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# Where Have All the Women Gone? Participant Gender in Epidemiological and Non-epidemiological Research of Schizophrenia

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## Abstract

Though archival literature states that schizophrenia occurs equally in males and females, recent epidemiological studies report higher incidence of schizophrenia in men than women. Moreover, there is longstanding evidence that women may be under-represented in non-epidemiological research literature. Our first goal was to quantify gender ratios in non-epidemiological research published in 2006. Secondly, we sought to investigate which factors contribute to high numbers of men in research studies. Our final goal was to compare gender ratios in non-epidemiological schizophrenia research to reported incidence rates. In a recent meta-analysis of incidence, there were 1.4 males for each female with schizophrenia. In non-epidemiological studies of the schizophrenia patients, there was an average of 1.94 men for every woman. Although the degree to which men outnumbered women varied according to study type and region of study, research studies included more men than women across all investigated variables. Either the incidence rates are higher for men than has previously been reported or women are less visible in research settings than in the greater community. Importantly, the discrepancy between gender ratios in epidemiological and non-epidemiological research is consistent. However, specific, identifiable factors are present when male participants are greatest, suggesting that many research environments yield a higher number of men. Thus much of our understanding of the illness and its treatment is based on research conducted disproportionately with men.

### Keywords

SCHIZOPHRENIA; EPIDEMIOLOGY; GENDER

## 1. Introduction

Recent epidemiological literature has shown unequal incidence of schizophrenia for men and women. Multiple meta-analyses found that approximately 60% of those who develop schizophrenia are men (Aleman, Kahn, & Selten, 2003; McGrath, Saha, Chant, & Welham,

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2008; McGrath et al., 2004). This is not clearly the result of a systematic bias in diagnostic systems. Rather, it appears that more males get ill. Younger male onset is one of the explanations evoked for higher male incidence (Hafner et al., 1989). In earlier studies that relied on the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychiatric Association, 1980), this could certainly be true since women who developed schizophrenia after age 45 were excluded and thus incidence - the number of new cases per year - of schizophrenia was biased towards men. Yet the recent meta-analyses used the DSM-III-R or later editions and the comparable International Statistical Classification of Diseases and Related Health (ICD) (World Health Organization, 1992), which removed the age "cutoff," and the gender imbalance endured.

However, this disparity seems to be exaggerated in the non-epidemiological research literature (e.g., concerned with features of the illness, treatment efficacy, cognitive processes, and neuroimaging), especially in recent decades. In non-epidemiological studies published from 1974 to 1976 in the Archives of General Psychiatry, Journal of Abnormal Psychology, and Journal of Nervous and Mental Disease, -59% of participants with schizophrenia were male (Wahl, 1977). However, in a later review of studies published in similar journals from 1985 to 1989, twice as many male as female patients were research participants (Wahl & Hunter, 1992), greatly surpassing the rates predicted by contemporary epidemiological research. Thus, although epidemiological and non-epidemiological research show greater proportions of men than women, this disproportion has become more pronounced in non-epidemiological research. Possible explanations for an imbalance of male and female schizophrenia patients in the non-epidemiological research literature include greater numbers of men among chronic patients (Häfner, 2003; Lewine, Burbach, & Meltzer, 1984), gender proportions differing by country (Bhatia, 2006; Jablensky et al., 1992), differential mortality (Phillips, Yang, Li, & Li, 2004), and effects of research agenda. To update this research and to investigate possible moderators of the effect, we analyzed the numbers of men and women with schizophrenia appearing in various research studies. Furthermore, as previous publications considered epidemiological and non-epidemiological research independently, we tested for statistical differences between these two literatures. We hypothesized that there would be fewer females in non-epidemiological schizophrenia research than expected from incidence rates. If true, this would suggest that the non-epidemiological literature is weighted toward male patients and may not generalize fully to female patients.

