Regarding the Article by Timmers et al

To the Editor:

In an article in Circulation Research, Timmers et al\(^1\) describe exaggerated left ventricular remodeling in p50 knockout (KO) mice. The results are in sharp contrast to all reports evaluating cardiac disease models in the p50 KO to date: tumor necrosis factor–overexpressing mice, but not p50 KO mice crossed with tumor necrosis factor–overexpressing mice, did develop heart failure.\(^2\) p50 KO mice did not develop cardiac hypertrophy after angiotensin stimulation.\(^3\) p50 KO mice were protected from ischemia/reperfusion injury.\(^4\) Regarding chronic myocardial infarction, we\(^5\) and others\(^6\) have described improved cardiac remodeling after myocardial infarction as measured by echocardiography. Timmers et al argue that the difference between their and the findings of others might be attributable to different imaging modalities (MRI in their study, echocardiography in the other studies); however, we cannot follow this argument. Although minor differences may be explained by the more thorough imaging using MRI, this cannot account for a total reversal of the results obtained by 2 other independent groups. It appears more likely that the strain of mice used in these experiments could be responsible: we had backcrossed the p50 KO mice for 10 generations to a C57BL/6 background, whereas Timmers et al used a mixed background (129Bl6) and bought control mice. Inappropriate controls resulting from a lack of backcrossing thus may account for the opposite results compared with all studies published to date.

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References

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