吉林大学植物科学学院本科生素质类项目推免加分申请表（试行）

姓名： 李祉祺 **学号：** 82210101 **年级专业：** 2021级农学

申请素质加分绩点： 0.02

素质类项目加分统计表

类别	序号	加分政策		GPA 加分		本人成果加分分值	具体成果名称/获奖等级
科研成果	1	大学生创新创业训练计划项目	国家级已结题优秀项目	负责人 0.05	学院认定加分（最高0.05），中间换人不加分。未结题、一般结题项目不加分		
				第二名 0.03			
	2	学术论文（本专业领域）	中国科学院文献情报中心期刊分区论文发表当年适用的吉林大学哲学社会科学学术刊物目录、北大核心期刊目录	一区/A类 一作0.2	学院认定加分（最高0.3），中间换人不加分。核心以上期刊加分，一般期刊不加分。二作及其他作者不加分。		
				二区/B类一作0.15			
				三区/C类一作0.1			
				四区/D类及非ABCD类的北大核心期刊一作0.05			
竞赛获奖	1	吉林大学本科学生学科竞赛体系	A类获得国家级奖项特等奖	0.15	竞赛项目参照学校认定的竞赛体系，主力队员（限前3名），学院认定加分（最高0.15），中间换人不加分		
			A类获得国家级奖项一等奖（金奖）	0.1			
			A类获得国家级奖项二等奖（银奖）	0.06			
			A类获得国家级奖项三等奖（铜奖）	0.04			
			B类获得国家级奖项特等奖	0.08			
			B类获得国家级奖项一等奖（金奖）	0.06			
			B类获得国家级奖项二等奖（银奖）	0.04			
			B类获得国家级奖项三等奖（铜奖）	0.02		0.02	全国大学生生命科学竞赛国家级三等奖
			C类获得国家级奖项特等奖	0.06			
			C类获得国家级奖项一等奖（金奖）	0.04			
			C类获得国家级奖项二等奖（银奖）	0.02			
			C类获得国家级奖项三等奖（铜奖）	0.01			
其他类别成果	1	服兵役：入伍服兵役退役复学，获得优秀义务兵、三等功以上（党委学生工作部、党委武装部认定加分）			主管部门已认定的成果（需要提供学校相关部门提供的素质加分证明材料）		
	2	志愿服务：中国青年志愿服务大赛（国家级）金奖、银奖前三名、（省级）金奖前两名。（团委认定加分） 社会工作（思想政治志愿服务）思想政治志愿服务骨干成员；（少数民族志愿服务）少数民族志愿服务骨干成员（党委学生工作部认定加分）					
	3	国际组织实习：国际组织实习（实习的国际组织认定以中组部、教育部等部委相关文件为准）三个月以上（实习结束应有该组织证明、实习录取和实习结束证明等，须在学校留档）。（国际合作与交流处、学生就业创业指导与服务中心认定加分）					
合计						0.02	

说明：1、素质各项加分总绩点最多不能超过0.4。 2、体育竞赛、艺术竞赛、入伍服役、志愿服务、国际组织实习等其它类别的素质加分项目，由体育学院、党委学生工作部、团委、党委武装部、国际合作与交流处、学生就业创业指导与服务中心等相关部门认定，学院参照执行。 3、证明材料竞赛证书、发表文章等附后，同时上交电子版扫描件。

专业审核小组是否通过：是（ √ ）否（ ）

专业审核小组成员签字：

姜文梁 李刻 缪伟伦 [illegible]

第九届全国大学生生命科学竞赛（创新创业类）奖项公示

各参赛单位：

依据全国大学生生命科学竞赛（创新创业类）各项规则及要求，现已完成所有比赛环节，6676 支报名团队，共计评出一等奖 280 项，二等奖 811 项，三等奖 1255 项，优秀组织奖 61 项。评审结果于 2024 年 7 月 30 日经组委会和监委会审定，参照《全国大学生生命科学竞赛章程》，现将获奖名单予以公示。

公示时间：2024 年 8 月 1 日至 2024 年 8 月 7 日，公示期 7 天。

如对公示项目有异议，请于公示期内提交以学校为单位加盖公章的书面意见。联系邮箱：limeng@mail.xhu.edu.cn。

附件：

1. 第九届全国大学生生命科学竞赛（创新创业类）一等奖获奖名单
2. 第九届全国大学生生命科学竞赛（创新创业类）二等奖获奖名单
3. 第九届全国大学生生命科学竞赛（创新创业类）三等奖获奖名单
4. 第九届全国大学生生命科学竞赛（创新创业类）优秀组织奖获奖名单

