Letters

RESEARCH LETTER

Sex Differences in Institutional Support for Junior Biomedical Researchers

Women are underrepresented in the biomedical research workforce. Only 30% of funded investigators are women.[1,2] Junior faculty women have fewer peer-reviewed publications than men[3,4] and are more often on clinician-educator (vs traditional) tracks.[5] One reason may be differences in early-career institutional support, which to our knowledge has not been previously examined.

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Methods | Application data from 2 New England biomedical research programs administered by the Medical Foundation Division of Health Resources in Action were analyzed. One program accepted applicants in a single field of study within 5 years of initial faculty appointment; the second program invited institutions to submit 2 basic science applicants within 2 years of initial appointment.

Data on start-up support provided by the employing institution (ie, salary and other support, including research technicians, equipment, and supplies) from all applications during 2012-2014 were extracted.

We compared support for men and women overall, and by scientific focus, terminal degree, and years since receiving terminal degree. Data concerning age and race were not collected. Publication data were incomplete and not included.

Two analyses were performed to examine associations with institutional characteristics. Applicants were employed by universities, hospitals, and other nonprofit research institutions. As a measure of overall institutional research resources, we stratified the sample by overall institutional funding from the National Institutes of Health (NIH) in fiscal year 2014. We also directly compared support for men and women who applied from the same institution.

Statistical analyses were performed using Epi Info version 7.1.4 (US Centers for Disease Control and Prevention). The *P* values were calculated using Kruskal-Wallis nonparametric tests (continuous variables), Spearman correlation analysis, or 2-tailed Fisher exact tests (categorical variables).

$P < .05$ (2-sided) was considered significant. The Hummingbird Institutional Review Board determined that this

Table 1. Characteristics of Men and Women Applying for Early-Career Awards[a]

	No. (%)[b]			
	Total	Men	Women	*P* Value[c]
No. of applicants	219 (100)	127 (58)	92 (42)	
Type of degree				
MD	29 (13)	16 (13)	13 (14)	.16
PhD	147 (67)	81 (64)	66 (72)	
MD, PhD	31 (14)	24 (18)	7 (8)	
Other[d]	12 (5)	6 (5)	6 (7)	
Type of research				
Basic[e]	170 (78)	108 (86)	62 (67)	<.001
Clinical[f]	49 (22)	19 (15)	30 (33)	
NIH funding to institution by quartile[g]				
1[h]	109 (50)	69 (54)	40 (43)	.02
2	57 (26)	31 (24)	26 (28)	
3	27 (12)	9 (7)	18 (20)	
4	26 (12)	18 (14)	8 (9)	
Institutions with >10 applicants	85 (39)	45 (35)	40 (43)	.26
Time since terminal degree, mean (SD), y	7.5 (3.2)	7.9 (3.4)	7.2 (3)	.08

Abbreviation: NIH, National Institutes of Health.

[a] Data were collected from applications submitted to 2 regional junior investigator programs from 2012-2014.

[b] Unless otherwise indicated.

[c] Calculated using the Kruskal-Wallis test.

[d] Included 1 or more persons with the following degrees: DPhil, DSc, DSc and MPH, DrPH, MBBS, MD and MPH, PhD and DVM, and ScD.

[e] Included animal studies as well as those involving cellular and molecular biology.

[f] Involved human participants or patient-derived data.

[g] Applicant institutions were ranked by fiscal year 2014 total institutional funding.

[h] Included the 25% of all 55 institutions with fiscal year 2014 NIH research funding of more than $122 million.