## 2. Methods

### 2.1 Epidemiological Comparative Value

As our baseline for comparison, we chose the incidence rate of 1.4 male schizophrenia patients to every female patient, or 58% males, taken from the most recent and complete schizophrenia meta-analysis (McGrath et al., 2008). We used this incidence value rather than estimated prevalence (i.e., 1.1 or 52% male [McGrath et al., 2008]) because it is a more consistent epidemiological measure (Babigian, 1980), supported by a more extensive body of literature, and because it was a more conservative baseline for our purposes. As an example of the discontinuity between incidence and prevalence estimates, a recent large-scale prevalence meta-analysis found no difference in the number of males and females diagnosed with schizophrenia (Saha, Chant, Welham, & McGrath, 2005), suggesting that different types of studies are included in prevalence analyses.

### 2.2 Non-epidemiological studies

A review was conducted of all non-epidemiological research articles published on schizophrenia in 2006 in the following seven high impact psychiatric journals: Archives of General Psychiatry, American Journal of Psychiatry, British Journal of Psychiatry, Biological

Psychiatry, Molecular Psychiatry, Schizophrenia Research, and Schizophrenia Bulletin. Authors J.Ge. and J.L. separately reviewed articles for relevance and derived values for analysis. Where the necessary values were vaguely or incompletely stated, they were resolved through consultation. Of 1969 articles published in those journals in 2006, 1341 were reviews, editorials, case studies, did not investigate schizophrenia patients, or did not indicate the number of male or female patients. Of 304 remaining articles, thirty-five studies of postmortem patients, Veterans Administration facilities (military hospitals), or single gender were excluded. Eleven articles investigated prodromal populations and did not include demographic data for those who have "converted" to schizophrenia after the prodromal phase, as participant counts in this study are of diagnosed- and not high-risk- cases. Ten studies appeared to involve samples that overlapped with other studies. Lastly, we required that included studies use relatively common diagnostic criteria (i.e. DSM-III-R or more recent; ICD 8 or more recent), report data on schizophrenia independently from general psychosis, and not only consider subtypes of the schizophrenia spectrum; 28 studies did not meet these inclusions criterion. After exclusions, 220 articles (referenced in Appendix 1) were analyzed. The proportion of male participants, calculated as number males divided by number participants (male plus female), was used in our analysis.

We coded the non-epidemiological articles according to seven multi-level categorical variables. 1. The journals investigated are listed above; 2. Study types were a) functional neuroimaging (fMRI, MEG, PET, EEG), b) anatomical (using imaging techniques to assess brain structure or chemistry), c) cognitive (examining performance on cognitive tasks), d) genetic (reporting results of various genetic and familial liability analyses), e) psychosocial (assessing psychosocial interventions, symptoms, and diagnostic criteria), and f) pharmacological (assessing different pharmacological manipulations); 3. The institutional settings were a) inpatient, b) outpatient, and c) mixed inpatient/outpatient populations; 4. The illness stages were a) prodromal, b) first-episode, c) first-admission, and d) lifetime; 5. Geographical breakdown was by continent and included: a) North America, b) Europe, c) Asia, d) Australia, and e) multiple sites; 6. We recorded the diagnostic system a) DSM (edition III-R or later) or b) ICD (edition 8 or later); 7. Diagnoses were a) schizophrenia only, b) schizophrenia or schizoaffective disorder, and c) schizophrenia spectrum. Additionally, two continuous variables, namely the mean age of participants, and the sample size were collected. We attempted to analyze refusal rates and information on the diagnostic process, but there were insufficient data.

### 2.3 Statistical analysis

Current research has many characteristics of meta-analysis. However, our focus is not the experimental endpoint of a group of studies but, rather, a statistics that is possibly ancillary (i.e., gender proportion), and thus unrelated to the endpoints of the separate studies considered in this review. Therefore, the assumptions underlying typical meta-analysis are not met (Hunter, 2004). In place of standard meta-analyses, we took the following straightforward statistical approach. The first set of analyses considered only the non-epidemiological data. We performed one-way ANOVAs followed by post hoc t-tests of significant findings. Regression coefficients determined whether the age of participants or sample size affected the proportion of male participants. Second, we compared the non-epidemiological data to the expected gender proportions, as defined by McGrath (2008), in t-tests for single means. We checked for normality for the distributions of all the proportions, and found no meaningful departure. We also applied the standard arc-sine transformation to the proportions and found no meaningful differences between those inferences and those obtained using just the t-test. Moreover we checked the analysis using chi square tests of equality and found no departure from the t-test results. The central fact of the proportions data is that virtually all are approximately centered in the interval [0.50, 0.60], in which it is known that the t-test using

normality is sound (except when the data are highly skewed, which was clearly not the case here).