全国大学生生命科学竞赛委员会

2024 年 7 月 31 日

468	吉林省	吉林大学	X20240153	利用CYR2实现光遗传学诱导Caspase-8介导的细胞凋亡	创新组	商芮阁	张贺	吴柳明	刘园园	王江柠		莫伟亮	左泽乘	三等奖
469	吉林省	吉林大学	X20241926	ZEA通过ROS-AMPK-m-TOR途径介导自噬恶化金黄色葡萄球菌诱诱导的小鼠乳腺炎	创新组	杨恒艺	赵科越	王柔杰	王珂怡	李博一		胡晓宇	付云贺	三等奖
470	吉林省	吉林大学	X20242422	十六碳酰胺对金葡菌性乳腺炎的保护作用及机制探究	创新组	孙浩	赵晓冉	陈秋洁				付云贺	胡晓宇	三等奖
471	吉林省	吉林大学	X20243448	染料木苷抗单增李斯特菌感染作用的机制研究	创新组	许竞文	张济川	林锐				王琳	王建锋	三等奖
472	吉林省	吉林大学	X20243867	银杏内酯对 DSS 诱导的小鼠结肠炎的影响及其机制	创新组	高姗	崔璐瑶	杜玉婷				付守鹏		三等奖
473	吉林省	吉林大学	X20243869	植酸对高脂饮食诱导的小鼠非酒精性脂肪肝的影响及机制	创新组	贺富鼎	李心怡	周文祎				胡桂秋		三等奖
474	吉林省	吉林大学	X20243965	缬氨酸对奶牛乳合成的影响及其机制	创新组	龙潇予	张佳琳					曹宇		三等奖
475	吉林省	吉林大学	X20244006	APPA对秀丽隐杆线虫的抗衰老作用及其机制的研究	创新组	曹娇飞	李沛真	吕文曦	安然	赵一品		王丽萍		三等奖
476	吉林省	吉林大学	X20244695	根皮苷对DSS诱导的小鼠结肠炎的影响及其机制	创新组	黄雅萍	丁惠萍					杨占清		三等奖
477	吉林省	吉林大学	X20245076	DNA调控纳米花结构仿酶的合成方法及有机磷农药检测应用	创新组	张宏润	林琪淇	李响				李红霞	闫旭	三等奖
478	吉林省	吉林大学	X20245336	溶木聚糖拟杆菌对高蛋白日粮雏鹅的抗高尿酸血症作用	创新组	仲广旭	王哲					张文龙		三等奖
479	吉林省	吉林大学	X20245645	食源性动植物蛋白组成、功能及加工特性研究	创新组	付雯菲	刘釜均	常楚豫	夏景昱	唐芊芊	刘益林	王翠娜		三等奖
480	吉林省	吉林大学	X20245997	淀粉纳米颗粒的制备及其在皮克林乳液中的应用研究	创新组	白锦林	胡函宾	陶子涵				闫晓侠		三等奖
481	吉林省	吉林大学	X20246324	卵清蛋白和溶菌酶异质蛋白复合凝聚机制与结构的研究	创新组	李晨曼	任健麒	王治	戴璐遥	吕丹	郑知媛	刘轩廷	刘静波	三等奖
482	吉林省	吉林大学	X20246376	“脂”消“康”长——蛋清蛋白双网络凝胶体系对肉糜凝胶性能改善的研究	创新组	曹斯佳	潘科研	褚泽敬	黄琪涵			刘轩廷	刘静波	三等奖
483	吉林省	吉林大学	X20246463	用于3D打印的藜麦蛋白/多糖静电络合稳定纯素高内相乳液:络合状态和胶凝型多糖的作用	创新组	王琦	郝怡琳					沈雪		三等奖
484	吉林省	吉林大学	Y20240620	医食无忧——“东方膳食AI平台”开创国民食养新形态	创业组(创意类)	张辰铭	刘振阳	华子涵	许超	于淼	苑一涵	王雪	白洪涛	三等奖
485	吉林省	吉林大学	Y20243450	骨松早诊——国内骨质AI诊疗系统先行者	创业组(创意类)	刘明磊	王可欣	王佳鑫	修乾伟	林正	朱圣基	秦彦国	逯家辉	三等奖
486	吉林省	吉林大学	Y20243669	未癌先知—结直肠癌早筛行业领航者	创业组(创意类)	王佳瑞	韩显铭	李珂云	王凤州	宁卓阳	李梓琦	王迪	逯家辉	三等奖
487	吉林省	吉林大学	Y20243712	绿酶—新型果蔬清洁剂创新者	创业组(创意类)	张蓬尹	玄澄超	唐艺嘉	李垚荣	杨嘉曦	张皓	韩葳葳	王嵩	三等奖
488	吉林省	吉林大学	Y20244242	炎洁康——国内首创抗HPV功能性卫生巾	创业组(创意类)	李静蕾	尹伊诺	王伟怡	黄宇			逯家辉	王艳	三等奖
489	吉林省	吉林大学	Y20244357	薯测病除——马铃薯早疫病早期检测模式开创者	创业组(创意类)	浮思怡	戴天雪	包欣鹭	李奕晗	艾叶	郭奕男	王迪	逯家辉	三等奖
490	吉林省	吉林大学	Y20244443	虫医生—环境友好型绿色害虫诱控剂	创业组(创意类)	潘显怡	张梓昂	李祉祺	包窈	高梓荀	徐洋	张大伟	王楚楚	三等奖
491	吉林省	吉林大学	Y20244840	菌肥宝	创业组(创意类)	韦冰豪	王静怡					任晓冬	姜丽艳	三等奖
492	吉林省	吉林大学	Y20244922	食菌芯知——开创食源性致病菌检测“芯”纪元	创业组(创意类)	法思睿	赵晗池	井怡文	李鹤	初加仪	李瑞	赵超	逯家辉	三等奖
493	吉林省	吉林大学	Y20245194	“绣”外慧中 ——松子壳再生利用与绣球菌栽培破局者	创业组(创意类)	邢心茹	陈湛昊	于露	赵虞茜	闫一晴		张大伟	许海	三等奖
494	吉林省	吉林大学	Y20245243	环境病原体在线快速检测设备	创业组(创意类)	任学志	张泽鑫	代云松	张爽	延依静		张大奕		三等奖

吉林大学植物科学学院本科生素质类项目推免加分申请表（试行）

姓名：徐洋　学号：82210107　年级专业：2021 级植物保护

申请素质加分绩点：0.02

素质类项目加分统计表

类别	序号	加分政策		GPA 加分		本人成果加分分值	具体成果名称/获奖等级
科研成果	1	大学生创新创业训练计划项目	国家级已结题优秀项目	负责人 0.05	学院认定加分（最高 0.05），中间换人不加分。未结题、一般结题项目不加分		
				第二名 0.03			
	2	学术论文（本专业领域）	中国科学院文献情报中心期刊分区论文发表当年适用的吉林大学哲学社会科学学术刊物目录、北大核心期刊目录	一区/A 类 一作 0.2	学院认定加分（最高 0.3），中间换人不加分。核心以上期刊加分，一般期刊不加分。二作及其他作者不加分。		
				二区/B 类一作 0.15			
				三区/C 类一作 0.1			
				四区/D 类及非 ABCD 类的北大核心期刊一作 0.05			
竞赛获奖	1	吉林大学本科学生学科竞赛体系	A 类获得国家级奖项特等奖	0.15	竞赛项目参照学校认定的竞赛体系，主力队员（限前 3 名），学院认定加分（最高 0.15），中间换人不加分		
			A 类获得国家级奖项一等奖（金奖）	0.1			
			A 类获得国家级奖项二等奖（银奖）	0.06			
			A 类获得国家级奖项三等奖（铜奖）	0.04			
			B 类获得国家级奖项特等奖	0.08			
			B 类获得国家级奖项一等奖（金奖）	0.06			
			B 类获得国家级奖项二等奖（银奖）	0.04			
			B 类获得国家级奖项三等奖（铜奖）	0.02		0.02	金兰契友—兰花与兰友的诚挚伙伴/全国大学生生命科学竞赛国家级三等奖
			C 类获得国家级奖项特等奖	0.06			
			C 类获得国家级奖项一等奖（金奖）	0.04			
			C 类获得国家级奖项二等奖（银奖）	0.02			
			C 类获得国家级奖项三等奖（铜奖）	0.01			
其他类别成果	1	服兵役：入伍服兵役退役复学，获得优秀义务兵、三等功以上（党委学生工作部、党委武装部认定加分）			主管部门已认定的成果（需要提供学校相关部门提供的素质加分证明材料）		
	2	志愿服务：中国青年志愿服务大赛（国家级）金奖、银奖前三名、（省级）金奖前两名。（团委认定加分） 社会工作（思想政治志愿服务）思想政治志愿服务骨干成员；（少数民族志愿服务）少数民族志愿服务骨干成员（党委学生工作部认定加分）					
	3	国际组织实习：国际组织实习（实习的国际组织认定以中组部、教育部等部委相关文件为准）三个月以上（实习结束应有该组织证明、实习录取和实习结束证明等，须在学校留档）。（国际合作与交流处、学生就业创业指导与服务中心认定加分）					
合　计						0.02	

说明：1、素质各项加分总绩点最多不能超过 0.4。　2、体育竞赛、艺术竞赛、入伍服役、志愿服务、国际组织实习等其它类别的素质加分项目，由体育学院、党委学生工作部、团委、党委武装部、国际合作与交流处、学生就业创业指导与服务中心等相关部门认定，学院参照执行。 3、证明材料竞赛证书、发表文章等附后，同时上交电子版扫描件。

