**Supplementary Methods and Table**

***Hemodynamics and infarct size measurements***

After the rats had been anesthetized with Zoletil-xylazine (20 mg/kg-9 mg/kg) intraperitoneally at the end of the study, the hemodynamic parameters were measured. A polyethylene Millar catheter was inserted into the left ventricle and connected to a transducer (Model SPR-407, Miller Instruments, Houston, TX). LV systolic and diastolic pressures were recorded as the mean of five consecutive pressure cycle measurements as described previously (1). In addition, the maximum rates of rise (+dP/d*t*) and decrease (-dP/d*t*) in LV pressure were also calculated. Electrophysiological tests were performed after the arterial pressure measurements had been made. After the electrophysiological tests had been completed, the atria and right ventricles of the hearts were trimmed off, and the left ventricles were rinsed in cold physiological saline, weighed, and flash frozen in liquid nitrogen. A section taken from the equator of the LV, fixed in 10% formalin, embedded in paraffin, and stained with hematoxylin and eosin and trichrome to determine the size of the infarct size as previously described (2).

***Ex vivo electrophysiological studies***

We then performed programmed electrical stimulation to evaluate the possible arrhythmogenic risk of sympathetic innervation. We used the Langendorff heart perfusion technique in order to avoid any potential confounding effects of post-MI hormonal activation on pacing-induced ventricular arrhythmias as previously reported (3). Modified Tyrode’s solution was used to perfuse the hearts at a constant flow rate of 4 ml/min and temperature of 37°C. Ventricular and atrial epicardial electrocardiograms were continuously recorded. After the hearts had been perfused, they were observed for 10 minutes to allow the contraction and rhythm to stabilize. A Bloom stimulator (Fischer Imaging Corporation, Denver, CO, USA) was used to generate pacing pulses, with a 120-ms cycle length (S1) for eight beats, followed by one to threeextrastimuli (S2, S3, and S4) at shorter coupling intervals to induce ventricular arrhythmias.The induction of ventricular tachyarrhythmia was defined as being the endpoint of ventricular pacing. Nonsustained ventricular tachyarrhythmias including ventricular fibrillation and tachycardia were considered to be those that lasted for ≤15 beats, and sustained ventricular tachyarrhythmias were considered to be those that lasted for >15 beats. We used a modified arrhythmia scoring system as previously reported (4). When more than one form of arrhythmia was observed in a single heart, the one with the highest score was used. The experiments were usually completed in under 10 minutes.

#### ***Western blot analysis of NF-κB, NLRP3*, *caspase-1, IL-1β and NGF***

Protein levels of NLRP3, NF-κB, caspase-1, and IL-1β were measured in myocardium from the border zone at day 3, and the protein level of NGF was measured in myocardium from the remote zone at day 28. The myocardium was homogenized in three volumes of Tris-buffered saline containing phosphatase and protease inhibitors (25 mM Tris-HCl, pH 7.4, 1 mM EDTA, 1 mM phenylmethylsulfonyl fluoride, 30 mM β-glycerophosphate, 1 mM EGTA, 150 mM NaCl, 5 mM sodium pyrophosphate, 30 mM sodium fluoride). The homogenates and supernatant were centrifuged for 10 minutes at 500×g and 4°C and 2000×g for 10 minutes, respectively. The pellets were resuspended in the same buffer and used as the nuclear fraction. The supernatant was then centrifuged for 1 h at 40,000×g and subsequently used as the cytosolic fraction. After separation of 20 μg of protein using 10% SDS-PAGE, the proteins were electrotransferred onto a nitrocellulose membrane and incubated with antibodies. After rinsing the membrane with a blocking solution, it was incubated for 2 hours at room temperature. Antigen-antibody complexes were detected using 5-bromo-4-chloro-3-indolyl-phosphate and nitroblue tetrazolium chloride (Sigma). A scanning densitometer was then used to volume-integrated films within the linear range of the exposure. All experiments were repeated three times, and the results were expressed as mean values.

The following primary antibodies were used in this study: NF-κB (p65) (nuclear fraction, Santa-Cruz Biotechnology), NLRP3 (Santa-Cruz Biotechnology), cleaved caspase-1 (Santa-Cruz Biotechnology), cleaved IL-1β (Cell Signaling Technology), NGF (Chemicon), and β-actin (Sigma-Aldrich).