Table 2. Institutional Start-up Support for Men and Women Applying for Early-Career Awards[a]

	Start-up Support, Median (IQR), in 1000s of US$			
	Total	Men	Women	*P* Value[b]
Overall	678 (216-1100)	889 (283-1250)	350 (180-775)	<.001
Type of degree				
MD	528 (150-900)	596 (50-1123)	474 (200-800)	.95
PhD	717 (240-1100)	936 (475-1250)	348 (180-750)	<.001
MD, PhD	800 (267-1393)	961 (271-1447)	500 (0-850)	.23
Type of research				
Basic[c]	811 (350-1200)	980 (504-1290)	585 (225-882)	<.001
Clinical[d]	210 (89-350)	162 (0-435)	213 (101-350)	.25
NIH funding to institution by quartile[e]				
1[f]	830 (263-1300)	1040 (409-1500)	368 (169-800)	<.001
2	600 (223-950)	725 (275-970)	388 (186-922)	.16
3	583 (210-750)	660 (331-1100)	541 (204-750)	.37
4	376 (150-1050)	537 (169-1160)	184 (117-600)	.16
Institutions with >10 applicants	575 (210-1080)	850 (258-1300)	483 (203-750)	.03

Abbreviations: NIH, National Institutes of Health; IQR, interquartile range.

[a] Data were collected from applications submitted to 2 regional junior investigator programs from 2012-2014.

[b] Calculated using the Kruskal-Wallis test.

[c] Included animal studies as well as those involving cellular and molecular biology.

[d] Involved human participants or patient-derived data.

[e] Applicant institutions were ranked by fiscal year 2014 total institutional funding.

[f] Included the 25% of all 55 institutions with fiscal year 2014 NIH research funding of more than $122 million.

study used coded administrative data and was therefore exempt from human studies review.

Results | The characteristics of the 219 applicants (127 men and 92 women) with complete data are reported in **Table 1.** An additional 8 men (6%) and 5 women (5%, $P > .99$) had incomplete data and were excluded.

Most applicants (67%) held PhD degrees. Women were in clinical research more frequently than men. There were no differences between men and women in terminal degree or years since receiving terminal degree.

Data on institutional start-up support are reported in **Table 2.** Overall, men reported significantly higher start-up support (median, $889 000 [interquartile range, $283 000-$1 250 000]) than women (median, $350 000 [interquartile range, $180 000-$775 000]; $P < .001$); 51 men (40%) and 11 women (12%) reported support of more than $1 million ($P < .001$).

Men had higher support regardless of degree, but the difference was statistically significant only for persons with PhD degrees. In basic sciences, men reported significantly more start-up support than women. Start-up support for clinical scientists was not significantly different for men and women.

Applicants were employed by 55 institutions. The top 25% (quartile 1) of the 55 institutions each received more than $122 million in NIH funding (quartile 2: >$46 million; quartile 3: >$19 million; quartile 4: ≤$19 million). Half of the applicants were from institutions in the top funding quartile (Table 1).

When stratified by NIH funding, men had larger start-up packages within each stratum (Table 2). Five institutions had more than 10 applicants. Support for men was higher in 4 of these institutions and overall (Table 2). In this group of 5 institutions, 4 women (10%) and 19 men (42%) received more than $1 million ($P = .001$).

Experience (years since receiving terminal degree) did not correlate with start-up package size for men ($r^2 = 0.01$; $P = .57$), women ($r^2 = 0$; $P = .60$), or overall ($r^2 = 0$; $P = .80$).

Discussion | In this preliminary study of early-career grant applicants administered by 1 organization, junior faculty women received significantly less start-up support from their institutions than men. This discrepancy was significant only among basic scientists and was not explained by degree, years of experience, or institutional characteristics.

The limitations include reliance on limited self-reported and administrative data. The representativeness and generalizability of these results to applicants to other foundations or institutions, or to other biomedical investigators, are unknown.

This first look suggests the need for systematic study of sex differences in institutional support and the relationship to career trajectories.

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Study concept and design: Sege.
Acquisition, analysis, or interpretation of data: All authors.
Drafting of the manuscript: Sege, Selk.
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Correction: This article was corrected on October 1, 2015, to fix 1 error in Table 1 and 1 error in Table 2.