专业审核小组是否通过：是（ ✓ ）否（　　）

专业审核小组成员签字：

时间： 2024 年 8 月 26 日

附件：佐证材料

CULSC 第八届全国大学生生命科学竞赛（创新创业类）

获奖证书

获奖项目：金兰契友——兰花与兰友的诚挚伙伴

获奖学生：潘显怡、包窈、徐洋

指导老师：卢可、王楚楚

获奖单位：吉林大学

获奖类型：指导教师三等奖（创业组）

证 书 号：CULSC2023CXCY1432

全国大学生生命科学竞赛委员会

二〇二三年八月

吉林大学植物科学学院本科生素质类项目推免加分申请表（试行）

姓名：包窈　　学号：82210319　　年级专业：2021级植物保护

申请素质加分绩点：0.02

素质类项目加分统计表

类别	序号	加分政策		GPA 加分		本人成果加分分值	具体成果名称/获奖等级
科研成果	1	大学生创新创业训练计划项目	国家级已结题优秀项目	负责人 0.05	学院认定加分（最高0.05），中间换人不加分。未结题、一般结题项目不加分		
				第二名 0.03			
	2	学术论文（本专业领域）	中国科学院文献情报中心期刊分区论文发表当年适用的吉林大学哲学社会科学学术刊物目录、北大核心期刊目录	一区/A类 一作0.2	学院认定加分（最高0.3），中间换人不加分。核心以上期刊加分，一般期刊不加分。二作及其他作者不加分。		
				二区/B类一作0.15			
				三区/C类一作0.1			
				四区/D类及非ABCD类的北大核心期刊一作0.05			
竞赛获奖	1	吉林大学本科学生学科竞赛体系	A类获得国家级奖项特等奖	0.15	竞赛项目参照学校认定的竞赛体系，主力队员（限前3名），学院认定加分（最高0.15），中间换人不加分		
			A类获得国家级奖项一等奖（金奖）	0.1			
			A类获得国家级奖项二等奖（银奖）	0.06			
			A类获得国家级奖项三等奖（铜奖）	0.04			
			B类获得国家级奖项特等奖	0.08			
			B类获得国家级奖项一等奖（金奖）	0.06			
			B类获得国家级奖项二等奖（银奖）	0.04			
			B类获得国家级奖项三等奖（铜奖）	0.02		0.02	金兰契友—兰花与兰友的诚挚伙伴/全国大学生生命科学竞赛国家级三等奖
			C类获得国家级奖项特等奖	0.06			
			C类获得国家级奖项一等奖（金奖）	0.04			
			C类获得国家级奖项二等奖（银奖）	0.02			
			C类获得国家级奖项三等奖（铜奖）	0.01			
其他类别成果	1	服兵役：入伍服兵役退役复学，获得优秀义务兵、三等功以上（党委学生工作部、党委武装部认定加分）			主管部门已认定的成果（需要提供学校相关部门提供的素质加分证明材料）		
	2	志愿服务：中国青年志愿服务大赛（国家级）金奖、银奖前三名、（省级）金奖前两名。（团委认定加分） 社会工作（思想政治志愿服务）思想政治志愿服务骨干成员；（少数民族志愿服务）少数民族志愿服务骨干成员（党委学生工作部认定加分）					
	3	国际组织实习：国际组织实习（实习的国际组织认定以中组部、教育部等部委相关文件为准）三个月以上（实习结束应有该组织证明、实习录取和实习结束证明等，须在学校留档）。（国际合作与交流处、学生就业创业指导与服务中心认定加分）					
合　　计						0.02	

说明：1、素质各项加分总绩点最多不能超过0.4。　2、体育竞赛、艺术竞赛、入伍服役、志愿服务、国际组织实习等其它类别的素质加分项目，由体育学院、党委学生工作部、团委、党委武装部、国际合作与交流处、学生就业创业指导与服务中心等相关部门认定，学院参照执行。　3、证明材料竞赛证书、发表文章等附后，同时上交电子版扫描件。

专业审核小组是否通过：是（ √ ）否（　　）

专业审核小组成员签字：

时间： 2024 年 8 月 26 日

附件：佐证材料

CULSC 第八届全国大学生生命科学竞赛（创新创业类）

获奖证书

获奖项目： 金兰契友——兰花与兰友的诚挚伙伴

获奖学生： 潘显怡、包窈、徐洋

指导老师： 卢可、王楚楚

获奖单位： 吉林大学

获奖类型： 指导教师三等奖（创业组）

证 书 号： CULSC2023CXCY1432

全国大学生生命科学竞赛委员会

二〇二三年八月

CULSC

吉林大学植物科学学院本科生素质类项目推免加分申请表（试行）

姓名：张梓昂　学号：82210136　年级专业：2021级植物保护

申请素质加分绩点：0.02

素质类项目加分统计表

类别	序号	加分政策		GPA 加分		本人成果加分分值	具体成果名称/获奖等级
科研成果	1	大学生创新创业训练计划项目	国家级已结题优秀项目	负责人 0.05	学院认定加分（最高0.05），中间换人不加分。未结题、一般结题项目不加分		
				第二名 0.03			
	2	学术论文（本专业领域）	中国科学院文献情报中心期刊分区论文发表当年适用的吉林大学哲学社会科学学术刊物目录、北大核心期刊目录	一区/A类 一作0.2	学院认定加分（最高0.3），中间换人不加分。核心以上期刊加分，一般期刊不加分。二作及其他作者不加分。		
				二区/B类一作0.15			
				三区/C类一作0.1			
				四区/D类及非ABCD类的北大核心期刊一作0.05			
竞赛获奖	1	吉林大学本科学生学科竞赛体系	A类获得国家级奖项特等奖	0.15	竞赛项目参照学校认定的竞赛体系，主力队员（限前3名），学院认定加分（最高0.15），中间换人不加分		
			A类获得国家级奖项一等奖（金奖）	0.1			
			A类获得国家级奖项二等奖（银奖）	0.06			
			A类获得国家级奖项三等奖（铜奖）	0.04			
			B类获得国家级奖项特等奖	0.08			
			B类获得国家级奖项一等奖（金奖）	0.06			
			B类获得国家级奖项二等奖（银奖）	0.04			
			B类获得国家级奖项三等奖（铜奖）	0.02		0.02	全国大学生生命科学竞赛国家级三等奖
			C类获得国家级奖项特等奖	0.06			
			C类获得国家级奖项一等奖（金奖）	0.04			
			C类获得国家级奖项二等奖（银奖）	0.02			
			C类获得国家级奖项三等奖（铜奖）	0.01			
其他类别成果	1	服兵役：入伍服兵役退役复学，获得优秀义务兵、三等功以上（党委学生工作部、党委武装部认定加分）			主管部门已认定的成果（需要提供学校相关部门提供的素质加分证明材料）		
	2	志愿服务：中国青年志愿服务大赛（国家级）金奖、银奖前三名、（省级）金奖前两名。（团委认定加分） 社会工作（思想政治志愿服务）思想政治志愿服务骨干成员；（少数民族志愿服务）少数民族志愿服务骨干成员（党委学生工作部认定加分）					
	3	国际组织实习：国际组织实习（实习的国际组织认定以中组部、教育部等部委相关文件为准）三个月以上（实习结束应有该组织证明、实习录取和实习结束证明等，须在学校留档）。（国际合作与交流处、学生就业创业指导与服务中心认定加分）					
合　计						0.02	

说明：1、素质各项加分总绩点最多不能超过 0.4。　2、体育竞赛、艺术竞赛、入伍服役、志愿服务、国际组织实习等其它类别的素质加分项目，由体育学院、党委学生工作部、团委、党委武装部、国际合作与交流处、学生就业创业指导与服务中心等相关部门认定，学院参照执行。 3、证明材料竞赛证书、发表文章等附后，同时上交电子版扫描件。

专业审核小组是否通过：是（√）否（　）

专业审核小组成员签字：

时间： 2024 年 8 月 26 日

附件：佐证材料

注：由于第九届全国大学生生命科学竞赛（创新创业类）国家级获奖证书尚未发放，因此佐证材料为竞赛官网公示文件。

第九届全国大学生生命科学竞赛（创新创业类）奖项公示

各参赛单位：

依据全国大学生生命科学竞赛（创新创业类）各项规则及要求，现已完成所有比赛环节，6676 支报名团队，共计评出一等奖 280 项，二等奖 811 项，三等奖 1255 项，优秀组织奖 61 项。评审结果于 2024 年 7 月 30 日经组委会和监委会审定，参照《全国大学生生命科学竞赛章程》，现将获奖名单予以公示。

