Installing Read by QxMD

A guide for QUB students and staff

This step-by-step guide shows how to:

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- Create a Read account
- Link your Read account to your QUB log-in
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QxMD Medical Software Inc.

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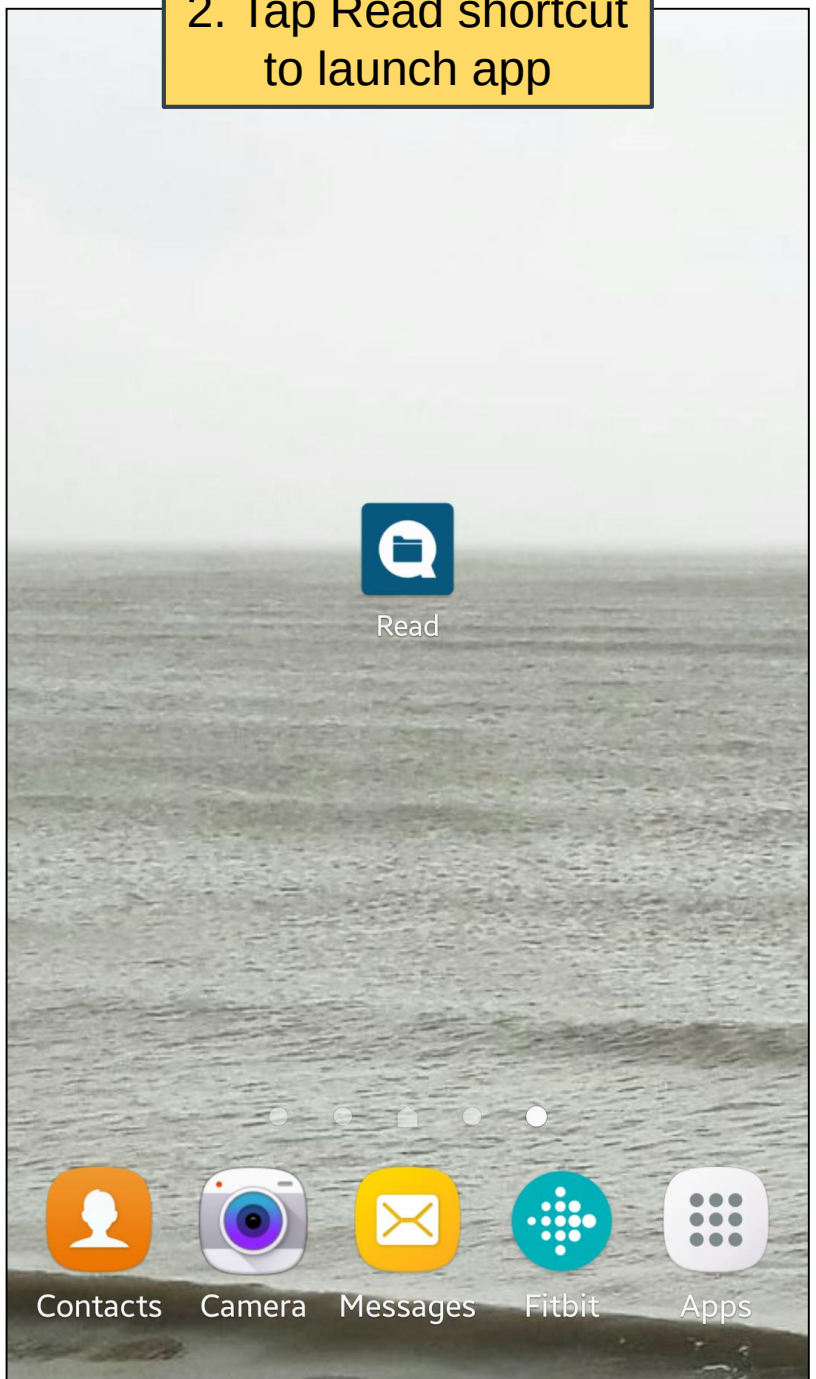
Read by QxMD keeps you up to date with breaking medical & scientific research.

