# Phospho-CrkL (Tyr207) (G4) rabbit mAb

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**Catalog:** #2091 **Store at:** -20ºC

*For Research Use Only. Not For Use In Diagnostic Procedures.*

| Applications | Detection | Clonality | Isotype |
|---|---|---|---|
| Flow Cytometry,WB | Anti-Rabbit IgG | Monoclonal | Rabbit IgGk |

**Format:** Unconjugated

**Cross Reactivity:** Predicted to work with mouse, rat, and other homologues.

**Formulation:** 1X PBS, 0.02% NaN3, 50% Glycerol, 0.1% BSA

**Preparation:** Protein A+G

**Reactivity:** Human,Mouse

**Recommended Usage:** 1µg/mL – 0.001µg/mL. It is recommended that the reagent be titrated for optimal performance for each application. See product image legends for additional information.

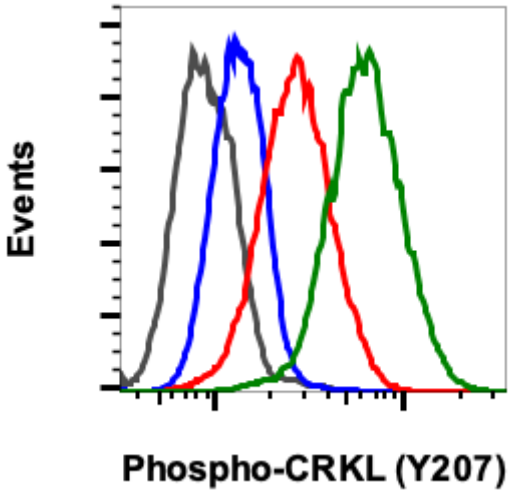
**Immunogen:** A synthetic phospho-peptide corresponding to residues surrounding Tyr207 of human phospho CrkL

**Description:** CrkL (v-Crk sarcoma virus CT10 oncogene-like protein) is an adaptor protein composed of one Src Homology 2 (SH2) and two Src Homology 3 (SH3) domains separated by flexible linker sequences that act as building blocks to assemble multiprotein complexes (1). The Crk adaptor proteins (Crk and CrkL) constitute an integral part of a network of essential signal transduction pathways in humans and other organisms that act as major convergence points in tyrosine kinase signaling. CRKL is required for the normal development of multiple tissues that rely on fibroblast growth factor 8 (FGF8). Phosphorylation of Crk on Tyr 221 or CrkL on Tyr 207 causes intramolecular binding of the linker region to the SH2 domain, sequestering the SH2 and SH3N and preventing them from binding target proteins (2,3). Mounting evidence indicates that dysregulation of Crk proteins is associated with human diseases, including cancer and susceptibility to pathogen infections.