公示时间：2024 年 8 月 1 日至 2024 年 8 月 7 日，公示期 7 天。

如对公示项目有异议，请于公示期内提交以学校为单位加盖公章的书面意见。联系邮箱：limeng@mail.xhu.edu.cn。

附件：

1. 第九届全国大学生生命科学竞赛（创新创业类）一等奖获奖名单
2. 第九届全国大学生生命科学竞赛（创新创业类）二等奖获奖名单
3. 第九届全国大学生生命科学竞赛（创新创业类）三等奖获奖名单
4. 第九届全国大学生生命科学竞赛（创新创业类）优秀组织奖获奖名单

全国大学生生命科学竞赛委员会

2024 年 7 月 31 日

468	吉林省	吉林大学	X20240153	利用CYR2实现光遗传学诱导Caspase-8介导的细胞凋亡	创新组	商芮阁	张贺	吴柳明	刘园园	王江柠		莫伟亮	左泽乘	三等奖
469	吉林省	吉林大学	X20241926	ZEA通过ROS-AMPK-m-TOR途径介导自噬恶化金黄色葡萄球菌诱诱导的小鼠乳腺炎	创新组	杨恒艺	赵科越	王柔杰	王珂怡	李博一		胡晓宇	付云贺	三等奖
470	吉林省	吉林大学	X20242422	十六碳酰胺对金葡菌性乳腺炎的保护作用及机制探究	创新组	孙浩	赵晓冉	陈秋洁				付云贺	胡晓宇	三等奖
471	吉林省	吉林大学	X20243448	染料木苷抗单增李斯特菌感染作用的机制研究	创新组	许竞文	张济川	林锐				王琳	王建锋	三等奖
472	吉林省	吉林大学	X20243867	银杏内酯对 DSS 诱导的小鼠结肠炎的影响及其机制	创新组	高姗	崔璐瑶	杜玉婷				付守鹏		三等奖
473	吉林省	吉林大学	X20243869	植酸对高脂饮食诱导的小鼠非酒精性脂肪肝的影响及机制	创新组	贺富鼎	李心怡	周文祎				胡桂秋		三等奖
474	吉林省	吉林大学	X20243965	缬氨酸对奶牛乳合成的影响及其机制	创新组	龙潇予	张佳琳					曹宇		三等奖
475	吉林省	吉林大学	X20244006	APPA对秀丽隐杆线虫的抗衰老作用及其机制的研究	创新组	曹娇飞	李沛真	吕文曦	安然	赵一品		王丽萍		二等奖
476	吉林省	吉林大学	X20244695	根皮苷对DSS诱导的小鼠结肠炎的影响及其机制	创新组	黄雅萍	丁惠萍					杨占清		三等奖
477	吉林省	吉林大学	X20245076	DNA调控纳米花结构仿酶的合成方法及有机磷农药检测应用	创新组	张宏润	林琪淇	李响				李红霞	闫旭	三等奖
478	吉林省	吉林大学	X20245336	溶木聚糖拟杆菌对高蛋白日粮雏鹅的抗高尿酸血症作用	创新组	仲广旭	王哲					张文龙		三等奖
479	吉林省	吉林大学	X20245645	食源性动植物蛋白组成、功能及加工特性研究	创新组	付雯菲	刘釜均	常楚豫	夏景昱	唐芊芊	刘益林	王翠娜		三等奖
480	吉林省	吉林大学	X20245997	淀粉纳米颗粒的制备及其在皮克林乳液中的应用研究	创新组	白锦林	胡函宾	陶子涵				闫晓侠		三等奖
481	吉林省	吉林大学	X20246324	卵清蛋白和溶菌酶异质蛋白复合凝聚机制与结构的研究	创新组	李晨曼	任健麒	王治	戴璐遥	吕丹	郑知媛	刘轩廷	刘静波	三等奖
482	吉林省	吉林大学	X20246376	“脂”消“康”长——蛋清蛋白双网络凝胶体系对肉糜凝胶性能改善的研究	创新组	曹斯佳	潘科研	褚泽敬	黄琪涵			刘轩廷	刘静波	三等奖
483	吉林省	吉林大学	X20246463	用于3D打印的藜麦蛋白/多糖静电络合稳定纯素高内相乳液:络合状态和胶凝型多糖的作用	创新组	王琦	郝怡琳					沈雪		三等奖
484	吉林省	吉林大学	Y20240620	医食无忧——“东方膳食AI平台”开创国民食养新形态	创业组(创意类)	张辰铭	刘振阳	华子涵	许超	于淼	苑一涵	王雪	白洪涛	三等奖
485	吉林省	吉林大学	Y20243450	骨松早诊——国内骨质AI诊疗系统先行者	创业组(创意类)	刘明磊	王可欣	王佳鑫	修乾伟	林正	朱圣基	秦彦国	逯家辉	三等奖
486	吉林省	吉林大学	Y20243669	未癌先知—结直肠癌早筛行业领航者	创业组(创意类)	王佳瑞	韩显铭	李珂云	王凤州	宁卓阳	李梓琦	王迪	逯家辉	三等奖
487	吉林省	吉林大学	Y20243712	绿酶—新型果蔬清洁剂创新者	创业组(创意类)	张篷尹	玄澄超	唐艺嘉	李垚荣	杨嘉曦	张皓	韩葳葳	王嵩	三等奖
488	吉林省	吉林大学	Y20244242	炎洁康——国内首创抗HPV功能性卫生巾	创业组(创意类)	李静蕾	尹伊诺	王伟怡	黄宇			逯家辉	王艳	三等奖
489	吉林省	吉林大学	Y20244357	薯测病除——马铃薯早疫病早期检测模式开创者	创业组(创意类)	浮思怡	戴天雪	包欣鹭	李奕晗	艾叶	郭奕男	王迪	逯家辉	三等奖
490	吉林省	吉林大学	Y20244443	虫医生--环境友好型绿色害虫诱控剂	创业组(创意类)	潘显怡	张梓昂	李祉祺	包窈	高梓荀	徐洋	张大伟	王楚楚	三等奖
491	吉林省	吉林大学	Y20244840	菌肥宝	创业组(创意类)	韦冰豪	王静怡					任晓冬	姜丽艳	三等奖
492	吉林省	吉林大学	Y20244922	食菌芯知——开创食源性致病菌检测“芯”纪元	创业组(创意类)	法思睿	赵晗池	井怡文	李鹤	初加仪	李瑞	赵超	逯家辉	三等奖
493	吉林省	吉林大学	Y20245194	“绣”外慧中 ——松子壳再生利用与绣球菌栽培破局者	创业组(创意类)	邢心茹	陈湛昊	于露	赵虞茜	闫一晴		张大伟	许海	三等奖
494	吉林省	吉林大学	Y20245243	环境病原体在线快速检测设备	创业组(创意类)	任学志	张泽鑫	代云松	张爽	延依静		张大奕		三等奖

附件4

第九届全国大学生生命科学竞赛（创新创业类）优秀组织奖获奖名单

（按学校名称排序）

安徽农业大学	佛山大学	福建农林大学
广东海洋大学	广西师范大学	贵州大学
杭州师范大学	杭州医学院	河北农业大学
河南大学	河南科技大学	河南师范大学
湖北文理学院	湖南农业大学	湖南人文科技学院
湖南师范大学	湖南中医药大学	华北理工大学
华中科技大学	吉林大学	吉林农业大学
江南大学	江苏大学	江苏海洋大学
江苏科技大学	兰州大学	辽宁科技大学
聊城大学	南昌大学	南京工业大学
南京林业大学	南京师范大学	南京信息工程大学
南京医科大学	齐鲁工业大学（山东省科学院）	青岛科技大学
青岛农业大学	曲阜师范大学	三峡大学
山东第一医科大学	深圳大学	苏州大学
台州学院	天津科技大学	温州医科大学
武汉工程大学	武汉科技大学	武汉理工大学
武汉轻工大学	西北农林科技大学	西华大学
徐州工程学院	徐州医科大学	盐城师范学院
扬州大学	浙江理工大学	浙江农林大学
浙江师范大学	中国计量大学	中南林业科技大学
重庆医科大学		

