Science & Society

Identifying Barriers to Career Progression for Women in Science: Is COVID-19 Creating New Challenges?
Diana S. Hansen1,2,*

This article summarizes discussions at a Gender Equity Workshop run during the Molecular Approaches to Malaria Conference in February 2020. Barriers to career progression in science for women and minority groups, along with suggestions to overcome ongoing roadblocks, are discussed. The emerging challenges that coronavirus disease 2019 (COVID-19) is bringing to this sector are also considered.

On 25 February 2020, I had the privilege to chair a workshop at the Molecular Approaches to Malaria (MAM) Conference held in Lorne, Australia. The title of the workshop was ‘Identifying Barriers of Career Progression in Science for Women and Minority Groups’. Joined by a diverse panel of invited speakers, including Katherine Andrews (Griffith Institute for Drug Discovery, Australia), Aung Pyae Phyo (Myanmar Oxford Clinical Research Unit), and Arlene Dent (Case Western Research University, USA), we discussed: the benefits of diverse research teams, the main roadblocks that women and minority groups (including ethnic and racial minorities) still encounter that hinder their careers, and a useful toolkit of ideas, suggestions, and good advice [1] proven to result in positive outcomes towards creating equal-opportunity working environments in the life sciences (Figure 1).

The Benefits of a Diverse Workforce in Science

Although social justice and, to some extent, legal compliance might have been the initial drivers towards the implementation of policies to promote inclusive working environments, it is becoming increasingly accepted that diversity in the workforce provides a competitive edge [2]. Research on financial performance from a group of 366 companies across the UK, Canada, the USA and Latin America revealed that gender-diverse companies are 15% more likely to financially outperform their counterparts [3]. Furthermore, ethnically diverse companies were found to be 35% more likely to have financial returns above their national industry median [3]. Diversity has also been shown to provide a competitive advantage in the academic sector. A recent study found that research publications authored by ethnically diverse and gender-diverse groups of researchers receive significantly higher numbers of citations than papers written by researchers of a single ethnic group or same gender [4].

Despite this increasing body of evidence, women in science, technology, engineering, or math (STEM) are under-represented in education and careers, and women from minority groups, such as African American, Hispanic, or indigenous women, are particularly under-represented [5]. This trend becomes evident after graduation from high school, where women are less likely than men to pursue a major STEM field. Women’s representation in science declines further at the graduate level and, yet again, in the transition to the workplace. Furthermore, representation of women at senior and professorial levels is extremely poor (i.e., 14.5% in the Australian academic sector) [6]. This demographic of workforce distribution, causing high levels of midcareer exhaustion, also results in a gender pay gap for STEM (ranging from 11% in engineering to 12.4% in science positions in Australia) [6], and a society that undervalues the opportunities and contributions a career in STEM can provide for girls and women.

Classical Barriers to Career Progression for Women in STEM

During the workshop at the MAM Conference, several different barriers were identified as key contributors hindering career progression for women in parasitology and public health. Cultural issues such as bias and stereotypes [7–9] (Box 1) along with often male-dominated working environments that are characterised by strong hierarchical natures, appeared to be the first hurdle encountered by young female graduates commencing a career in STEM.

In addition to these culturally posed challenges, women are more likely to be the primary carer not only for young children but also for ill or aging members of their families. Balancing work and carer responsibilities could be even more challenging for scientists in single-parent scenarios. Remarkably, the years of intensive research and long hours in the laboratory, usually required to establish an independent research career path, often coincide with the period in which people tend to start families. The unavoidable drop in productivity that women experience during these years, usually measured as the number of papers published and their impact, has an inexorable detrimental impact on grant success rates, which largely depend on a strong publication record in un forgiving funding environments. These are important obstacles for women choosing to take career breaks or part-time appointments when raising children. Furthermore, working conditions and job insecurity were also identified as having a strong negative impact on women maintaining careers in science. The vast majority of appointments are grant-dependent short-term contracts, with unclear pathways to promotion. Whilst this also impacts male researchers, career disruptions due to carer responsibilities and the impact on
their track records make these uncertain scenarios more challenging for women, encouraging them to seek alternative positions outside the academic sector.