**References:**

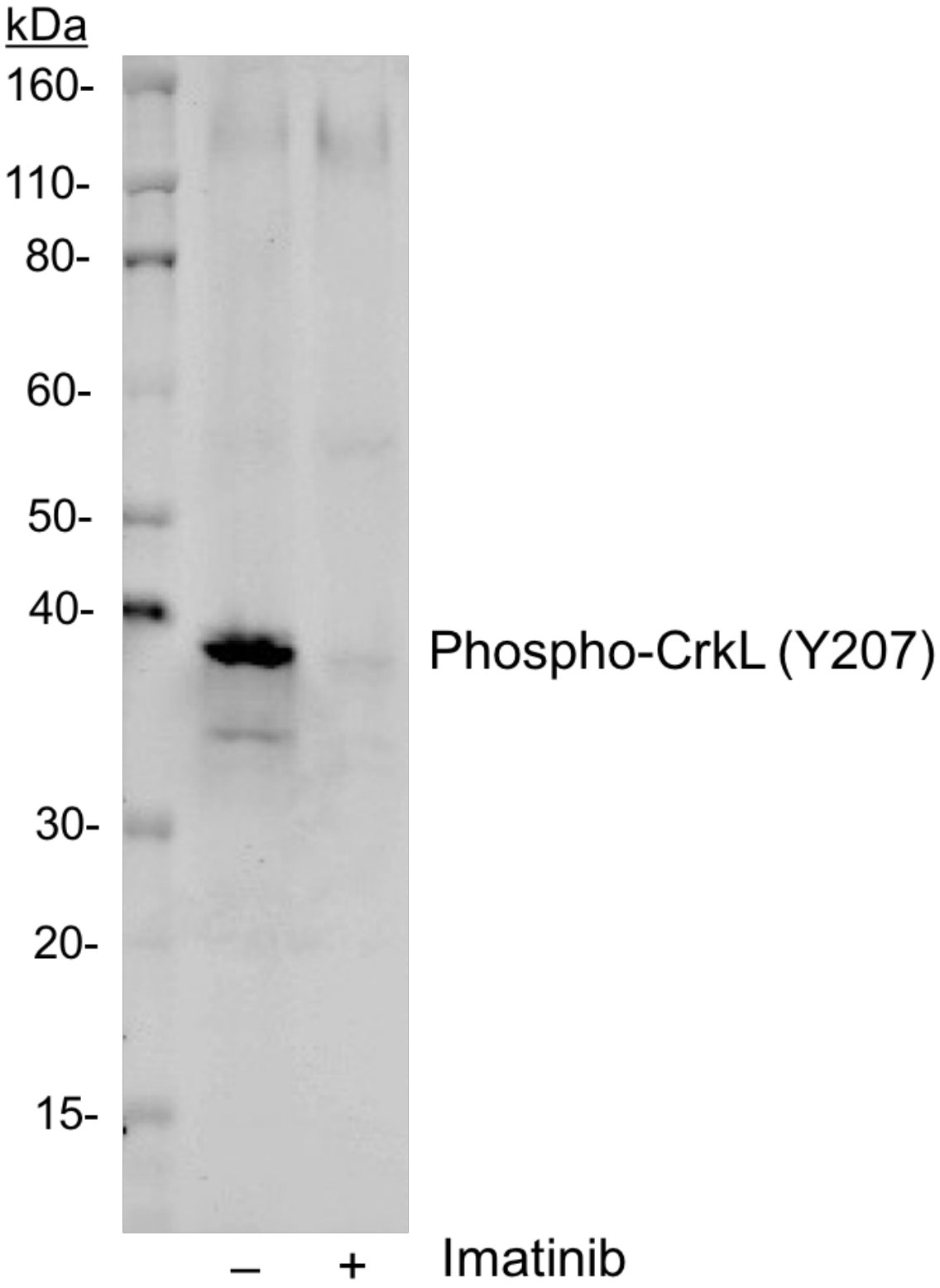
1. Tten Hoeve, J.,et al., (1993). Oncogene 8: 2469-2474.
2. Rosen MK, et al., (1995) Nature, 374 477-479.
3. Kobashigawa Y, et al., (2007) Nat Struct Mol Biol.14:503-510.

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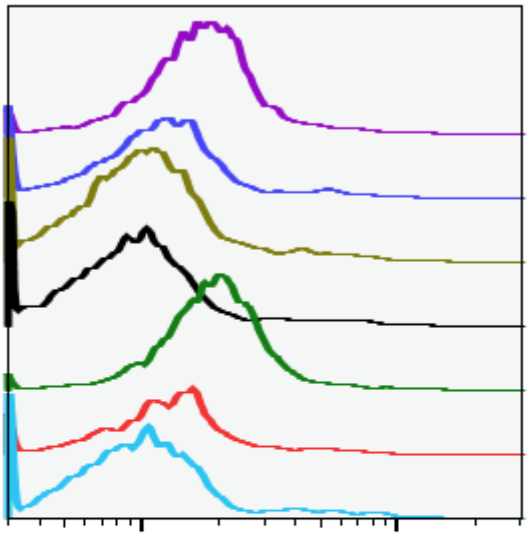



Flow cytometric analysis of K562 cells treated with imatinib (red) or treated with pervanadate (green) using Phospho-CRKL (Tyr207) (G4) Rabbit mAb CRKLY207-G4 #2091 at 0.05 ug/mL, or concentration-matched Rabbit (G9) mAb IgG Isotype Control #2141 for cells treated with imatinib (black) or treated with pervanadate (blue).