吉林大学植物科学学院本科生素质类项目推免加分申请表（试行）

姓名：商芮阁　学号：82210427　年级专业：2021级园艺

申请素质加分绩点：0.05

素质类项目加分统计表

类别	序号	加分政策		GPA 加分		本人成果加分分值	具体成果名称/获奖等级
科研成果	1	大学生创新创业训练计划项目	国家级已结题优秀项目	负责人 0.05	学院认定加分（最高0.05），中间换人不加分。未结题、一般结题项目不加分		
				第二名 0.03			
	2	学术论文（本专业领域）	中国科学院文献情报中心期刊分区论文发表当年适用的吉林大学哲学社会科学学术刊物目录、北大核心期刊目录	一区/A类 一作0.2	学院认定加分（最高0.3），中间换人不加分。核心以上期刊加分，一般期刊不加分。二作及其他作者不加分。		
				二区/B类一作0.15		0.05	Scientific reports期刊以第一作者身份发表文章
				三区/C类一作0.1			
				四区/D类及非ABCD类的北大核心期刊一作0.05			
竞赛获奖	1	吉林大学本科学生学科竞赛体系	A类获得国家级奖项特等奖	0.15	竞赛项目参照学校认定的竞赛体系，主力队员（限前3名），学院认定加分（最高0.15），中间换人不加分		
			A类获得国家级奖项一等奖（金奖）	0.1			
			A类获得国家级奖项二等奖（银奖）	0.06			
			A类获得国家级奖项三等奖（铜奖）	0.04			
			B类获得国家级奖项特等奖	0.08			
			B类获得国家级奖项一等奖（金奖）	0.06			
			B类获得国家级奖项二等奖（银奖）	0.04			
			B类获得国家级奖项三等奖（铜奖）	0.02			
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			C类获得国家级奖项三等奖（铜奖）	0.01			
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	2	志愿服务：中国青年志愿服务大赛（国家级）金奖、银奖前三名、（省级）金奖前两名。（团委认定加分） 社会工作（思想政治志愿服务）思想政治志愿服务骨干成员；（少数民族志愿服务）少数民族志愿服务骨干成员（党委学生工作部认定加分）					
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form the Death-Inducing Signaling Complex (DISC), the local zymogen concentration increases, resulting in the activation of caspase 8 precursors. Research has demonstrated that these stable homodimers undergo self-activation through cleavage by their own proteases, while also being susceptible to the action of neighboring dimers. The activation of the Caspase 8 precursor requires two cleavages. The first cleavage occurs at residue D374, yielding the p43/p41 and p12 subunits. Subsequent cleavages at residues D216 and D384 give rise to the p26/p24, p18, and p10 subunits. The p18 subunit and the p10 subunit then form a heterotetramer and disengage from the DISC[10]. This heterotetramer represents active Caspase 8, which is eventually released into the cytoplasm to initiate apoptosis[11,12].

Light-induced cell death induction offers several notable advantages compared to methods that using chemical compounds: faster and easier signal transmission, precise control of the strength and duration of cell death stimuli by altering light dosage and duration, and the ability to restrict the induction of cell death to selected cells or tissues of interest. Various methods using optogenetics exist to activate proteins. Recently, light-sensitive protein domains, such as LOV2 (Light-Oxygen-Voltage 2), have been used as tools to catalyze the separation of caspase-3 and -7 subunits when activated[13,14]. Certainly, there have been studies involving the fusion of caspase 8 with mutant variants of the N-terminal PHR of the blue light receptor CRY2 to induce cell apoptosis. These studies have provided alternative approaches for blue light-regulated apoptosis in HEK293T cells and zebrafish cells[15]. The exploration of the blue light-dependent interaction between CRY2 and CIB1 to regulate caspase8 activity in HeLa cells remains limited. Here, we suggested a method for modulating cell apoptosis through the CRY2-CIB1 interaction, aiming to diversify blue light-induced cell apoptosis approaches and may offer a fresh perspective on employing optogenetic tool in cancer treatment.

Materials and methods

Plasmids constructs

For constructing Opto-Caspase8 plasmids, a pCI (neo) (Promega, E1841) was used. Myc, Flag or GFP was inserted into pCI (neo) with EcoRI restriction site to construct pCI (neo) flag, pCI (neo) myc, and pCI (neo) GFP. The coding sequences of CRY2 PHR or CIB1 N-terminal domain were bridged with Caspase8 by overlapping PCR and then inserted into the pCI (neo) Flag, pCI (neo) Myc or pCI (neo) GFP plasmid using XbaI / XmaI restriction sites to obtain pCI (neo) Myc-PHR-Caspase8, pCI (neo) Flag-PHR-Caspase8 and pCI (neo) GFP-PHR-Caspase8. In order to exclude the effect of endotoxin for apoptosis so all of the plasmids were extracted using Qiagen (12943) plasmid extraction kit. Primers used for vector construction are listed in Supplementary Table 1.

Human cell culture and transfected

HEK-293T cells (ATCC, ATCC®CRL-11268TM) or HeLa cells (ATCC, CRM-CCL-2™) were cultured in DMEM (invitrogen, 10569-044) supplemented with 10% (v/v) FBS (Invitrogen 10100147), 100 U/ml penicillin, and 100 mg/ml streptomycin (Hyclone, SV30010) in humidified 5% (v/v) CO2 in air, at 37 °C. Cells were seeded at a density of 3×10^5 cells per well in a six-well plate and transfected using Lipofectamine 3000 transfection methods as manual instructions described.

Co-immunoprecipitation (Co-IP) assays

Transfected cells were exposed to blue light (30 μmol m^{-2} s^{-1}) or kept in the dark for the indicated time before being lysed. Cell pellets were lysed using Pierce IP Lysis Buffer (87787, Pierce) supplemented with 1×EDTA-free Protease Inhibitor Cocktail Tablets (4693159001, Roche), and then incubated on ice for 15 min. After centrifugation at 14,000×*g* for 10 min at 4 °C, the supernatant was mixed with 20 μl of GFP trap beads and incubated with vertical blending at 4 °C for 2 h. The beads were washed 5 times with washing buffer [20 mM HEPES (pH 7.5), 40 mM KCl, 1 mM EDTA] and denatured by thoroughly mixing with 30 μl of 4× Loading buffer and heating at 100 °C for 10 min. Co-IP samples were detected by Western blot and probed with anti-GFP (MBL, 598) or anti-Flag (MBL, M185-3S), respectively.

Caspase8 cleavage assay

Transfected HeLa or HEK293T cells were either exposed to blue light (30 μmol m^{-2} s^{-1}) or kept in the dark for the indicated time. Then, the cells were dissociated from the dishes with TrypLE™ Express (1x) (Gibco, 12605-028) at 37 °C for 5 min. After centrifugation at 800×*g* for 5 min, the supernatant was discarded. The cell pellets were lysed with Pierce IP Lysis Buffer (87787, Pierce) supplemented with 1×EDTA-free Protease Inhibitor Cocktail Tablets (4693159001, Roche) and incubated on ice for 15 min. The mixtures were centrifuged at 14,000×*g* for 10 min at 4 ℃ to remove cell debris. The supernatants were boiled with 4× Loading buffer for 10 min. Then, the samples were detected by western blot and probed with anti-Caspase8 (Abcam, ab32397), anti-Caspase3 (MBL, M097-3), and anti-Actin (MBL, M177-3). The Western blot band intensities were analyzed with ImageJ software, and all the band intensities were normalized to actin.

