Instructions for publication updates in the fortnightly SBMS Update

If you have recently had a new publication accepted or published, please let us know.

We are looking mainly for papers that are predominantly driven through SBMS (but of course big deal papers with you as minor author are also of great interest).

Send the information to Marrianne in this format.

- In the Subject line write '**SBMS new publication'**
- Title
- Full author list (unless this is enormous). Bold any SBMS or FoM authors
- Journal name
- 2-3 sentences about why you did this, what you found, what you will do next, why it matters, etc. You could include anything of particular interest for an internal audience (e.g. where the PhD student who did the work has now started their postdoc, a cool technique, facility or resource you used, etc).

E.g.

Abnormal behavior and cortical connectivity deficits in mice lacking Usp9x
Kasherman MA, Currey L, Kurniawan ND, **Zalucki O**, Vega MS, Jolly LA, Burne THJ, Wood SA, **Piper M.**
Cerebral Cortex, 2021
We deleted, specifically in the forebrain, the gene *Usp9x* (which encodes a deubiquitylating enzyme that regulates the stability of many autism spectrum disorder related proteins) and demonstrated that USP9X is a key regulator of brain formation and function. Because USP9X has the potential to regulate stability and/or activity of many different substrates, potentially concurrently, we think it might occupy a pivotal position in the autism spectrum disorder (ASD) genetic network.

Identification of regulatory elements required for Stra8 expression, in fetal ovarian germ cells of the mouse
Allen Feng, Guillaume Burnet, Cassy Spiller, Fiona Cheung, Kallayanee Chawengsaksophak, Peter Koopman and **Josephine Bowles**
Development, 2021
There is ongoing controversy as to whether retinoic acid (RA) is actually required to trigger meiosis *in vivo*. To address this, we made subtle mutations in two retinoic acid response elements (RAREs) in the promoter of the *Stra8* gene and showed that this critical pre-meiotic marker is a direct RA target *in vivo* (we used CRISPR/Cas9 to make these mutations in the mouse model).