Supplementary Figure 1. Osteoporotic phenotype of \textit{Fcgr3}^{-/-} mice due to the suppression of osteoclast formation by Fc\textgammaRIII.  
\textbf{(a)} mRNA expression of the four Fc\textgammaRs during the course of osteoclastogenesis.  
\textbf{(b)} BMD measured in 20 longitudinal sectional divisions of the femurs of Fcgr3^{+/+} and Fcgr3^{-/-} mice by DEXA (n=9).  
\textbf{(c)} The trabecular number and thickness of the femurs of Fcgr3^{+/+} and Fcgr3^{-/-} mice determined by microcomputed tomography (n=9).  
\textbf{(d)} Bone formation, as observed by calcein double labeling at an interval of 4 d. Representative data of nine mice are shown. The parameters for osteoblastic bone formation, as determined by bone histomorphometric analysis (n=9).  
\textbf{(e)} BrdU incorporation in BMMs stimulated with M-CSF alone or RANKL/M-CSF for the indicated periods. Data are representative of more than 3 independent experiments with triplicate culture wells.  
\textbf{(f)} \textit{In vitro} differentiation of osteoclasts of Fcgr3^{+/+} and Fcgr3^{-/-} BMMs in coculture with osteoblasts. Data are representative of more than 3 independent experiments with triplicate culture wells. All data are shown as the mean ± s.e.m. Statistical analyses were performed using unpaired two-tailed Student’s t-test (*, P < 0.05; **, P < 0.01; n.s., not significant).
Supplementary Figure 2. Serum level of IgGs in Fcgr3−/− mice and FcγRI and FcγRIV expressions in Fcgr3−/− osteoclast precursor cells. (a) The concentration of IgG1 and IgG2a in the serum of control and Fcgr3−/− mice (n=9). (b) The mRNA expression of FcγRI and FcγRIV in control and Fcgr3−/− cells 24 h after RANKL stimulation. Data are representative of three independent experiments with triplicate samples. All data are shown as the mean ± s.e.m. Statistical analyses were performed using unpaired two-tailed Student’s t-test (n.s., not significant).
**Supplementary Figure 3.** The role of FcRγ-mediated signaling in compensation for the lack of DAP12-mediated costimulatory signaling. (a) *In vitro* osteoclastogenesis of *Tyrobp*−/− and *Tyrobp*−/−*Fcgr3*−/− cells. Representative data (left) and quantification (n=3, right) of three independent experiments are shown. In the absence of osteoclast supporting cells, including osteoblasts, *Tyrobp*−/− cells do not undergo osteoclast differentiation, because the FcRγ-mediated ITAM signal is not sufficiently activated in this culture system. However, deletion of the *Fcgr3* gene restored the osteoclast differentiation of *Tyrobp*−/− cells. (b) The retroviral introduction of FcγRIII (pMX-FcγRIII) suppressed osteoclastogenesis in *Tyrobp*−/− cells (left). Data are representative of three independent experiments with triplicate culture wells. The expression of FcγRIII was analyzed in osteoclast precursor cells 48 h after RANKL stimulation by flow cytometry (right). Data are representative of three independent experiments. (c) Deletion of the *Fcgr3* gene promoted the activation of ITAM
signaling and NFATc1 induction in Tyrobp−/− cells. RANKL-induced phosphorylation of PLCγ2 (left) and calcium oscillations (middle) in Tyrobp−/− and Tyrobp−/−Fcgr3−/− cells. NFATc1 induction in during osteoclastogenesis (right). All data are representative of three independent experiments. (d) Deletion of the Fcgr3 gene did not rescue the bone resorbing activity of mature osteoclasts derived from wild-type (WT), Tyrobp−/− and Tyrobp−/−Fcgr3−/− cells. (e) The crosslinking of FcγR by plate-bound IgG2a rescued osteoclastogenesis in Tyrobp−/− cells. Representative data (left) and quantification (n=3, right) of three independent experiments are shown. (f) The IgG1 IC (TNP-BSA together with α-TNP IgG1) rescued osteoclastogenesis in Tyrobp−/−Fcgr2b−/− cells. Representative data (left) and quantification (n=3, right) of three independent experiments are shown. (g) The crosslinking of FcγRγ promoted phosphorylation of PLCγ2 and NFATc1 induction in Tyrobp−/− cells. Data are representative of three independent experiments. (h) The crosslinking of FcγRγ did not rescue the bone resorbing activity of the mature Tyrobp−/− osteoclasts. Data are representative of three independent experiments. FcγRγ-mediated ITAM signaling compensates for the lack of DAP12 signaling. However, the activation of neither Ig-like receptor- (a-c) nor FcγRs-mediated (e-g) FcγRγ signaling rescued the bone resorption activity (d and h), suggesting that the enhancement of FcγRγ signaling compensates for osteoclast differentiation, but not function. All data are shown as the mean ± s.e.m. Statistical analyses were performed using unpaired two-tailed Student’s t-test (*, P < 0.05; **, P < 0.01; ***, P < 0.001; n.s., not significant).

