with and without a BMI >25 (N=251 and N=125, respectively) and healthy comparison subjects (participants without schizophrenia) with and without hypertension (N=9,002 and N=33,705, respectively) or with and without a BMI >25 (N=16,983 and N=24,295, respectively). We adjusted all measures for age, gender, education, and ethnicity in all models. We included the vascular risk factor of interest (hypertension or elevated BMI) and the presence or absence of a schizophrenia diagnosis in

FIGURE 1. Health-Related Quality of Life for Patients With Schizophrenia and Comparison Subjects With and Without Hypertension and With and Without a Body Mass Index (BMI) Over 25



*p<0.001. **p<0.0001.

the main effect model. The interaction between a schizophrenia diagnosis and the vascular risk factor was entered in a second step. We used Taylor linearization to adjust standard errors of means of estimates for complex survey sampling design effects including clustering data, and used the REGRESS procedure in the SUDAAN software package (4).

Hypertension, but not a BMI >25, had a significant negative impact on quality of life in patients with schizophrenia (Figure 1). We found no significant interaction between schizophrenia and a BMI >25 on quality of life.

Friedman et al. (1) stress the importance of addressing individual cardiovascular risk factors in patients with schizophrenia. The dissociation between the effect of hypertension and a BMI >25 on quality of life in these patients is in line with this. Our findings stress the importance of screening for and treating hypertension in patients with schizophrenia, given its association with a poorer quality of life in this patient population.

References

1. Friedman JI, Wallenstein S, Moshier E, Parrella M, White L, Bowler S, Gottlieb S, Harvey PD, McGinn TG, Flanagan L, Davis KL: The effects of hypertension and body mass index on cognition in schizophrenia. Am J Psychiatry 2010; 167:1232–1239
2. Ranganath C, Minzenberg MJ, Ragland JD: The cognitive neuroscience of memory function and dysfunction in schizophrenia. Biol Psychiatry 2008; 64:18–25
3. Narvaez JM, Twamley EW, McKibbin CL, Heaton RK, Patterson TL: Subjective and objective quality of life in schizophrenia. Schizophr Res 2008; 98:201–208
4. Research Triangle Institute: Software for Survey Data Analysis (SUDAAN), version 9.0. Research Triangle Park, NC, Research Triangle Institute, 2004

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Dr. Le Foll has received research grants from Pfizer and has performed consulting work for Richter Pharmaceuticals. The remaining authors report no financial relationships with commercial interests.

This letter (doi: 10.1176/appi.ajp.2011.10101470) was accepted for publication in March 2011.

Corrections

At the time the article "Imaging Dopamine Transmission in Cocaine Dependence: Link Between Neurochemistry and Response to Treatment" by Diana Martinez, M.D., et al., was published online on March 15, 2011, the bar graphs in figure 2 were labeled incorrectly: the Responders and Nonresponders bars were flipped. The bars on the left for the nondisplaceable binding potential in the baseline condition and following methylphenidate actually represent the responders and the bars on the right represent the nonresponders. This change was made for the article's online posting on March 24, 2011, and for the article's print appearance in the June 2011 issue and for its online posting as part of that issue.

At the time the article "Association of Schizophrenia in 22q11.2 Deletion Syndrome and Gray Matter Volumetric Deficits in the Superior Temporal Gyrus," by Eva W.C. Chow et al., was published online on March 1, 2011, the first paragraph in the Results section misreported the mean age at time of scan for one of the study groups. The final sentence of the paragraph should have read as follows:

"The mean age at time of scan in the nonpsychotic group was 27.8 years (SD=10.1)."

This change was made for the article's online posting on April 7, 2011, and for the article's print appearance in the May 2011 issue (p. 524) and for its online posting as part of the issue.