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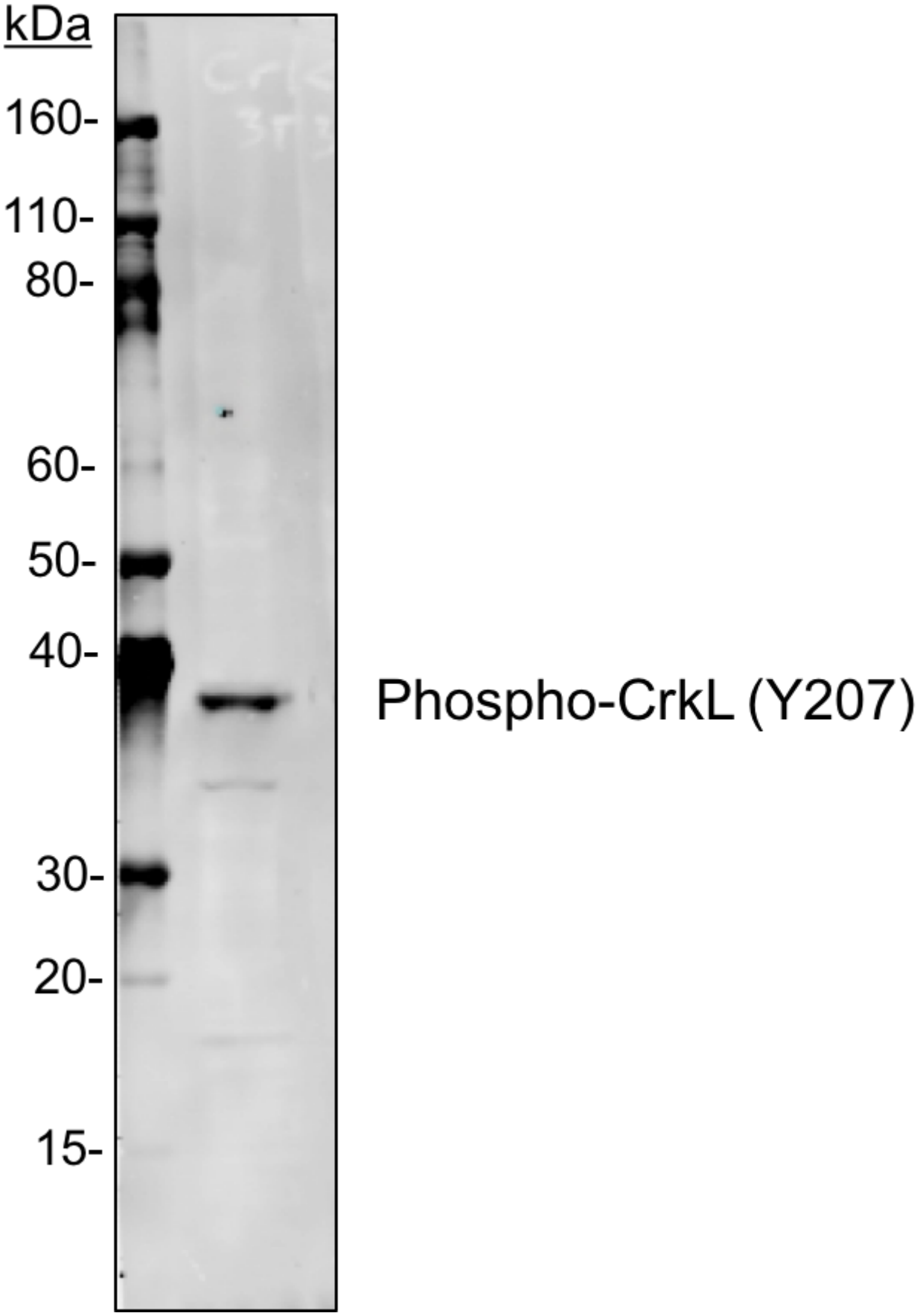



Western blot analysis of K562 cell extract untreated or treated with imatinib using 1ng/mL CrkL (Tyr207) antibody CrkLY207-G4. Cat. #2091.

