

Pik3c2a Cas9-CKO Strategy

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Project Overview

Project Name

Pik3c2a

Project type

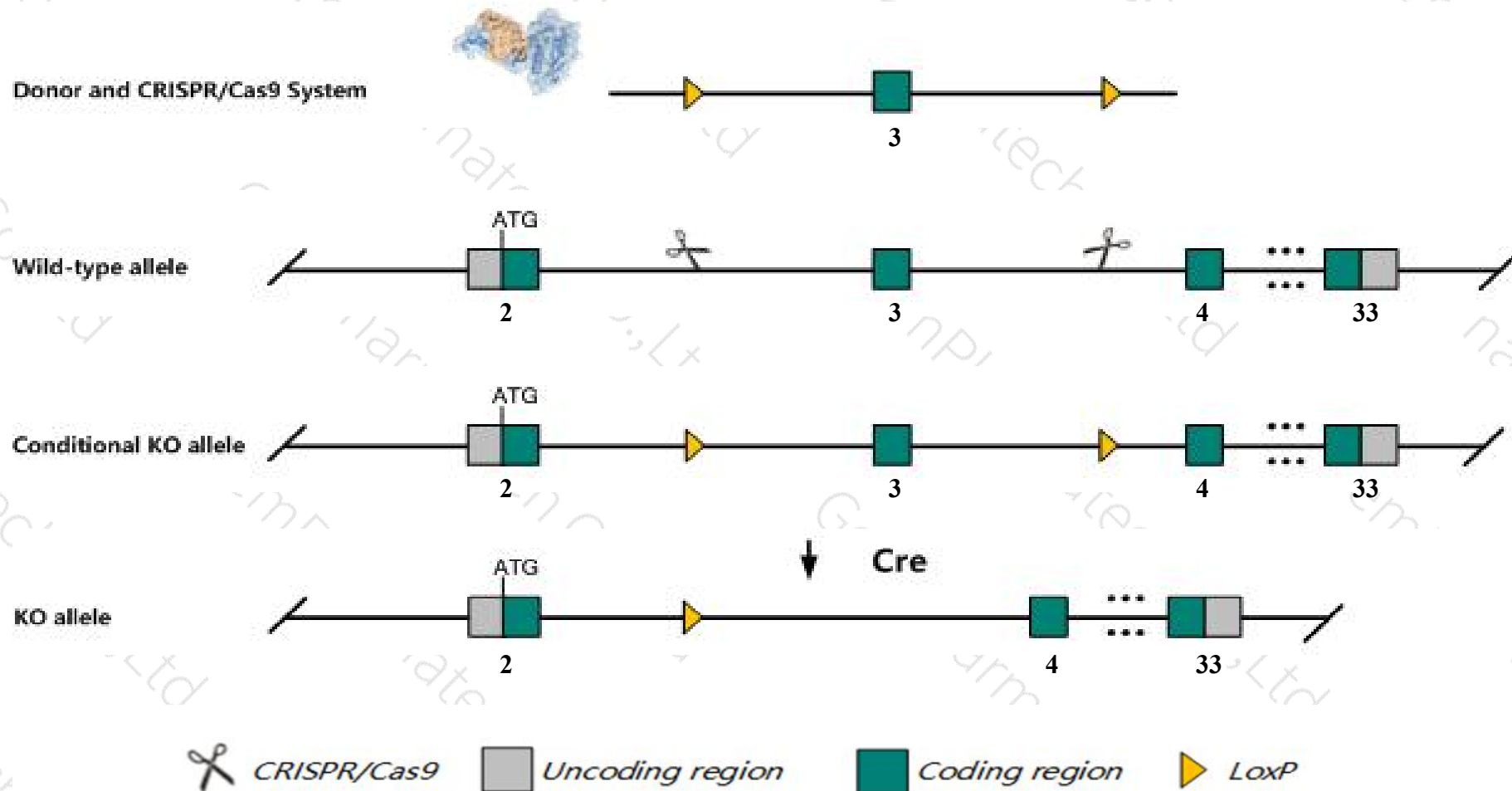
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Pik3c2a* gene. The schematic diagram is as follows:



Technical routes

- The *Pik3c2a* gene has 7 transcripts. According to the structure of *Pik3c2a* gene, exon3 of *Pik3c2a-201* (ENSMUST00000170430.2) transcript is recommended as the knockout region. The region contains 104bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Pik3c2a* gene. The brief process is as follows: CRISPR/Cas9 system and Donor were microinjected into the fertilized eggs of C57BL/6JGpt mice. Fertilized eggs were transplanted to obtain positive F0 mice which were confirmed by PCR and sequencing. A stable F1 generation mouse model was obtained by mating positive F0 generation mice with C57BL/6JGpt mice.
- The flox mice will be knocked out after mating with mice expressing Cre recombinase, resulting in the loss of function of the target gene in specific tissues and cell types.