## 3. Results

### 3.1 Non-epidemiological Research Literature

The 220 articles from the non-epidemiological research literature included 147,725 males and 104,853 females. Thus, the non-epidemiological research literature is 66% male and 34% female [male proportion= $M$=0.66, SD=0.12, N=220], translating to an average proportion greater than the epidemiological baseline. We analyzed articles from Schizophrenia Bulletin and Schizophrenia Research separately from the other five journals in order to rule out the possibility that gender ratios in articles published in journals focusing specifically on schizophrenia might be unrepresentative. However, there were no differences in effect or levels of significance. In order to directly compare our findings to previous reports (13, 14), we separately considered the articles from the Archives of General Psychiatry. Table 1 reports our findings alongside those of Wahl (1977) and Wahl & Hunter (1992).

### 3.2 Comparing Epidemiological & Non-epidemiological Research

In epidemiological studies, 58% of those diagnosed with schizophrenia were men, while 66% of non-epidemiological research participants were men. We examined whether the difference between epidemiological and non-epidemiological literature was significant. A t-test for single means confirmed that the difference was significant, with proportions in non-epidemiological research being consistently higher [$t(219)=9.64$, $p<0.001$].

### 3.3 Moderating Variables in Non-epidemiological Research

Of the 220 articles reviewed, only 11% had an equal or greater number of female than male participants (16 articles had more women than men and eight articles had a one-to-one gender ratio).

The remaining analyses investigated the seven multi-level categorical variables (identified in Methods). Table 2 summarizes findings for the first five of these variables. As seen in the table, there were numerical differences of male participants exceeded that of females in every journal, in every study type, in every type of sample, institution, and continent. Critically, in most cases, the male proportion in the non-epidemiological studies significantly exceeded the imbalance that would have been expected based on the baseline incidence value taken from the epidemiological literature. Indeed, 19 of the 25 post hoc t-test comparisons were significant, even after control for multiple comparisons, suggesting a clear and consistent excess of males in non-epidemiological research relative to the value expected based on epidemiological findings.

Though the majority of comparisons favored our hypothesis, there were exceptions, as follows. The category of genetic articles did not yield a significant result. This group of studies was consistent with the proportion of 0.58from the epidemiological literature (McGrath et al., 2008). In addition, gender proportions for articles that investigated pharmacological topics, were published in Molecular Psychiatry, looked at converted cases in prodromal samples, or were conducted in Asia, Australia or in multiple locations did not differ significantly from incidence estimates. Note that in nearly all of these cases where significance was not maintained following correction, males still exceeded females numerically.

With respect to the diagnostic system (variable 6 in Methods), a one-way ANOVA revealed a trend level significance with respect to diagnostic system used [$F(1, 217)=3.78$, $p=0.053$] with DSM diagnoses resulting in a higher proportion of males. This is qualified by the far larger

number of studies that used DSM (N=194) as compared to ICD (N=25). Diagnosis (variable 7 in Methods) did not affect the proportion male. In fact, though the differences were not significant, those studies including schizophrenia spectrum diagnoses yielded the most female participants. We then considered the effect of the remaining variables. The number of participants did not correlate to the proportion of males. As the mean age of the patient sample rose, the proportion of females increased significantly, although this was a modest effect [$R^2$=0.04, p<0.005].