Flow cytometry

Transfected HeLa cells were exposed to blue light (50 μmol m^{-2} s^{-1}) for 3 h. The cells were then labeled with Alexa Fluor 488 annexin V and propidium iodide (PI) according to the protocol of the Alexa Fluor 488 Annexin V/ Dead Cell Apoptosis Kit (V13241, Invitrogen) for flow cytometry. The labeled cells were analyzed using the BD Aria II system. The relative cell death rate was calculated as the number of apoptotic cells in blue light divided by the number of apoptotic cells in the dark. For FAM-LETD-FMK caspase-8 assay in Hela cells using flow cytometry, the transfected HeLa cells were exposed to blue light (30 μmol m^{-2} s^{-1}) for 12 h, stained with the

Image-iT LIVE Green Caspase-8 Detection Kit (I35105, Invitrogen) then the labeled cells were analyzed using the BD Aria II system.

Microscope imaging

For the CRY2-PHR-mCherry oligomerization assay in HEK293T cells, the transfected cells were exposed to 50% laser power of the Zeiss confocal LSM880 for the indicated time. Image analysis was performed using Zen software (Zeiss) and processed with Adobe Photoshop. For active Caspase8 detection by microscope, the transfected HEK293T cells were exposed to blue light (30 μmol m^{-2} s^{-1}) for 3 h or kept in the dark and stained with the Image-iT LIVE Green Caspase-8 Detection Kit (I35105, Invitrogen). For the DAPI-labeled detection assay, the transfected HeLa cells were exposed to pulse blue light (50 μmol m^{-2} s^{-1}) for 12 h (10 min on/10 min off) or kept in the dark and stained with DAPI (10 μg/ml). The images were obtained using the Zeiss Observer A1 reverse fluorescence microscope. Image analysis was performed using Zen software and processed with Adobe Photoshop. Fluorescent intensity was analyzed and obtained using ImageJ software.

Quantification and statistical analysis

All data were collected using Excel and analyzed using ANOVA with a two-tailed Student's t-test for statistical significance.

Results

Design and creation of opto-caspase8

In previous studies, it has been found that CRY2 or its PHR domain (CRY2PHR) underwent oligomerization in a blue light dependent manner[16–18]. To confirm CRY2PHR can underwent oligomerization in human cells, we fused PHR to mcherry and transfected into HEK293T cells, and we found that PHR-mcherry can cluster in a blue light dependent manner (Fig. 1A) as expected. To control the caspase8 mediated signaling pathway with

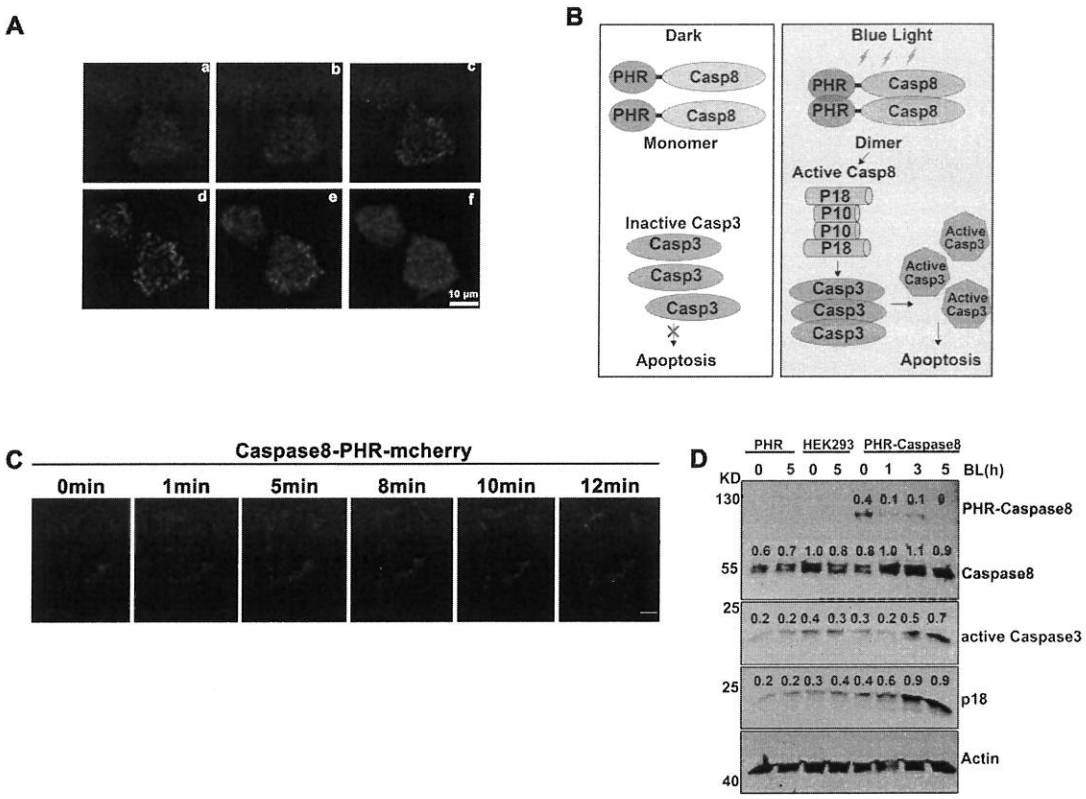

Figure 1. Design of optogenetic tools to control cell apoptosis by blue light. (**A**) Blue light induces CRY2-PHR-mcherry oligomerization in HEK293T cells. a. dark; b. blue light 1 min; c. blue light 5 min; d. blue light 10 min; e. blue to dark 5 min; e. blue to dark 10 min. (**B**) The photo-responsive region of *Arabidopsis thaliana* cryptochrome 2 (PHR, amino acid 1–498) is fused with Caspase8. In the dark, the engineered PHR-Casp8 exhibits monomer. Upon blue light illumination, PHR drives dimerization of PHR-Casp8 and promoters the apoptosis. Casp8, Caspase8; Casp3, Caspase3; PHR, the N-terminal domain of CRY2. (**C**) Clustering of caspase-8-PHR-mCherry in response to blue light in HEK293T cells, bar = 10 μm. (**D**) Blue light actives Opto-Caspase8-V1 in HEK293 T cells. Transfected HEK293T cells were crushed by Pierce IP lysis buffer, total cell lysates were analyzed by western blotting probed with anti-Caspase8, anti-Caspase3, actin was used as a loading control.

blue light, we first fused the PHR domain of CRY2 with caspase8 (PHR-Caspase8) as the new optogenetic tool (Opto-Casp8-V1). As illustrated in Fig. 1B, the PHR-Caspase8 was monomer and kept inactive in darkness. Under blue light irradiation, the PHR-Caspase8 dimerized and oligomerized because of the blue light specific oligomerization of PHR. The oligomerized PHR-Caspase8 then self-cleavaged and released the activated caspase8 domain (P18 and P10) to active downstream caspase3 and promote cell apoptosis. To further confirm the ability of the fusion protein to aggregate, we assessed the aggregation of caspase-PHR-mCherry in HEK293T cells following blue light induction. The results demonstrated that it can indeed aggregate (Fig. 1C). To confirm this process, we transferred the constructed Opto-Casp8-V1 (PHR-Caspase8) including the PHR-only and empty vector control into HEK293T cells, respectively, and treated them with blue light for 0, 1, 3 and 5 h respectively, lysed the cells, and detected the protein level by western. We analyzed the status of PHR-Caspase8, the activated P18 and the downstream protein from dark to blue light irradiation. As excepted, the abundance of the precursor PHR-Caspase8 was decreased after blue light irradiation (Fig. 1D), which suggested the PHR-Caspase8 could self-cleavage and consume in blue light driven by the oligomerization of PHR. On the other hand, the activated P18 of caspase8 and the activated caspase3 were accumulated from dark to blue light irradiation (Fig. 1D), suggesting that the activity of PHR-Caspase8 could be controlled by blue light to active the downstream signaling pathway in blue light.