- According to the existing MGI data, Mice homozygous for a gene trap allele show chronic renal failure and a range of renal lesions that precede immune involvement. Mice heterozygous for a kinase-inactivating allele show defects in platelet formation, platelet membrane morphology and dynamics, and an enrichment of barbell proplatelets.
- The *Pik3c2a* gene is located on the Chr7. If the knockout mice are crossed with other mice strains to obtain double gene positive homozygous mouse offspring, please avoid the two genes on the same chromosome.
- This Strategy is designed based on genetic information in existing databases. Due to the complexity of biological processes, all risk of loxp insertion on gene transcription, RNA splicing and protein translation cannot be predicted at existing technological level.

Gene information (NCBI)

Pik3c2a phosphatidylinositol-4-phosphate 3-kinase catalytic subunit type 2 alpha [Mus musculus (house mouse)]

Gene ID: 18704, updated on 19-Feb-2019

Summary



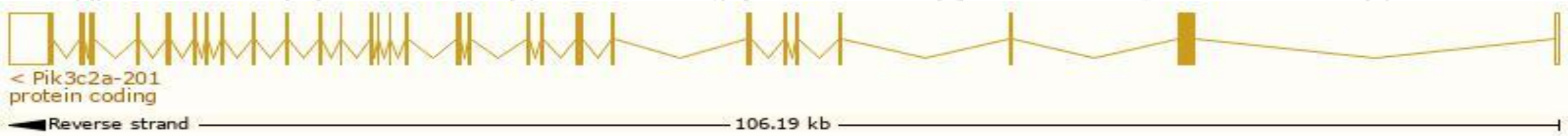
Official Symbol	Pik3c2a provided by MGI
Official Full Name	phosphatidylinositol-4-phosphate 3-kinase catalytic subunit type 2 alpha provided by MGI
Primary source	MGI:MGI:1203729
See related	Ensembl:ENSMUSG00000030660
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	Cpk-m, PI3KC2
Expression	Ubiquitous expression in placenta adult (RPKM 5.5), limb E14.5 (RPKM 4.0) and 25 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