## 4. Discussion

Our analysis revealed a widespread mismatch between the incidence of schizophrenia in females and their participation in research studies. While the expected epidemiological incidence rate was 58% males and 42% females, non-epidemiological study participant groups consistently included a higher proportion of males, averaging 66% of study samples. Additional points emerge from the examination of moderators of this effect. There was some variability in gender proportions as a function of study characteristics, but all variables contained numerically more males than females and, for most, these differences were significant compared with the epidemiological literature, and survived statistical corrections for multiple testing. Our estimate of the magnitude of this effect may be conservative. Had we used the prevalence values that appear in the literature (i.e., 52% male; McGrath et al., 2008) or had we included studies of VA populations, the differences would have been greater. Thus, the evidence against equal frequency of the disorder across gender is strong in the epidemiological research – however, this imbalance is significantly exaggerated in published non-epidemiological research studies.

It is instructive to consider our findings relative to those of previous reports (Wahl, 1977; Wahl & Hunter, 1992). In the 1970's, gender proportions in epidemiological and non-epidemiological seemed to be matched. However, the 1980's saw large jumps in the number of male research participants. Our findings are comparable to those of the 1980's. Thus, males appear to have remained overrepresented in the literature for over two decades.

We found a significant correlation of age and the gender of participants, with fewer males the older the mean age of the sample. Age-of-onset literature predicts such an effect (Häfner et al., 1989). However, the correlation accounted for less than 5% of the variance in our sample. When comparing the number of participants to their gender, it made sense that larger samples might liken the gender proportions of non-epidemiological research to epidemiological findings. Yet, the regression was not significant, suggesting that the nature of the research, more than the size of the sample, influenced the number of females.

Since the proportion of males was significantly greater than the epidemiological baseline for nearly all the categorical variables, the elevated number of males in non-epidemiological research does not appear to be an isolated occurrence. The few studies that resemble the incidence rate- namely genetic, Molecular Psychiatry, prodromal, Asian, Australian, and transcontinental studies- offer insight into characteristics that increase the number of females in patient samples. Because schizophrenia is a heterogeneous group of disorders and variation has been shown between sites {Hambrecht, 1992 #59}, it is likely that schizophrenia – and, consequently, gender ratios- will differ with the setting. There was a trend level effect for diagnostic system but, given the unbalanced subgroups, this possible effect requires further investigation. Unexpectedly, the type of diagnosis (e.g. schizophrenia only, schizophrenia spectrum) did not show a significant gender effect. Contrary to literature which suggests women are more likely to be diagnosed with schizoaffective disorder (Tsuang, Dempsey, & Rauscher, 1976), there were more women in the studies limited to schizophrenia diagnoses

only, compared to those including schizophrenia along with schizoaffective and/or schizophrenia spectrum diagnoses.

Explaining the considerable difference between the incidence literature and the non-epidemiological literature is difficult. First, it is possible that the incidence of schizophrenia in women is overestimated. The diagnosis of schizophrenia is difficult and may be affected by psychiatrists' biases. A study that examined diagnostic changes in patient charts over seven years found that significantly more females than males initially are diagnosed with bipolar disorder or a psychotic disorder outside of the schizophrenia spectrum, while more males than females receive an initial schizophrenia diagnosis that is later a diagnosis outside of the schizophrenia spectrum (Chen, Swann, & Burt, 1996). Other studies find that gender does not predict diagnosis accuracy (Moilanen et al., 2003). However, the fact that the prodromal conversion literature approximates the epidemiological literature weakens this explanation. Another explanation is that a greater proportion of female patients in hospitals and clinics choose not to participate in research. The literature on this point is inconsistent (Covell, Frisman, & Essock, 2003; Moilanen et al., 2003). However, there was not sufficient information in our data to address this point. Future studies would benefit from considering the relationship of gender and variables during recruitment, such as refusal rates.