Whereas the aforementioned barriers are somehow potentially possible to measure, other elements, including the lack of role models and mentors, are more difficult to define with quantitative data but were also identified as important detrimental factors in career progression for women in STEM. Perhaps even more important than mentors, a crucial and difficult-to-measure hurdle for women, particularly for those trying to break through the ‘glass ceiling’ and consolidate their position in senior management, is the lack of sponsors\(^*\) willing to advocate on their behalf to help them promote and advance their careers to professorial positions. This lack of support networks contributes to women usually feeling isolated and out of place in STEM fields. These growing feelings of misfit or ‘imposter syndrome’ are highly demoralising and result in women questioning their abilities and on occasion, lead to thoughts of leaving the field.

Most of the barriers, discussed here, experienced by women pursuing a career in STEM were shared by women and males from minority groups. Scientists from minority groups also face additional challenges [10], including reduced access to resources and technology as well as negative perceptions of their own career success, in part due to reduced networking opportunities and role models.

The participants at the Gender Equity Workshop at MAM 2020 discussed various strategies and resources that helped them overcome obstacles during their careers in parasitology (Box 2). Among them, identifying early in their career a diversity-prone working environment, appropriate mentors, and a good group of collaborators seem to be important for women to avoid isolation, stay connected and further their networking skills to boost their careers in science. It was clear from discussions at the workshop that this is not an easy path. Gender equity, diversity, and inclusion in STEM careers are work in progress. Being patient, persistent, and remaining aligned with the core principles and values that inspire women to start a career in parasitology, are all desirable qualities to successfully navigate this path.

As for employers, it is clear that there is an urgent need for further resources in order to improve recruitment, development, and retention of women in science careers (Box 2). A commitment to training towards

---

Box 1. Stereotypes and Common Biases Encountered by Women in STEM

- Men are better than women for STEM fields
- Women are not interested in careers in science
- Successful women behave in masculine ways
- Gender bias in peer review
- Gender bias in job applications
- Gender bias in promotions

---

Figure 1. The Panellists and Audience at the Breakfast Workshop Entitled ‘Identifying Barriers of Career Progression in Science for Women and Minority Groups’. Held during the Molecular Approaches to Malaria 2020 Conference, in Lorne, Australia, from 23–27 February 2020, the session chaired by Dr Diana Hansen from the Walter and Eliza Hall Institute, Australia (middle right panel), featured (from bottom left to right) Dr Arlene Dent (Case Western Research University, USA), Dr Aung Pyae Phyo (Myanmar Oxford Clinical Research Unit), and Professor Katherine Andrews (Griffith Institute for Drug Discovery, Australia). The top left shows the audience, with Dr Julia Cutts (Burnet Institute, Australia) asking a question of the panel.
recognising stereotypes and unconscious bias along with transparent recruitment and promotion processes will be required to close the gap [6]. Clear strategic plans to promote cultural shifts in organisations are needed to ensure that women and minority groups have access to developmental opportunities and adequate paths to advance their careers in science.

A New Element in the Equation: COVID-19

When I was invited to contribute this article in February it would have been hard to predict that a global pandemic would spread around the world with unprecedented consequences for health systems and global economies. Emerging evidence is starting to reveal alarming figures on the gendered impact of COVID-19, and the STEM workforce is no exception.

A recently released report commissioned by the Australian government and produced by the Rapid Research Information Forum (RRIF) estimated that the pandemic will result in greater disadvantages for women than men in the STEM sector [11]. The report also warns that women from culturally diverse backgrounds, Aboriginal women and women who identify as lesbian, gay, bisexual, trans/transgender, intersex, queer/questioning, and asexual (LGBTIQA+) are potentially facing additional barriers during the pandemic. Evidence shows that, while work-from-home policies apply equally to women and men, women in heterosexual relationships seemed to bear the major burden of home-schooling of their children, meal preparation and general housework compared with men, while trying to manage their paid workload [11]. There is also evidence that the pandemic has resulted in women being less likely than men to attend STEM workplaces, with women accounting for 16% of staff visiting campuses in Australia, compared with 28% males between March and April 2020 [11].

Academic output, including not only submission of manuscripts but also grant applications, appeared to be compromised for women in STEM in New Zealand during the pandemic. A recent analysis revealed that female academics are submitting significantly fewer papers than men and starting fewer new projects [12]. This reduction in the quality and quantity of women’s research publications is anticipated to harm job and funding prospects now and for several years to come. Furthermore, with income losses in universities and the research sector facing budget cuts, STEM jobs are at ongoing risk, and short-term contracts that usually mainly employ women are likely to be the ones to take the hardest hit. Indeed, whereas Australia’s professional, scientific and technical service industry recorded job losses of 4.8% for men from mid-March to mid-April 2020, jobs in the sector were down 6.3% for women [13].