Medical

2. Tap Read shortcut to launch app



3. Tap GET STARTED
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Read
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GET STARTED

LOG IN

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First Name

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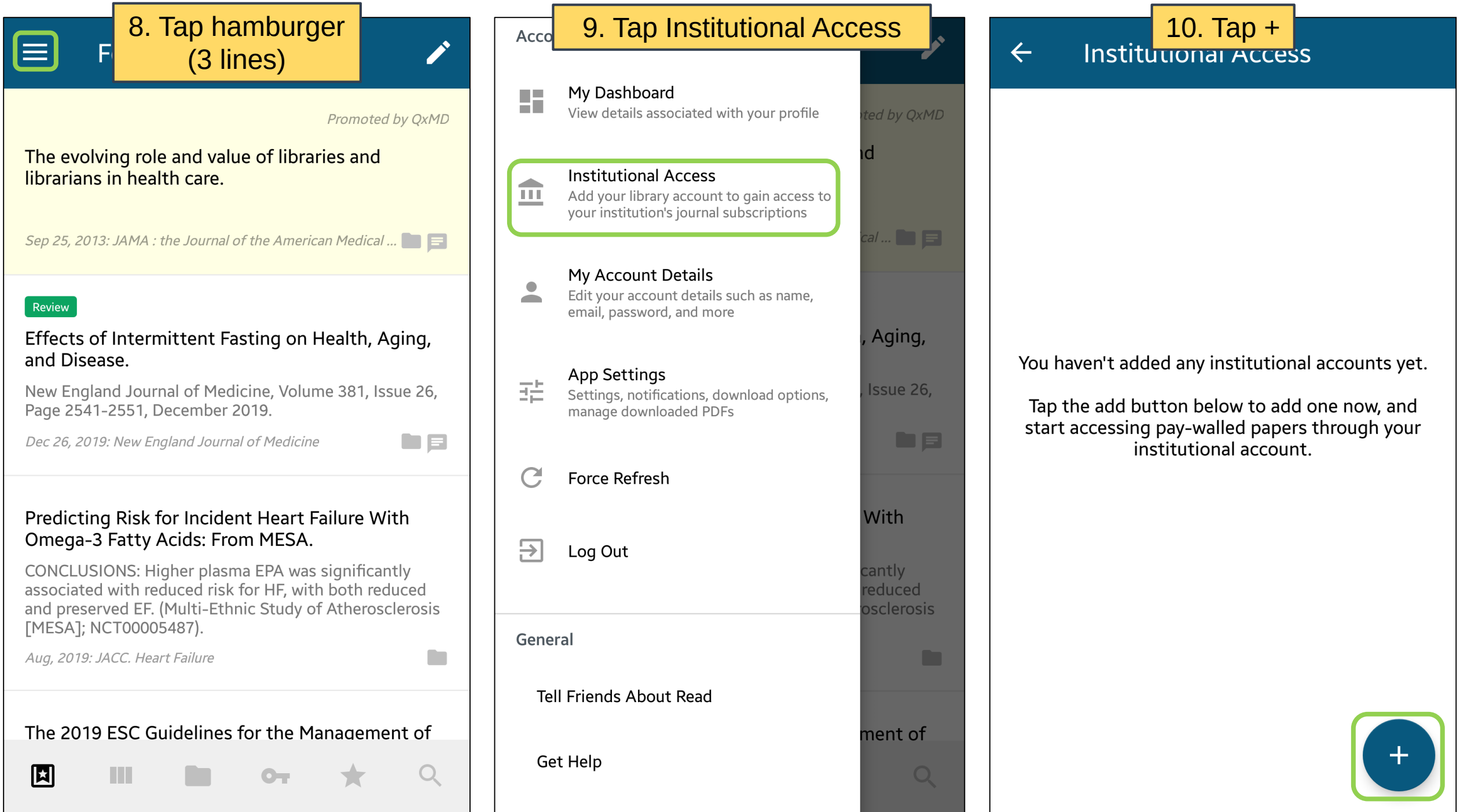
Create Account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Review
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Dec 26, 2019: New England Journal of Medicine
Predicting Risk for Incident Heart Failure With Omega-3 Fatty Acids: From MESA.
CONCLUSIONS: Higher plasma EPA was significantly associated with reduced risk for HF, with both reduced and preserved EF. (Multi-Ethnic Study of Atherosclerosis [MESA]; NCT00005487).
Aug, 2019: JACC. Heart Failure
The 2019 ESC Guidelines for the Management of
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11. Tap search