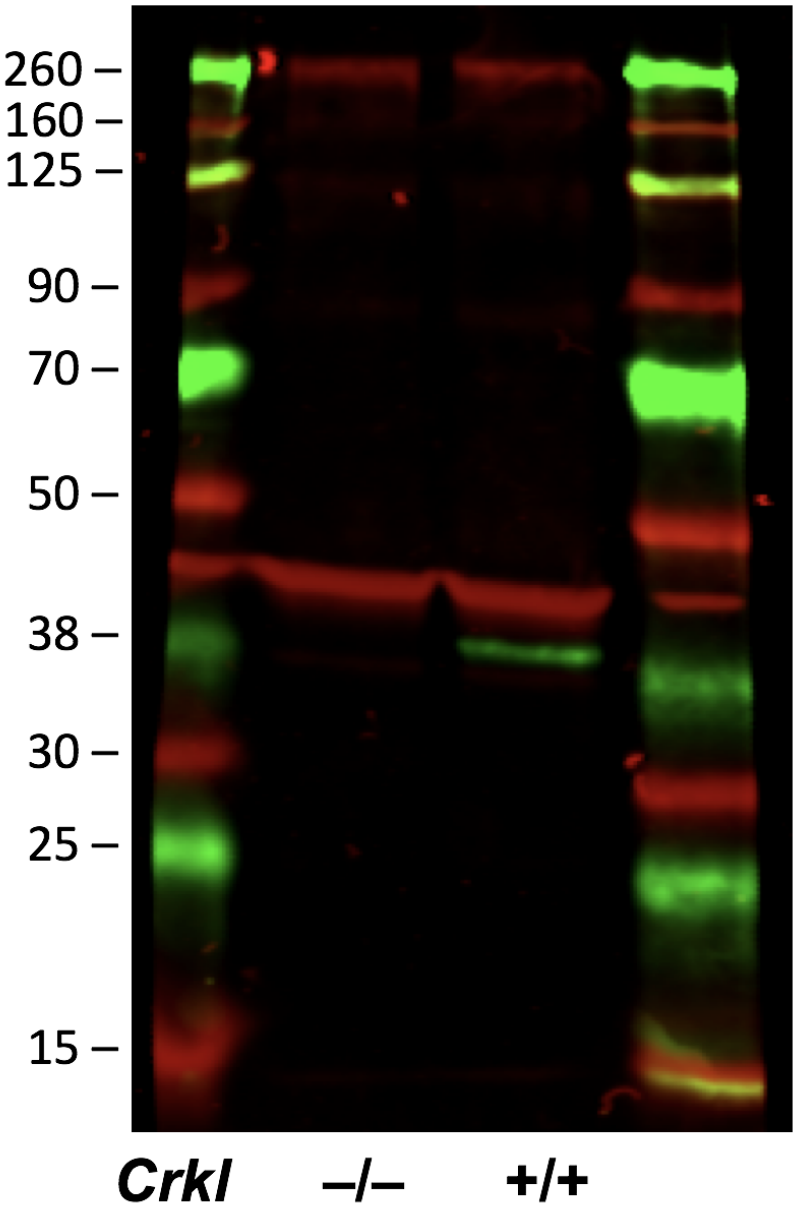


| | SampleID | Median : BL1-A |
|---|---|---|
| ☐ | Pv G4 N | 1712 |
| ☐ | Imat G4 N | 1165 |
| ☐ | Pv G4 P | 961 |
| ☐ | Imat G4 P | 870 |
| ☐ | Pv G4 | 1897 |
| ☐ | Imat G4 | 1272 |
| ☐ | Imat 2' only | 963 |

Peptide blocking flow cytometric analysis of K562 cells secondary antibody only negative control (light blue) or treated with imatinib (red) or treated with pervanadate (green) or imatinib and blocked with phospho-peptide (black) or pervanadate and blocked with phospho peptide (gold) or imatinib and blocked with non-phospho peptide (dark blue) or pervanadate and blocked with non-phospho peptide (purple) using Phospho-CrkL (Tyr207) antibody CrkLY207-G4 at 0.01µg/mL. Cat. #2091.