Supplementary Figure 3 continued.
Supplementary Figure 4. Bone phenotype of Fcgr2b⁻/⁻ mice. (a) BMD measured in 20 longitudinal sectional divisions of the femurs of Fcgr2b⁺/⁺ and Fcgr2b⁻/⁻ mice by DEXA (n=10). (b) The trabecular number and thickness of the femurs of Fcgr2b⁺/⁺ and Fcgr2b⁻/⁻ mice determined by microcomputed tomography (µCT) (n=10). (c) Bone formation, as observed by calcein double labeling at an interval of 4 d. Representative data of ten mice are shown. The parameters for osteoblastic bone formation, as determined by bone histomorphometric analysis (n=10). (d) Osteoclastogenesis of Fcgr2b⁺/⁺ and Fcgr2b⁻/⁻ cells cultured in the presence of RANKL and M-CSF (left panel) or cocultured with osteoblasts (right panel) in the presence of 10% FBS. Data are representative of three independent experiments with triplicate culture wells. (e) Increase in the serum level of the IgG subclasses in Fcgr2b⁻/⁻ mice with age. Twenty-week-old wild-type (WT) mice were analyzed as control. Each dot indicates a single mouse. (f) µCT of the proximal femur of 6-week-old Fcgr2b⁺/⁺ and Fcgr2b⁻/⁻ mice (top left, axial view of the metaphyseal region; bottom left, longitudinal view). Representative data of ten mice are shown. Bone volume, trabecular thickness, trabecular number and trabecular separation were determined by µCT analysis (right). (g) The parameters for osteoclastic bone resorption, as determined by bone morphometric analysis (n=10). (h) The parameters for osteoblastic bone formation, as determined by bone morphometric analysis (n=10). All data are shown as the mean ± s.e.m. Statistical analyses were performed using unpaired two-tailed Student’s t-test (*, P < 0.05; **, P < 0.01; ***, P < 0.001; n.s., not significant).
Supplementary Figure 5. Bone phenotype of Fcgr2b+/−Fcer1g−/− mice. (a) The parameters for osteoclastic bone resorption in Fcgr2b+/−Fcer1g+/− (Control), Fcgr2b+/−Fcer1g−/− and Fcgr2b−/−Fcer1g−/− (DKO) as determined by bone morphometric analysis (n=9). (b) The parameters for osteoblastic bone formation, as determined by bone morphometric analysis (n=9). All data are shown as the mean ± s.e.m. Statistical analyses were performed using unpaired two-tailed Student’s t-test (*, P < 0.05; **, P < 0.01; n.s., not significant).
Supplementary Figure 6. The effect of the sialylation of IgGs on osteoclastogenesis. (a) The glycosilation status of IgGs purified from Fcgr2b−/− and control mice. Sialylation and the core glycan of IgGs were detected by blotting with sumbuccus nigla lectin (SNA) and lens culinaris agglutinin (LCA), respectively. Data are representative of three independent experiments. (b) The effect of sialylated IgGs on osteoclastogenesis. BMMs were cultured on plates coated with the same amount of native (non-treated) and desialylated IgGs. Representative data (left) and quantification (n=3, right) of three independent experiments are shown. (c) Desialylation of IgGs purified from Fcgr2b−/− and control mice. Data are representative of three independent experiments. Data are shown as the mean ± s.e.m. Statistical analyses were performed using unpaired two-tailed Student’s t-test (*, P < 0.05; ***, P < 0.001; n.s., not significant).
Supplementary Figure 7. The knockdown efficiency of shRNA for FcγRs. The mRNA expression of FcγRI, FcγRIIB, FcγRIII and FcγRIV in BMMs transduced with a retroviral vector expressing shRNA for the corresponding FcγRs. Data are representative of three independent experiments with triplicate samples and are shown as the mean ± s.e.m. Statistical analyses were performed using unpaired two-tailed Student’s t-test (*, P < 0.05; **, P < 0.01; n.s.).
Supplementary Figure 8.  Direct effect of immune complex on osteoclastic bone resorption.  (a) The osteoclast number, eroded surface and bone volume in calvarial bone of wild-type (WT), Fcgr2b<sup>−/−</sup>, Fcgr3<sup>−/−</sup> and Fcer1g<sup>−/−</sup> mice 2 d after injection of TNP-BSA alone (Control), TNP-BSA with α-TNP-IgG1 (IgG1 IC) or TNP-BSA with α-IgG2a (IgG2a IC) (n=9).  (b) Inflammatory cell infiltration in calvarial bone of wild-type (WT), Fcgr2b<sup>−/−</sup>, Fcgr3<sup>−/−</sup> and Fcer1g<sup>−/−</sup> mice 2 d after injection of TNP-BSA alone (Control), TNP-BSA with α-TNP-IgG1 (IgG1 IC) or TNP-BSA with α-IgG2a (IgG2a IC) (left, n=9).  LPS was injected into WT mice as control (n=9).  The bone destruction image of the calvarial bone in LPS-injected mice (right).  (c) Bone morphometric analysis of the tibiae of Fcgr2b<sup>+/−</sup> and Fcgr2b<sup>−/−</sup> mice intravenously injected with TNP-BSA alone (Control) or IgG1 IC.  Representative data of ten mice are shown.  (d) The parameters for osteoclastic bone resorption in mice injected with the IgG1 IC, as determined by bone morphometric analysis (n=10).  (e) The parameters for osteoblastic bone formation in mice injected with the IgG1 IC (n=10).  All data are shown as the mean ± s.e.m.  Statistical analyses were performed using unpaired two-tailed Student’s t-test (*, P < 0.05; **, P < 0.01; n.s., not significant).
Supplementary Figure 9. The compensation mechanism of DAP12-mediated signals for the loss of FcRγ. (a) The mRNA expression of DAP12 in Fcer1g<sup>+</sup> and Fcer1g<sup>−</sup> BMMs. (b) Hyperactivation of PLCγ2 in Fcer1g<sup>−</sup> BMMs. (c) The mRNA expression of DAP12-associating Ig-like receptors, such as TREM2 and SIRPβ1, in Fcer1g<sup>+</sup> and Fcer1g<sup>−</sup> BMMs. All data are representative of more than three independent experiments with triplicate samples and are shown as the mean ± s.e.m. Statistical analyses were performed using unpaired two-tailed Student’s t-test (*, P < 0.05; **, P < 0.01; n.s., not significant).
Supplementary Figure 10. Full blots of the indicated figures.