Blue light induce opto-caspase8 cluster and self-cleavage

Building upon earlier findings that the *Arabidopsis* blue light receptor CRY2 can interact with the transcription factor CIB1 in a blue light-dependent manner, we created an optogenetic tool for caspase8 by fusing the N-terminal domain of CIB1 (amino acids 1–170) with caspase8 to generate CIB1N-caspase8, which was then combined with PHR-caspase8 to produce Opto-Casp8-V2 (Fig. 2A). This design ensured that the optogenetic system could effectively regulate the caspase8-mediated signaling pathway via blue light. To validate this approach, we transfected HEK293T cells with GFP-PHR-caspase8/Flag-CIB1N-caspase8 (Opto-Casp8-V2 cassette), treated the cells with blue light, and subsequently lysed them. We performed immunoprecipitation (IP) with GFP trap and observed that all the proteins of GFP-PHR-caspase8/Flag-CIB1N-caspase8 underwent blue light-dependent cleavage and interacted in a blue light-enhanced manner in co-IP (Fig. 2B). These results indicate that Opto-Casp8-V2 can effectively function in cells, similar to Opto-Casp8-V1.

We next evaluated the effectiveness of PHR-caspase8 and CIB1N-caspase8 in the human cervical cancer cell line HeLa. We firstly transfected HeLa cells with PHR-caspase8 and CIB1N-caspase8, treated them with blue light, and observed that PHR-caspase8 exhibited highly efficient self-cleavage and induced downstream accumulation of caspase3, whereas CIB1N-caspase8 could not be activated by blue light (Fig. 2C). We compared the cleavage efficiency mediated by Opto-Casp8-V1 and Opto-Casp8-V2 in HeLa cells and found that Opto-Casp8-V2 demonstrated significantly more effective self-cleavage and consumption than Opto-Casp8-V1 under blue light, indicating that CIB1N-caspase8 could enhance the activation of PHR-caspase8 in the Opto-Casp8-V2 optogenetics cassette (Fig. 2D). This finding further suggests that, in addition to the oligomerization of CRY2, the CRY2-CIB1 protein–protein interaction can further promote the activation of precursor caspase8 in blue light.

Optogenetic control of caspase8-mediated apoptosis and programmed cell death

We utilized FAM-LETD-FMK caspase-8 to evaluate the effectiveness of caspase-8 activation in live cells using our Caspase8 optogenetic tool. Plasmids encoding Opto-Caspase8-V1 and Opto-Casp8-V2 were transfected into HEK293T cells, and after blue light stimulation, the cells were labeled with FAM-LETD-FMK and imaged using an inverted microscope (Zeiss Axio Observer A1). Our results demonstrated that Opto-Casp8-V1 and Opto-Casp8-V2 induced greater activation of caspase-8 in live cells than cells transfected with the CIB1N-Caspase8 control (Fig. 3A,B). Moreover, Opto-Casp8-V2 exhibited significantly higher activation efficiency than Opto-Casp8-V1 (Fig. 3A,B), confirming the usefulness of CIB1N-Caspase8 as an optogenetic tool for caspase8 signaling pathway activation. We further examined the morphological changes in cells transfected with Opto-Casp8-V1 and Opto-Casp8-V2, and our data showed that Opto-Casp8 induced more active Caspase8 in HEK293T cells after effective self-cleavage under blue light, resulting in apoptosis (Fig. 3A,B). A time-course assay of FAM-LETD-FMK caspase-8 activity demonstrated a positive correlation between blue light treatment duration and FAM-LETD-FMK caspase-8 activity (Fig. 3C), further supporting the ability of Opto-Casp8 to trigger apoptosis in human cells.

To investigate the efficacy of Opto-Casp8-V1 and Opto-Casp8-V2 in inducing cell apoptosis via the caspase8-mediated signaling pathway under blue light, we employed flow cytometry to monitor apoptosis in cells induced by Opto-Casp8-V1 and Opto-Casp8-V2. During apoptosis, phosphatidylserine (PS) flips to the outer layer of the lipid membrane[19,20]. Annexin-V is a Ca^{2+}-dependent phospholipid-binding protein with a molecular weight of 35–36 kD, which exhibits high affinity for phosphatidylserine[21]. The combination of phosphatidylserine and Annexin-V exposed on the lateral side of the cell is indicative of cell apoptosis[22]. Therefore, we used Alexa Fluor® 488 Annexin-V/PI with flow cytometry to verify cell apoptosis. Plasmids encoding Opto-Casp8-V1 and Opto-Casp8-V2 were transfected into HeLa cells, and after blue light stimulation, Alexa Fluor® 488 Annexin-V and PI-labeled cells were detected by flow cytometry. Our results indicated that compared to the control group (CIB1N-Caspase8), Opto-Casp8-V1 and Opto-Casp8-V2 significantly promoted cell apoptosis under blue light (Fig. 3D). Additionally, Opto-Casp8-V2 exhibited a stronger effect on promoting cell apoptosis than Opto-Casp8-V1 (Fig. 3D,E), which is consistent with the previous results of cleavage efficiency. Similarly, we employed flow cytometry to assess caspase-8 activity in HeLa cells following transfection with Opto-Casp8-V1 and Opto-Casp8-V2 plasmids. The results revealed that, after transfection with Opto-Casp8-V2, its activity was significantly higher than Opto-Casp8-V1, and both Opto-Casp8-V1 and Opto-Casp8-V2 activities were

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Optogenetic induction of caspase-8 mediated apoptosis by employing *Arabidopsis* cryptochrome 2

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Apoptosis, a programmed cell death mechanism, is a regulatory process controlling cell proliferation as cells undergo demise. Caspase-8 serves as a pivotal apoptosis-inducing factor that initiates the death receptor-mediated apoptosis pathway. In this investigation, we have devised an optogenetic method to swiftly modulate caspase-8 activation in response to blue light. The cornerstone of our optogenetic tool relies on the PHR domain of *Arabidopsis thaliana* cryptochrome 2, which self-oligomerizes upon exposure to blue light. In this study, we have developed two optogenetic approaches for rapidly controlling caspase-8 activation in response to blue light in cellular systems. The first strategy, denoted as Opto-Casp8-V1, entails the fusion expression of the *Arabidopsis* blue light receptor CRY2 N-terminal PHR domain with caspase-8. The second strategy, referred to as Opto-Casp8-V2, involves the independent fusion expression of caspase-8 with the PHR domain and the CRY2 blue light-interacting protein CIB1 N-terminal CIB1N. Upon induction with blue light, PHR undergoes aggregation, leading to caspase-8 aggregation. Additionally, the blue light-dependent interaction between PHR and CIB1N also results in caspase-8 aggregation. We have validated these strategies in both HEK293T and HeLa cells. The findings reveal that both strategies are capable of inducing apoptosis, with Opto-Casp8-V2 demonstrating significantly superior efficiency compared to Opto-Casp8-V1.