Acknowledgements

The author is supported by the Australian Government National Health and Medical Research Council Independent Research Institutes Infrastructure Support Scheme and the Australian Academy of Science Regional Collaboration Grant.

Resources

www.mam2020conference.com.au/
https://libre.ify.com/2016/09/how-sponsoring-could-help-to-bridge-the-gender-gap-in-the-u-s-stem-sector-danika-kimball/
www.mbie.govt.nz/science-and-technology/science-and-innovation/agencies-policies-and-budget-initiatives/diversity-in-science/

© 2020 Elsevier Ltd. All rights reserved.

We should remain hopeful that anticipated COVID-19-related funding cuts in the sector do not compromise ongoing equity programs as this could jeopardise all progress made in STEM workforce diversity. The future of women in STEM could be at risk if the improvements achieved in recent years are lost. As a community, it is critical to start the conversation now, on how to mitigate the impact that COVID-19 has on job security and career progression for women in STEM.

References

1. Australian Commission of Human Rights (2013) Women in Male-dominated Industries, Department of Families, H., Community Services and Indigenous Affairs, Commonwealth of Australia
2. Powell, K., ed (2018) The power of diversity. Nature 558, 19-22
3. Hunt, V. et al. (2018) Delivering through diversity. McKinsey & Company, M. (Ed).
4. Alshehi, B.K. et al. (2018) The preeminence of ethnic diversity in scientific collaboration. Nat. Commun. 9, 5163
5. Hill, C. et al. (2013) Why so few?. American Association of University Women
6. Commonwealth of Australia (2019) Advancing Women in STEM, Department of Industry Innovation and Science
7. Carli, L.L. et al. (2016) Stereotypes about gender in science. Women & Scientists. Psychol. Women Quart. 40, 244–260
8. Kaatz, A. et al. (2014) Threats to objectivity in peer review: the case of gender. Trends Pharmacol. Sci. 35, 371–373
Malaria, caused by protozoan parasites of the genus *Plasmodium*, remains a major public health issue in the tropics and a potentially fatal disease, although major progress has been achieved in the last two decades with the scaling up of priority interventions (prevention and management of malaria cases). Severe malaria, due mainly to *Plasmodium falciparum*, is responsible for nearly half a million deaths annually, affecting mostly children under 5 years of age and pregnant women living in sub-Saharan Africa, as well as non-immune travelers [1]. Although *P. falciparum* is the most prevalent and dangerous species, other *Plasmodium* species, including *Plasmodium vivax*, *Plasmodium malariae*, *Plasmodium ovale*, and *Plasmodium knowlesi*, account for a nonnegligible impact on human health. Malaria symptoms, ranging from cyclic febrile episodes to life-threatening complications (severe anemia, respiratory distress, and coma), are linked mainly to the parasite’s development in human red blood cells (RBCs) [2].

Studying the complex host-pathogen interactions is of major importance for deciphering the cellular and molecular mechanisms involved in malaria infections and for developing more effective therapies and a vaccine [3,4].

In this respect, spectacular progress has been made in recent years in our understanding of the function of RIFINS, which are used by *P. falciparum* to escape the host’s immune system. More precisely, some RIFIN variants were shown to be able to bind to either leucocyte immunoglobulin-like receptor B1 (LILRB1) or leucocyte-associated immunoglobulin-like receptor 1 (LAIR1) [7–9] and inhibit the activation of LILRB1–expressing immune cells. However, one of the missing pieces of the puzzle was a comprehensive definition of the inhibitory function of RIFINS. This has been achieved recently in a striking study published by Harrison and colleagues. They elucidated the structural basis on which RIFINs activate LILRB1 by mimicking the binding mode of the natural ligand of LILRB1 for suppression of immune cell function [10].

**Spotlight**

**RIFINing Plasmodium–NK Cell Interaction**

Didier Ménard,1,* Sandrine Houzé,2,* and Nicolas Papon3,4

Among many *Plasmodium* proteins involved in the erythrocytic cycle, some exposed on the erythrocyte surface drive antigenic variability. Recently, Harrison et al. elucidated the structural basis by which RIFINS activate LILRB1 and suppress immune cell function. This breakthrough points to an additional strategy for survival in the human host.