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COMMENT & RESPONSE

Amyloid Pathology, Cognitive Impairment, and Alzheimer Disease Risk

To the Editor In a recent meta-analysis, Ms Jansen and colleagues[1] stated that "the presence of SCI [subjective cognitive impairment] in a memory clinic population might not be associated with an increased risk for AD [Alzheimer disease]."

This conclusion cannot be drawn from the cross-sectional design of the included studies. It also contradicts a recent meta-analysis of longitudinal studies,[2] which identified an increased dementia risk among patients with SCI.

The authors said that they did not find an increased frequency of amyloid positivity in SCI. However, this conclusion appears doubtful for a number of reasons.

First, the main analyses included diagnostic group, age, sex, education, and *APOE-ε4* genotype as independent variables. The authors did not consider the increase in *APOE-ε4* prevalence among patients with SCI (39.6% vs 29.5% in cognitively normal, $P < .001$). Because *APOE-ε4* is strongly associated with amyloid positivity, modeling *APOE* genotype as an independent variable may mask an increased prevalence of amyloid positivity in the SCI group.

Second, the prevalence estimates in Figure 2 in the article were derived from another model, which included neither *APOE* genotype nor education. However, comparing highly educated volunteer controls (64.3% with high education) with less well-educated patients with SCI (39.6% with high education, $P < .001$) requires an adjustment for education because higher education (possibly by conferring cognitive reserve) is associated with higher prevalence of amyloid positivity (as depicted in the Supplement for the article).

Third, little information was provided on the differentiation of patients with SCI and controls within the contributing samples with the exception that patients with SCI consulted a memory service. Because the clinical definition of SCI in this meta-analysis was limited, the composition of the SCI and control groups may have varied between the contributing samples, and some control groups may have included patients with SCI (who had not attended a memory service).

There are strong international efforts to identify the earliest signs of AD for future dementia prevention. In this regard, SCI is a clinical condition of great interest and potential.[3] Research on the association of SCI with AD biomarkers is just beginning and requires in-depth analysis of existing data and new studies that focus on SCI.

The conclusion of Jansen et al[1] on the lack of an association of SCI with amyloid positivity is premature because it was based on a crude look at heterogeneous data and did not consider the refined clinical differentiation of SCI, which is required to use it as the first clinical marker of AD.

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Conflict of Interest Disclosures: The authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest and none were reported.

1. Jansen WJ, Ossenkoppele R, Knol DL, et al; Amyloid Biomarker Study Group. Prevalence of cerebral amyloid pathology in persons without dementia: a meta-analysis. *JAMA*. 2015;313(19):1924-1938.

2. Mitchell AJ, Beaumont H, Ferguson D, Yadegarfar M, Stubbs B. Risk of dementia and mild cognitive impairment in older people with subjective memory complaints: meta-analysis. *Acta Psychiatr Scand*. 2014;130(6):439-451.

3. Jessen F, Amariglio RE, van Boxtel M, et al; Subjective Cognitive Decline Initiative (SCD-I) Working Group. A conceptual framework for research on subjective cognitive decline in preclinical Alzheimer's disease. *Alzheimers Dement*. 2014;10(6):844-852.

In Reply Drs Wagner and Jessen disagree with our conclusion that participants with SCI do not have an increased risk for AD compared with cognitively normal participants. We think that this conclusion is justified because participants with SCI had a similar prevalence of amyloid positivity, the pathological hallmark of AD, as cognitively normal participants.

Wagner and Jessen argue that a meta-analysis[1] found that SCI was associated with an increased risk for dementia. However, the outcome measure used in that study was any type of dementia, and therefore it is unclear whether this finding also applies to AD-type dementia.

In addition, Wagner and Jessen outline several possible methodological issues that might explain why amyloid positivity in SCI was not increased relative to cognitively normal participants in our study.

First, they suggest that correction for the *APOE* genotype could have masked an increased prevalence of amyloid positivity in patients with SCI. However, the analysis presented in Figure 2 in the article was performed without correction for *APOE-ε4* carrier status and showed no difference in amyloid