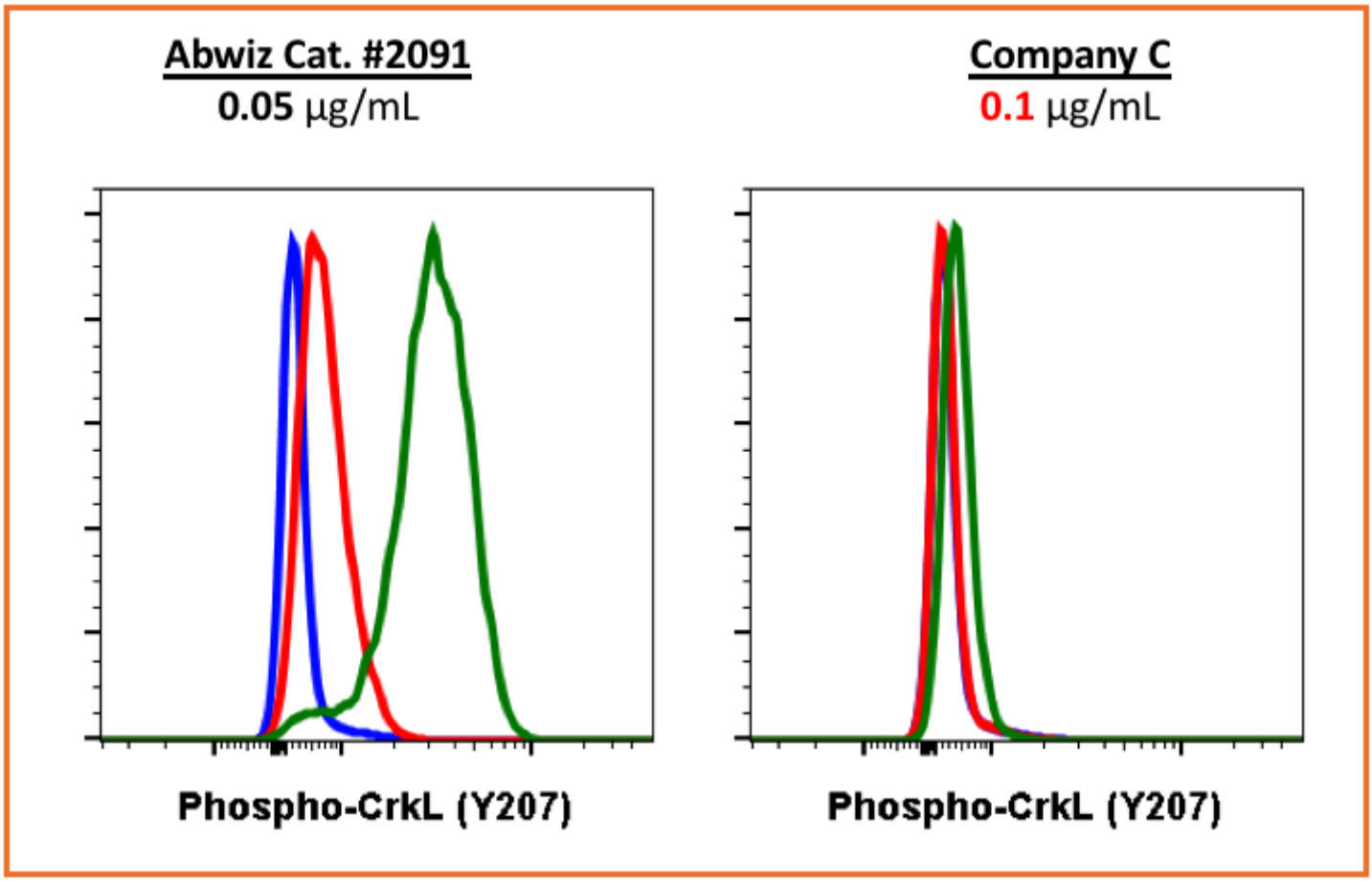



Western blot analysis of NIH3T3 cell extract using 1µg/mL Phospho-CrkL (Tyr207) antibody CrkLY207-G4. Cat. #2091.




Western blot of E10.5 mouse wild-type (+/+) or Crkl knock out (-/-) whole embryos. The red channel was stained using a β-actin loading control and the green channel was stained using 1:500 dilution of Phospho-CrkL (Tyr207) antibody CrkLY207-G4 Cat. #2091. Phospho CrkL antibody staining is absent in the knock out lysate.

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Flow cytometric analysis of K562 cells secondary antibody only negative control (blue) or treated with imatinib (red) or with pervanadate (green) using 0.05 µg/mL of Phospho-CrkL (Tyr207) antibody CrkLY207-G4 (Abwiz Cat. #2091) or Company C antibody at 0.1ug/mL.