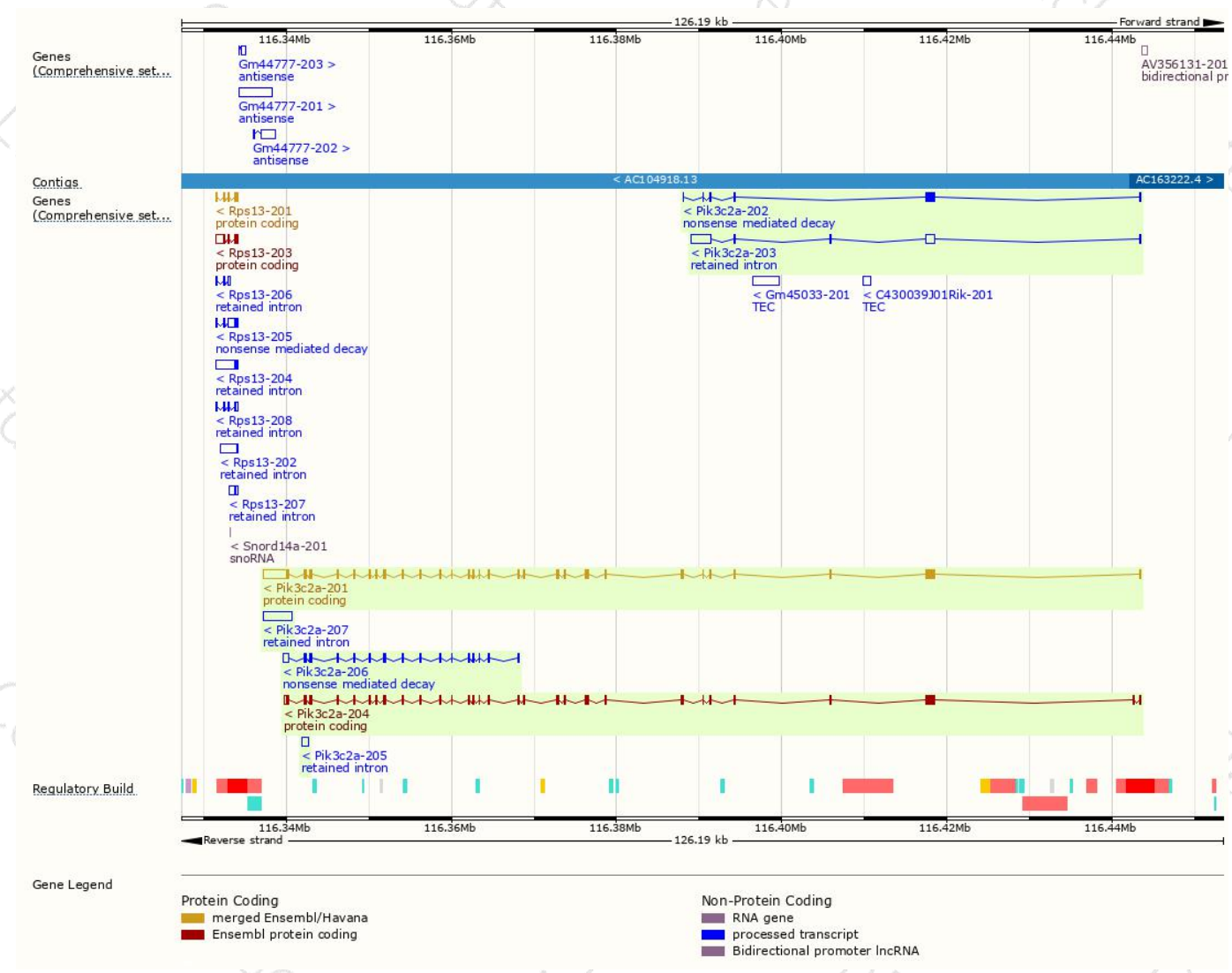
The gene has 7 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Pik3c2a-201	ENSMUST00000170430.2	8044	1686aa	Protein coding	CCDS52371	F8VPL2	TSL:5 GENCODE basic APPRIS P1
Pik3c2a-204	ENSMUST00000206219.1	5670	1686aa	Protein coding	CCDS52371	F8VPL2	TSL:5 GENCODE basic APPRIS P1
Pik3c2a-206	ENSMUST00000206385.1	2830	448aa	Nonsense mediated decay	-	A0A0U1RNT0	CDS 5' incomplete TSL:1
Pik3c2a-202	ENSMUST00000205378.1	1710	364aa	Nonsense mediated decay	-	A0A0U1RNH9	TSL:1
Pik3c2a-203	ENSMUST00000205767.1	3849	No protein	Retained intron	-	-	TSL:2
Pik3c2a-207	ENSMUST00000206805.1	3354	No protein	Retained intron	-	-	TSL:NA
Pik3c2a-205	ENSMUST00000206248.1	741	No protein	Retained intron	-	-	TSL:NA

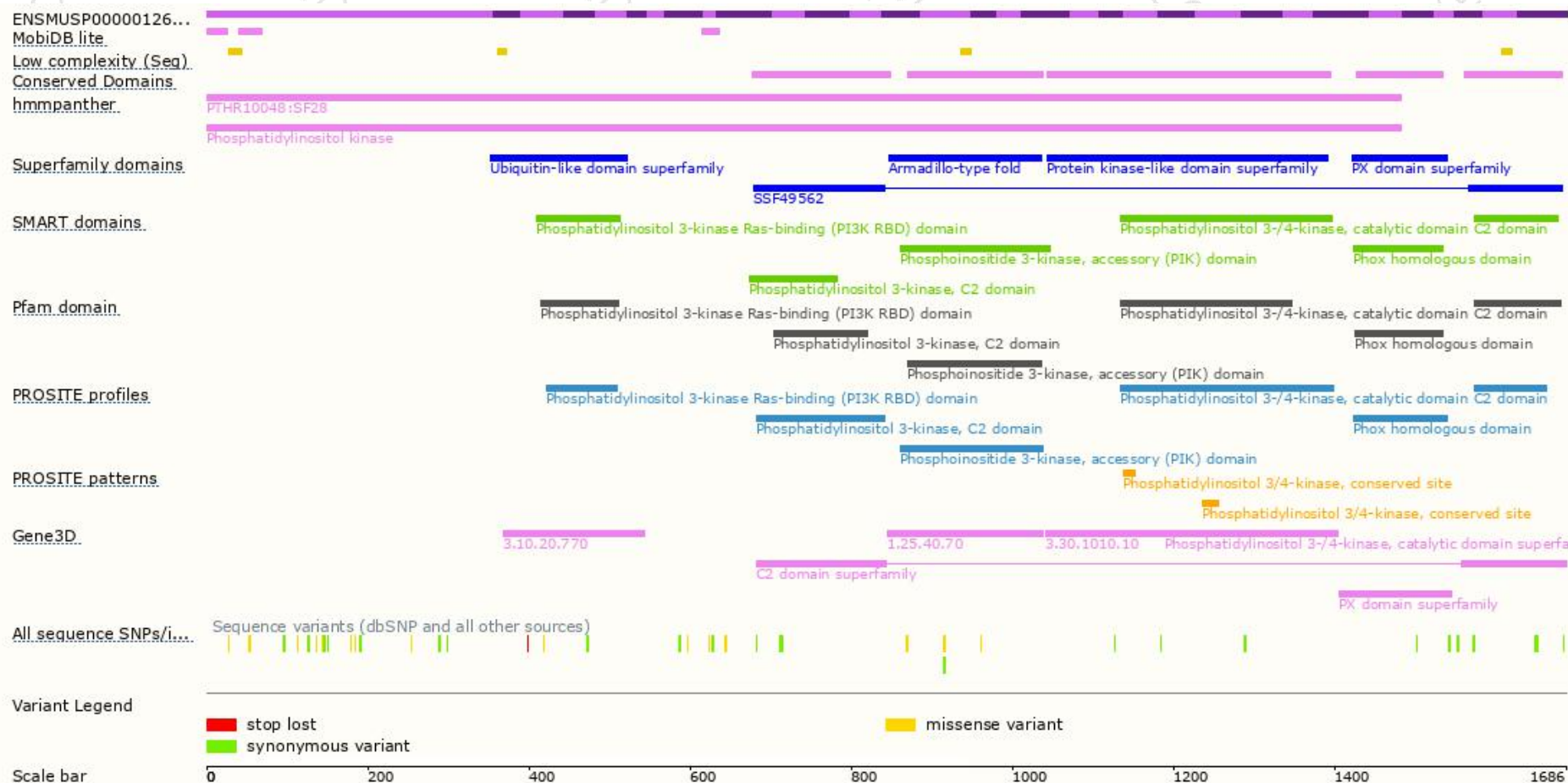
The strategy is based on the design of *Pik3c2a-201* transcript,The transcription is shown below