Select Your Institution

2

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Queen's University

Queen's University, Belfast

13. Choose Automatic Login Using Library Login

Select Your Login Preference

Automatic Login Using Library Login

Manual Login

14. Enter your QUB number and password, then tap tick

Username

Password

Your password is encrypted and saved locally on your device. It is never stored on or sent to our servers.

15. Tap SAVE AND LOG IN

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Would you like to log in to your institution now, to confirm the entered credentials work as expected?

SAVE AND LOG IN

JUST SAVE, DON'T LOG IN

CANCEL

16. Tap CONTINUE

Login Successful!

Login to your institution was successful! Your settings will now be saved.

CONTINUE

17. Tap back arrow

Institutional Access

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Tip: Long press a row to delete

18. The first time you access an article, tap ALLOW

New England Journal of Medicine. 2019 Dec 26; 381 (26) : 2541-2551.

Effects of Intermittent Fasting on Health, Aging, and Disease.

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Review

New England Journal of Medicine. 2019 Dec 26; 381 (26) : 2541-2551.

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The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

Dan L. Longo, M.D., *Editor*

Effects of Intermittent Fasting on Health, Aging, and Disease

Rafael de Cabo, Ph.D., and Mark P. Mattson, Ph.D.

ACCORDING TO WEINDRUCH AND SOHAL IN A 1997 ARTICLE IN THE *JOURNAL*, reducing food availability over a lifetime (caloric restriction) has remarkable effects on aging and the life span in animals.[1] The authors proposed that the health benefits of caloric restriction result from a passive reduction in the production of damaging oxygen free radicals. At the time, it was not generally recognized that because rodents on caloric restriction typically consume their entire daily food allotment within a few hours after its provision, they have a daily fasting period of up to 20 hours, during which ketogenesis occurs. Since then, hundreds of studies in animals and scores of clinical studies of controlled intermittent fasting regimens have been conducted in which metabolic switching from liver-derived glucose to adipose cell–derived ketones occurs daily or several days each week. Although the magnitude of the effect of intermittent fasting on life-span extension is variable (influenced by sex, diet, and genetic factors), studies in mice and nonhuman primates show consistent effects of caloric restriction on the health span (see the studies listed in Section S3 in the Supplementary Appendix, available with the full text of this article at NEJM.org).

Studies in animals and humans have shown that many of the health benefits of intermittent fasting are not simply the result of reduced free-radical production or weight loss.[2-5] Instead, intermittent fasting elicits evolutionarily conserved, adaptive cellular responses that are integrated between and within organs in a manner that improves glucose regulation, increases stress resistance, and suppresses inflammation. During fasting, cells activate pathways that enhance intrinsic defenses against oxidative and metabolic stress and those that remove or repair damaged molecules (Fig. 1).[5] During the feeding period, cells engage in tissue-specific processes of growth and plasticity. However, most people consume three meals a day plus snacks, so intermittent fasting does not occur.[2,6]

Preclinical studies consistently show the robust disease-modifying efficacy of intermittent fasting in animal models on a wide range of chronic disorders, including obesity, diabetes, cardiovascular disease, cancers, and neurodegenerative brain diseases.[3,7-10] Periodic flipping of the metabolic switch not only provides the ketones that are necessary to fuel cells during the fasting period but also elicits highly orchestrated systemic and cellular responses that carry over into the fed state to bolster mental and physical performance, as well as disease resistance.[11,12]

Here, we review studies in animals and humans that have shown how intermittent fasting affects general health indicators and slows or reverses aging and disease processes. First, we describe the most commonly studied intermittent-fasting regimens and the metabolic and cellular responses to intermittent fasting. We then present and discuss findings from preclinical studies and more recent clinical studies that tested intermittent-fasting regimens in healthy persons and in

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