Fig. 2a left (α-phospho-PLCγ2)  
Fig. 2a left (α-PLCγ2)  
Fig. 2a right (α-NFATc1)  
Fig. 2a right (α-β-actin)  
Fig. 2e (α-FcRγ)  
Fig. 2e (α-FcγRIII)  
Fig. 2g (α-OSCAR)  
Fig. 2g (α-β-actin)  
Fig. 2h (α-OSCAR)  
Fig. 2h (α-PIR-A)  
Fig. 2h (α-FcRγ)
Fig. 2i left (α-OSCAR)

Fig. 2i middle and right (α-PIR-A/B)

Fig. 2i left, middle and right (α-β-actin)

Fig. 5b left (α-phospho-PLCγ2)

Fig. 5b right (α-NFATc1)

Fig. 5b left (α-PLCγ2)

Fig. 5b right (α-β-actin)

Fig. 6e left (α-phospho-PLCγ2)

Fig. 6e left (α-PLCγ2)

Supplementary Figure 10 continued.
**Supplementary Figure 2c left (α-phospho-PLCγ2)**  
Supplementary Figure 2c left (α-PLCγ2)

**Supplementary Figure 2c right (α-NFATc1)**  
Supplementary Figure 2c right (α-β-actin)

**Supplementary Figure 2g left (α-phospho-PLCγ2)**  
Supplementary Figure 2g left (α-PLCγ2)

**Supplementary Figure 2g right (α-NFATc1)**  
Supplementary Figure 2g right (α-β-actin)

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**Supplementary Figure 10 continued.**
Supplementary Fig. 5a (SNA)  

Supplementary Fig. 5a (LCA)  

Supplementary Fig. 5a (IgG)  

Supplementary Fig. 5c (SNA)  

Supplementary Fig. 5c (IgG)  

Supplementary Fig. 9b (α-phospho tyrosine)  

Supplementary Fig. 9b (α-DAP12)  

Supplementary Figure 10 continued.