It may be that many women with schizophrenia never enter the treatment settings where most clinical research is conducted. Our findings are most consistent with this hypothesis. The prodromal patients matched the epidemiological ratio, while chronic and first episode populations contained a far higher number of men. Of note, the recruitment of prodromal patients typically involves very aggressive case finding methods, often outside of mental health treatment settings, methods more akin to those typically employed in epidemiological and genetic research. Indeed, the difference between the proportion of males in first episode studies and that of prodromal studies is dramatic: the first episode samples have nearly 150% more males than prodromal samples. This suggests that only some portion of cases present for care in the settings captured in the first episode literature, and that portion is comprised very largely of male patients. It is possible that there are substantial numbers of female patients with schizophrenia who remain fully outside of the clinical settings where schizophrenia research is conducted. There is evidence that women display different symptomatology than men (Angermeyer, Kuhn, & Goldstein, 1990; Grossman, Harrow, Rosen, & Faull, 2006; Hambrecht, Maurer, & Hafner, 1992; Wahl, 1977; Wahl & Hunter, 1992) and that these differences are not always apparent at onset, but rather develop as the illness progresses (Willhite et al., 2008). Thus gender differences may not be pronounced affecting study participation and trajectory of illness until after the prodromal stage.

Careful screening during the recruitment process, as in genetic studies that employ large, representative populations, seems to result in gender ratios similar to epidemiological predictions. This may explain why gender proportions in studies reported in Molecular Psychiatry, publishing with a heavy emphasis on genetic research, more nearly approximates actual incidence proportions. Since the degree of unevenness varied according to continent of study, we must also consider the impact of culture, economy, and human participant protection practices. Stringent ethical guidelines can make it more difficult to recruit female participants, particularly those of childbearing years.

## 5. Conclusions

In an effort to understand the gender discrepancy, we separately examined a number of broad and obvious differences across studies that might moderate the effect, including journal of publication, study type, sample, institution, continent, and diagnostic criteria. With limited exceptions, a difference in the number of male and female participants appears across the

variables. These dimensions are overlapping and are not offered as independent tests of the discrepancy. Rather, as a whole, they offer converging evidence of the discrepancy, and the few exceptions help to 'prove the rule' by suggesting particular instances in which the degree of difference is ameliorated (i.e., in genetics studies that may have more rigorous ascertainment procedures). A large implication of this discrepancy may be that many of the findings that we typically consider to be "true" about schizophrenia may be truer for men than for women. Indeed, research on animal models of schizophrenia routinely finds sex-based differences (Häfner, 1991; Walker, 1999), suggesting biological differences. Most of our research-based evidence regarding the characteristics, correlations, etiology, and treatments of schizophrenia has come primarily from the study of male patients and may not fully apply to females with schizophrenia. The degree to which this knowledge generalizes to women is a question that has received some, but not adequate attention in the literature. Therefore, stratification by gender, and similar strategies, may need to be used in future schizophrenia studies in order to increase the relevance of findings to women.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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## Table 1

The percentage of male patients among all study participants with schizophrenia.

| | Arch Gen Psychiatry | All Journals Considered[†] |
|---|---|---|
| **1974-1976** (Wahl, 1977) | 51% (N=52)[‡] | 59% (N=87) |
| **1985-1989** (Wahl and Hunter, 1992) | 71% (N=63) | 69% (N=198) |
| **2006** (present study) | 68% (N=16) | 66% (N=220) |

[†] 3 journals in 1977; 5 journals in 1992; 7 journals in 2006. See text for specific journals.

[‡] Number of journal articles included in the comparison, sometimes notated as k.

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**Table 2**

Non-epidemiological research findings by individual variable levels. The values from the 'Proportion Male' column (where proportion male plus proportion female equals 1) were contrasted, by t-tests for single means, with the epidemiological baseline of 58% (or 0.58) of schizophrenia patients being men, as set by a recent meta-analysis(McGrath et al., 2008). Because of the large number of t-tests, we adopted Holm p-value cutoffs in place of the more standard $p<0.05$ cutoff. If a p-value is less than the value in the 'Holm p' column, it is significant. This is also indicated by the asterisks and bold font.

