Resource Summary Report

Generated by NIF on Sep 17, 2026

pKK-BiFC-Venus

RRID:Addgene_105804
Type: Plasmid

Proper Citation

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Plasmid Information

URL: http://www.addgene.org/105804

Proper Citation: RRID:Addgene_105804

Bacterial Resistance: Ampicillin

Defining Citation: PMID:29590189

Vector Backbone Description: Backbone Marker:Sammarco & Grabczyk (PMID: 16212928); Vector Backbone:BI-16 (constructed based on pcDNA5/FRT/TO from Invitrogen); Vector Types:Mammalian Expression, Flp-In competent; Bacterial Resistance:Ampicillin

Comments: Addgene has found the VN173 mutation (I152L) is not present in this plasmid. This vector is a member of the pKK vector family. It enables concomitant expression of two genes, which transcription is driven by a tetracycline responsive bidirectional CMV promoter. This vector is a derivate of the BI16 plasmid (PMID: 16212928) and is suitable for stable cell line generation using the Flp-In system (see Szczesny RJ et al. for detailed description and protocols; bioRxiv, doi: https://doi.org/10.1101/160101). pKK-BiFC-Venus vector encodes fragments of Venus (VN173, VC155) that become fluorescent when they reassemble into full fluorescent protein. Thus, it is useful for protein-protein interactions studies involving bimolecular fluorescence complementation approach (BiFC) (PMID: 16454041, 18846096). Here, an improved version of VN173 fragment is used (I152L) to inhibit self-assembly (PMID:21091444). Venus fragments coding sequences were cloned from Addgene ID: 22010 and 22011 and VN173 was subjected to site-directed mutagenesis to introduce mutation I152L.

Plasmid Name: pKK-BiFC-Venus

Record Creation Time: 20220422T221513+0000

Record Last Update: 20260912T091420+0000

Ratings and Alerts

No rating or validation information has been found for pKK-BiFC-Venus.

No alerts have been found for pKK-BiFC-Venus.

Data and Source Information

Source: Addgene

Usage and Citation Metrics

We found 2 mentions in open access literature.

Listed below are recent publications. The full list is available at NIF .

Jahid S, et al. (2022) Structure-based design of CDC42 effector interaction inhibitors for the treatment of cancer. Cell reports, 39(1), 110641.

Hao Z, et al. (2021) ??-Glutamyl carboxylase mutations differentially affect the biological function of vitamin K-dependent proteins. Blood, 137(4), 533.