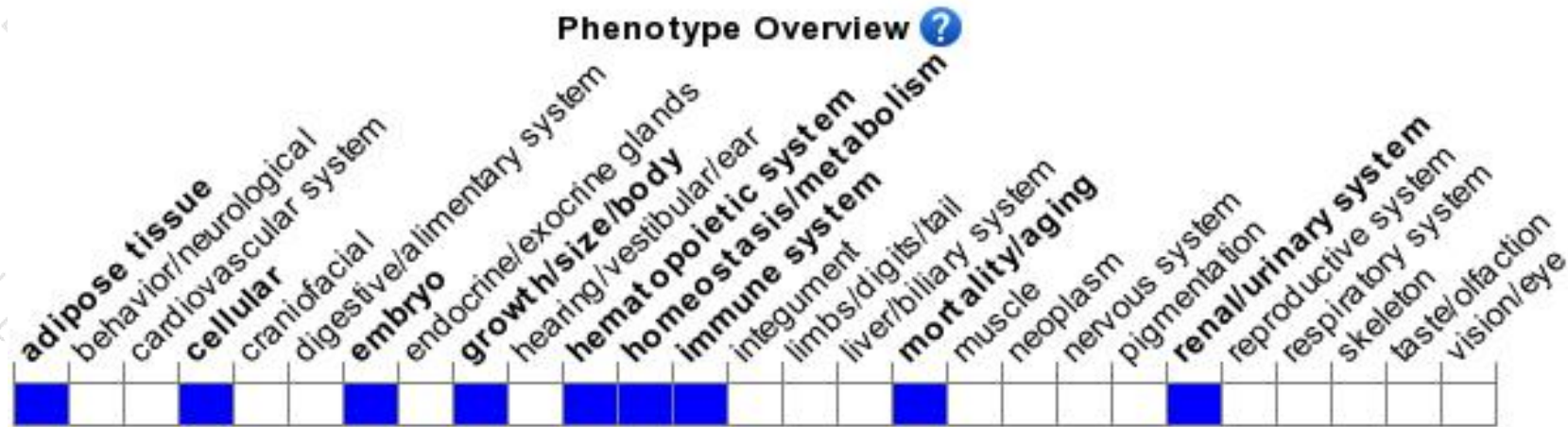
Genomic location distribution



Protein domain



Mouse phenotype description(MGI)



Phenotypes affected by the gene are marked in blue. Data quoted from MGI database(<http://www.informatics.jax.org/>).

According to the existing MGI data, Mice homozygous for a gene trap allele show chronic renal failure and a range of renal lesions that precede immune involvement. Mice heterozygous for a kinase-inactivating allele show defects in platelet formation, platelet membrane morphology and dynamics, and an enrichment of barbell proplatelets.

If you have any questions, you are welcome to inquire.

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