| Variable | Variable Level | N[1] | Mean % Male[2] | (SD) | t-value | p-value | (df) | Holm p[3] |
|---|---|---|---|---|---|---|---|---|
| Journal | Mol Psychiatry | 5 | 0.56 | (0.10) | -0.47 | 0.6621 | (4) | 0.05 |
| | **Br J Psychiatry** | **14** | **0.66** | **(0.11)** | **2.77** | **0.0159** * | **(13)** | **0.025** |
| | **Schizophr Bull** | **19** | **0.68** | **(0.14)** | **3.02** | **0.0074** * | **(18)** | **0.0167** |
| | **Biol Psychiatry** | **24** | **0.67** | **(0.12)** | **3.49** | **0.0020** * | **(23)** | **0.0125** |
| | **Arch Gen Psychiatry** | **16** | **0.68** | **(0.10)** | **4.11** | **0.0009** * | **(15)** | **0.01** |
| | **Am J Psychiatry** | **24** | **0.68** | **(0.11)** | **4.63** | **0.0001** * | **(23)** | **0.0083** |
| | **Schizophr Res** | **118** | **0.65** | **(0.13)** | **6.13** | **0.0000** * | **(117)** | **0.0071** |
| Study Type | genetic | 38 | 0.59 | (0.09) | 0.56 | 0.5772 | (37) | 0.05 |
| | **pharmacological** | **32** | **0.65** | **(0.11)** | **3.32** | **0.0023** * | **(31)** | **0.025** |
| | **psychosocial** | **40** | **0.64** | **(0.12)** | **3.31** | **0.0020** * | **(39)** | **0.0167** |
| | **anatomical** | **23** | **0.71** | **(0.14)** | **4.29** | **0.0003** * | **(22)** | **0.0125** |
| | **functional imaging** | **13** | **0.74** | **(0.11)** | **5.56** | **0.0001** * | **(12)** | **0.01** |
| | **cognitive** | **74** | **0.68** | **(0.12)** | **7.29** | **0.0000** * | **(73)** | **0.0083** |
| Illness Stage | prodromal | 3 | 0.56 | (0.21) | -0.17 | 0.8796 | (2) | 0.05 |
| | **mixed sample** | **8** | **0.68** | **(0.09)** | **3.35** | **0.0122** * | **(7)** | **0.025** |
| | **first episode** | **30** | **0.71** | **(0.11)** | **6.07** | **0.0000** * | **(29)** | **0.0167** |
| | **chronic** | **143** | **0.64** | **(0.12)** | **6.56** | **0.0000*** | **(142)** | **0.0125** |
| Treatment Setting | **outpatient** | **46** | **0.64** | **(0.12)** | **3.56** | **0.0009** * | **(45)** | **0.05** |
| | **inpatient** | **39** | **0.68** | **(0.12)** | **5.27** | **0.0000** * | **(38)** | **0.025** |
| | **in and out patients** | **43** | **0.69** | **(0.11)** | **6.69** | **0.0000** * | **(42)** | **0.0167** |
| Continent | Asia | 29 | 0.59 | (0.12) | 0.49 | 0.6267 | (28) | 0.05 |

| Variable | Variable Level | N[1] | Mean % Male[2] | (SD) | t-value | p-value | (df) | Holm p[3] |
|---|---|---|---|---|---|---|---|---|
| | Australia | 4 | 0.72 | (0.10) | 2.73 | 0.0721 | (3) | 0.025 |
| | cross-continental sites | 25 | 0.62 | (0.08) | 2.1 | 0.0466 | (24) | 0.0167 |
| | **Europe** | **47** | **0.67** | **(0.11)** | **5.48** | **0.0000** * | **(46)** | **0.0125** |
| | **North America** | **114** | **0.68** | **(0.13)** | **8.51** | **0.0000** * | **(113)** | **0.01** |

[1] Signifies the number of articles included in the comparison, sometimes notated as k

[2] as compared to epidemiological baseline (0.58 proportion males)

[3] threshold for significance after correction for multiple comparisons(Holm, 1979).

* significant p-values as compared to Holm adjusted thresholds