Malaria symptoms, ranging from cyclic febrile episodes to life-threatening complications (severe anemia, respiratory distress, and coma), are linked mainly to the parasite’s development in human red blood cells (RBCs). One of the main challenges for malaria parasites is to achieve effective proliferation and differentiation in RBCs — requires escape from the human immune system. The selection of highly polymorphic sequences at the end of some chromosomes (i.e., subtelomeres) encoding variant polypeptides of multicopy gene families seems to explain how malaria parasites have adapted to this barrier [5]. Some of these variant polypeptides have been shown to be involved in host cell adhesion, notably in promoting the parasite’s invasion of RBCs. However, the major role of these variant polypeptides, exposed on the surface of infected RBCs, is to ensure antigenic variation and therefore to circumvent the host’s immunity response. Among hundreds of adhesive proteins, three main families are well documented [6]. These include (i) the var family, encoding the variant antigen *P. falciparum* erythrocyte membrane protein 1 (PfEMP1); (ii) the subtelomeric variant open reading frame (ORF) family (stever), encoding STEVOR proteins; and (iii) variant polypeptides from the repetitive interspersed family (rif) encoding RIFINS [6]. PfEMP1, STEVOR, and RIFINS are responsible for characteristic protruberances on the surface of the RBCs in association with a submembranous network of knob proteins (Figure 1A).

This unique RBC membrane shape is known to promote the binding of infected erythrocytes to the endothelium (cytoadherence), a major mechanism to circumvent removal of infected RBCs by the spleen and the main pathophysiological phenomenon responsible for the severe forms of malaria [6]. Besides this function, variant surface antigens also play a crucial role in regulating interactions of infected RBCs with immune cells for dampening the host response.

To date, many parasite proteins have been described as essential for *Plasmodium* parasites to achieve their complex life cycle in the human host during various steps. One of the main challenges for malaria parasites is to achieve effective proliferation and differentiation in RBCs — requires escape from the human immune system. The selection of highly polymorphic sequences at the end of some chromosomes (i.e., subtelomeres) encoding variant polypeptides of multicopy gene families seems to explain how malaria parasites have adapted to this barrier [5]. Some of these variant polypeptides have been shown to be involved in host cell adhesion, notably in promoting the parasite’s invasion of RBCs. However, the major role of these variant polypeptides, exposed on the surface of infected RBCs, is to ensure antigenic variation and therefore to circumvent the host’s immunity response. Among hundreds of adhesive proteins, three main families are well documented [6]. These include (i) the var family, encoding the variant antigen *P. falciparum* erythrocyte membrane protein 1 (PfEMP1); (ii) the subtelomeric variant open reading frame (ORF) family (stever), encoding STEVOR proteins; and (iii) variant polypeptides from the repetitive interspersed family (rif) encoding RIFINS [6]. PfEMP1, STEVOR, and RIFINS are responsible for characteristic protruberances on the surface of the RBCs in association with a submembranous network of knob proteins (Figure 1A).

This unique RBC membrane shape is known to promote the binding of infected erythrocytes to the endothelium (cytoadherence), a major mechanism to circumvent removal of infected RBCs by the spleen and the main pathophysiological phenomenon responsible for the severe forms of malaria [6]. Besides this function, variant surface antigens also play a crucial role in regulating interactions of infected RBCs with immune cells for dampening the host response.

In this respect, spectacular progress has been made in recent years in our understanding of the function of RIFINS, which are used by *P. falciparum* to escape the host’s immune system. More precisely, some RIFIN variants were shown to be able to bind to either leucocyte immunoglobulin-like receptor B1 (LILRB1) or leucocyte-associated immunoglobulin-like receptor 1 (LAIR1) [7–9] and inhibit the activation of LILRB1–expressing immune cells. However, one of the missing pieces of the puzzle was a comprehensive definition of the inhibitory function of RIFINS. This has been achieved recently in a striking study published by Harrison and colleagues. They elucidated the structural basis on which RIFINs activate LILRB1 by mimicking the binding mode of the natural ligand of LILRB1 for suppression of immune cell function [10].

The *Plasmodium* RIFIN family is composed of approximately 150–200 members that are structurally categorized into two subtypes (A and B). Most of the RIFINS belong to subtype A. The canonical domain architecture of *P. falciparum* type A RIFINS consists of a C-terminal signal peptide (SP), a first short variable