#### ***Immunohistochemical studies of NF-κB, NLRP3, CD68, IL-1β, tyrosine hydroxylase, growth associated factor 43 and neurofilament***

To better understand NF-κB functionality, immunohistochemical staining was employed using an antibody (Santa-Cruz Biotechnology) against the nuclear localization sequence of the p65 subunit at the day 3 from the border zone. We counted 200 cells in each field of view to measure the ratio of positively stained cells.

To confirm inflammasome activation in the myocardium, double-staining with NLRP3 and IL-1β was performed at the day 3 from the border zone.

During the inflammatory phase of wound healing, macrophages have been shown to be a major source of IL-1β (5). Therefore, we then double-stained macrophages with IL-1β to evaluate the function of macrophages from the border zone at day 3. Five-µm thick cryosections were prepared and incubated with antibodies against CD68 (a general marker of all macrophages; Abcam, Cambridge, MA) and IL-1β (Cell Signaling Technology). These antibodies have been tested for specificity in rats. Directlyconjugated antibodies with identical isotypes were used as negative controls. The average of 10 random scans per section was used for analysis. The results were quantified as the ratio of positively stained area to the total area at 400× magnification.

 Myocardium from the remote zone at day 28 was subjected to immunofluorescence staining to evaluate the quantification and spatial distribution of sympathetic nerve fibers. Five-µm thick sections of paraffin-embedded tissues were prepared and incubated with anti-growth associated protein 43 (a marker of nerve sprouting, 1:400; Chemicon, CA, USA), anti-tyrosine hydroxylase (1:200; Chemicon, CA, USA), and anti-neurofilament antibodies (a marker of sympathetic nerves, 1:1000; Chemicon, CA, USA) in 0.5% bovine serum albumin in phosphate-buffered saline overnight at 37°C. Monoclonal goat anti-mouse IgG conjugated to rhodamine for growth associated protein 43 and neurofilaments, and fluorescein isothiocyanate for tyrosine hydroxylase was used as the second antibody. Directlyconjugated antibodies with identical isotypes were used as negative controls. Nerve density was measured on tracings used computerized planimetry (Image Pro Plus, Media Cybernetics, Silver Spring, Maryland, USA) as described previously (6), and qualitatively estimated from 10 randomly selected fields at a magnification of 400×. The results were expressed as the ratio of labeled nerve fiber area to total area.

**Supplementary Table**

Cardiac morphology, hemodynamics and tissue NE concentrations at 3 days post MI

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
|  | **Sham** |  |  |  | **Infarction** |
| **Parameters** | **Saline** | **Taurine** |  |  | **Vehicle** | **Taurine** |
| No. of rats | 7 | 7 |  |  | 7 | 8 |
| Body weight, g | 352 ± 14 | 362 ± 18 |  |  | 348 ± 19 | 351 ± 15 |
| Heart rate, bpm | 395 ± 22 | 373 ± 29 |  |  | 405 ± 24 | 411 ± 21 |
| LVESP, mm Hg | 109 ± 8 | 107 ± 9 |  |  | 98 ± 11 | 101 ± 12 |
| LVEDP, mm Hg | 5 ± 1 | 5 ± 3 |  |   | 21 ± 5\* | 18 ± 8\* |
| +dp/d*t*, mm Hg/sec | 7988 ± 302 | 8152 ± 342 |  |  | 2652 ± 298\* | 3266 ± 316\*† |
| -dp/d*t*, mm Hg/sec | 7128 ± 279 | 7082 ± 322 |  |  | 2064 ± 302\* | 2872 ± 289\*† |
| Infarct size, % | … | … |  |  | 37 ± 2 | 38 ± 5 |
| LVW/BW, mg/g | 1.99 ± 0.21 | 2.05 ± 0.31  |  |  | 3.62 ± 0.57\* | 3.39 ± 0.62\* |
| RVW/BW, mg/g | 0.64 ± 0.21 | 0.71 ± 0.33  |  |  | 0.92 ± 0.25 | 0.85 ± 0.31 |
| LungW/BW, mg/g | 4.08 ± 0.45 | 4.52 ± 0.51  |  |  | 6.36 ± 0.72\* | 5.68 ± 0.49† |
| LV NE, μg/g protein | 1.21 ± 0.27 | 1.13 ± 0.18 |  |  | 1.04 ± 0.23 | 1.16 ± 0.26 |

Values are mean ± SD. BW, body weight; LungW, lung weight; LVEDP, left ventricular end-diastolic pressure; LVESP, left ventricular end-systolic pressure; LVW, left ventricular weight; RVW, right ventricular weight. \**p* < 0.05 compared with respective sham; †*p* < 0.05 compared with vehicle-treated infarcted group.

**References.**

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