

## Let's keep this brief

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### Editorial

The regular articles and technical advances published in this issue are an average of 9,050 words, with 8.4 display items (figures and tables). Do we always need so many words to convey a message? We think not. With this editorial, we issue a call for brief reports.

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The *JCI* has never specified a minimum length requirement, and indeed, many of the manuscripts we've seen cross our collective desk over the years could have been suitable for the category of brief reports. But as we haven't had a dedicated section, it is likely that authors shied away from a concise manuscript in the belief that more was better. Going forward, we want to change that notion.

Our articles, whether they are of the regular, technical, or brief variety, are diverse in focus and aim and may differ in structure. However, one goal of all the articles is the same: to communicate to a biomedical audience; and this can be done in multiple ways. Brevity cannot replace clear thinking and strategic writing; authors will still need to organize ideas carefully and express them coherently. The level of insight into mechanism in brief reports will not be held to a lower standard — we simply seek to publish more discrete, highly significant findings that can be appropriately conveyed in a more concise format.

In addition to welcoming regular-length research articles and technical advances, we are now formally entertaining submissions of shorter communications, designated

Brief Reports, with a maximum word limit of 3,500 words, 3 succinct figures, and 1 table. The results and discussion section should be merged, and key methods should be included in their own section. A concise supplemental data section will be allowed, to contain only materials that are not integral to the main manuscript. The flat fee for such articles will be \$2,500, and they will be made freely available from the date of publication, as are all the rest of our research articles.

We offer a few examples of articles published elsewhere that have captured the essence of what we are looking for. In their article in the *Journal of Immunology* (1), Tang and colleagues resolved the important paradox of why blockade of CD28 costimulatory signals in nonobese diabetic mice resulted in disease exacerbation and a loss of regulatory T cells. CD28 was previously known to be a pathway utilized primarily by effector T cells. The authors showed that CD28-dependent IL-2 production by effector T cells was required to support regulatory T cell survival and expansion. Another fine example is the description by Soriano (2) of the ROSA26 Cre reporter mouse. The construction and description of Cre reporter

mice has revolutionized our ability to see genes turn on and off in specific locations. Another example is an article by Kirchner and colleagues, which showed that the current model of the hunger hormone ghrelin as a factor that informs the brain about an empty stomach may need to be revised (3). The authors studied loss- and gain-of-function models of the ghrelin activator GOAT (ghrelin octanoyl acyl transferase) and found that dietary lipids can modify ghrelin molecules to regulate energy expenditure and adiposity.

These are but a few examples of the type of article we are looking for, with the same high quality and novel insight as our longer papers but appropriate for presentation in a more abbreviated format. Note that we do not mean to discourage authors from sending in longer manuscripts — sometimes it really does take 8,500 words and 9 figures to fully prove a hypothesis. We welcome your feedback and your submissions.

**Ushma S. Neill**  
Executive Editor

1. Tang Q, et al. CD28 controls peripheral homeostasis of CD4<sup>+</sup>CD25<sup>+</sup> regulatory T cells. *J Immunol.* 2003;171(7):3348–3352.
2. Soriano P. Generalized lacZ expression with the ROSA26 Cre reporter strain. *Nat Genet.* 1999; 21(1):70–71.
3. Kirchner H, et al. GOAT links dietary lipids with the endocrine control of energy balance. *Nat Med.* 2009;15(7):741–745.