Apoptosis, a programmed cell death, is a complex process regulated by multiple genes that maintains organism development and internal environment homeostasis[1,2]. It is a cellular response to specific information from the environment such as information transmission, gene expression, and protein synthesis. Physiological and pathological stimuli can trigger apoptosis via three main pathways: death receptor, mitochondrial signals, and endoplasmic reticulum signals[3]. The death receptor pathway is mainly activated by caspase, a cysteine-containing aspartate-specific protease, which is the core component of this pathway[4]. In terms of mechanism, cell apoptosis is controlled through two pathways: the extrinsic/death receptor pathway and the intrinsic/mitochondrial pathway. Both of these pathways involve apoptotic initiators and caspases, which are members of the cysteine-aspartic protease family capable of cleaving substrate proteins that promote cell death. Specifically, the extrinsic/death receptor pathway includes Caspase-8, while the intrinsic/mitochondrial pathway involves Caspase-9 and Caspase-3/-7[5]. Caspase 8, a key promoter in the death receptor-mediated apoptosis pathway, is activated through oligomerization and self-cleavage, which triggers downstream caspases and ultimately leads to cell apoptosis[6]. Therefore, the controlled oligomerization of caspase 8 can achieve automatic regulation of apoptosis. However, to regulate caspase 8 cellular function precisely, the intensity, location, and duration of signaling events need to be modulated, especially for the activation process, in which the oligomerization of caspase 8 needs to be under microscale control for downstream ultrasensitive digital signaling responses.

It has long been established that caspases can cleave and activate themselves or other proteins through their aspartate-specific proteolytic enzyme activity. To induce aggregation activation of Caspase 8, a fusion was created by linking the Caspase 8 precursor with the dimerization domain FKBP (the binding domain of FK506). When the dimerization ligand FK1012 homodimerizes, the fused Caspase 8 precursor also polymerizes, leading to dimerization of the Caspase 8 precursor and subsequent activation[7,8]. Dixit and Salvesen consolidated these findings under the Induced Proximity Model (IPM)[9]. According to this model, when zymogens are recruited to

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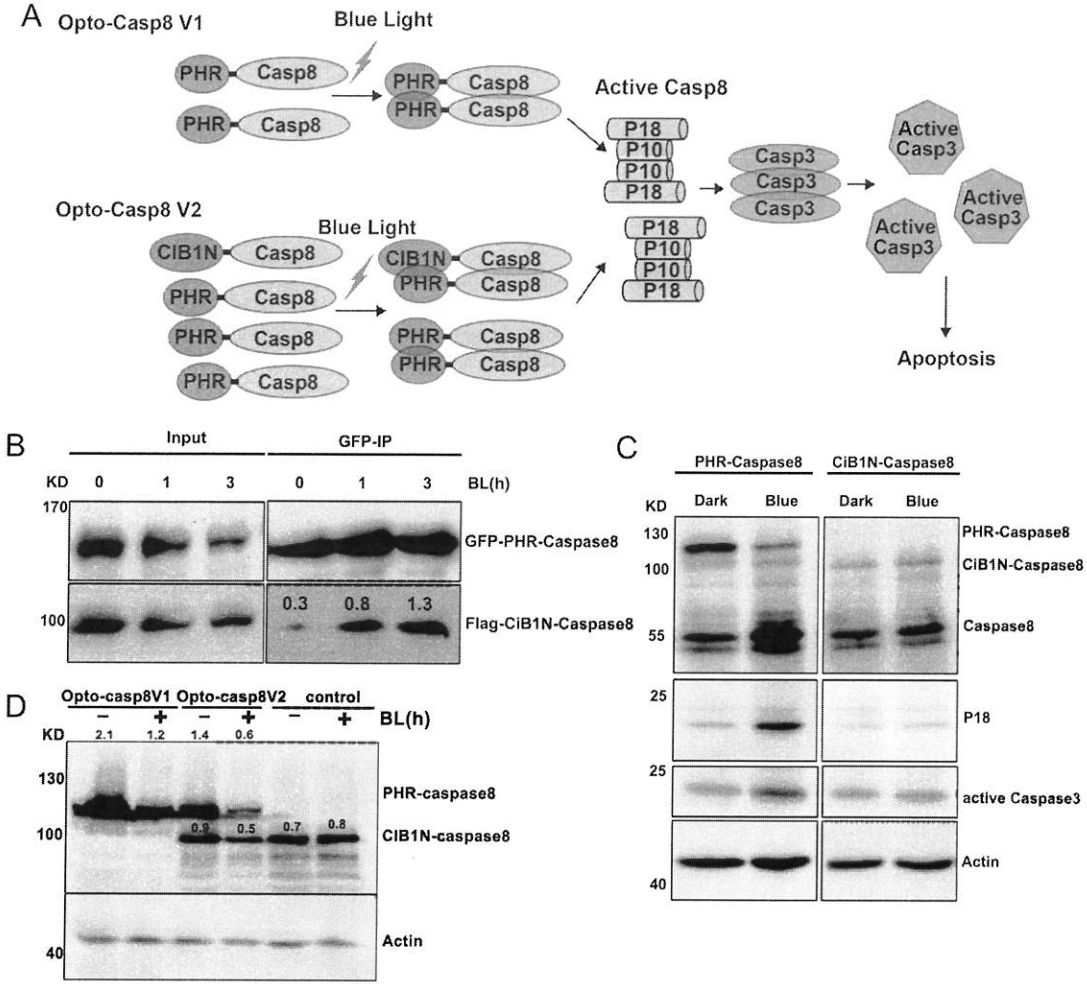

Figure 2. Blue light trigger Opto-Caspase8 cleavage and activation. (**A**) N-terminal part of cryptochrome interacting basic-helix–loop–helix protein CIB1 (amino acids 1–170, CIB1N) are fused with Caspase8, PHR-Caspase8 is marked as Opto-Casp8 V1 and PHR-Caspase8/CiB1N-Caspase8 as Opto-Casp8 V2. CIB1N, the N-terminal domain of CIB1. (**B**) Co-IP assay showed blue light enhanced the interaction of Opto-Caspase8 V2 in HEK293 T. Transfected HEK293T cells were treated with blue light (30 μmol m^{-2} s^{-1}) for indicated time before lysed. The immunoprecipitation signals were probed by anti-GFP or anti-Flag, respectively. (**C**) Blue light actives Opto-Caspase8 V1 in HeLa cells. (**D**) Cleavage efficiency of caspase8 between Opto-Caspase8 V1 and Opto-Caspase8 V2 in Hela cells.

noticeably higher than the control (Fig. 4A,B). This indicates that Opto-Casp8-V1 and Opto-Casp8-V2 are capable of inducing cell apoptosis in HeLa cells.

The process of apoptosis involves multiple stages of morphological changes. In the initial stage, chromatin condenses and separates, distributing along the nuclear membrane. The cytoplasm also undergoes shrinkage, but its membrane remains intact with selective permeability. In the late stage of apoptosis, the chromatin breaks into fragments of varying sizes, which aggregate with organelles such as mitochondria and are surrounded by the inverted cell membrane. Subsequently, they gradually separate to form condensed nuclei[23,24]. In light of these observations, we employed DAPI nuclear staining to detect programmed cell death induced by Opto-Casp8. Our results demonstrated that Opto-Casp8 V1 and Opto-Casp8-V2 triggered the shrinkage of a large number of nuclei to form condensed nuclei, ultimately promoting cell death (Fig. 4C,D). In summary, our optogenetic tools (Opto-Casp8-V1 and Opto-Casp8-V2) exhibit caspase-8 activation activity in a blue light-dependent manner, subsequently activating downstream proteins to control cell apoptosis with blue light.

Discussion

Caspase 8 is a critical factor in the apoptotic program of cell death, and its activation is essential for the functionality of the apoptotic pathway. Resistance to cell apoptosis is not only a hallmark of cancer but also significantly associated with enhanced drug resistance in tumor cells[25]. Targeted cell apoptosis is considered a highly promising approach in cancer treatment[26]. In this study, we developed Opto-Casp8 as a genetic tool for regulating cell apoptosis. Our results demonstrate that Opto-Casp8 induces apoptosis in HEK293T and HeLa cells, consistent with previous studies using Opto-BAX with CRY2-CIB1 or Opto-Caspase7 to regulate cell apoptosis[13,14].

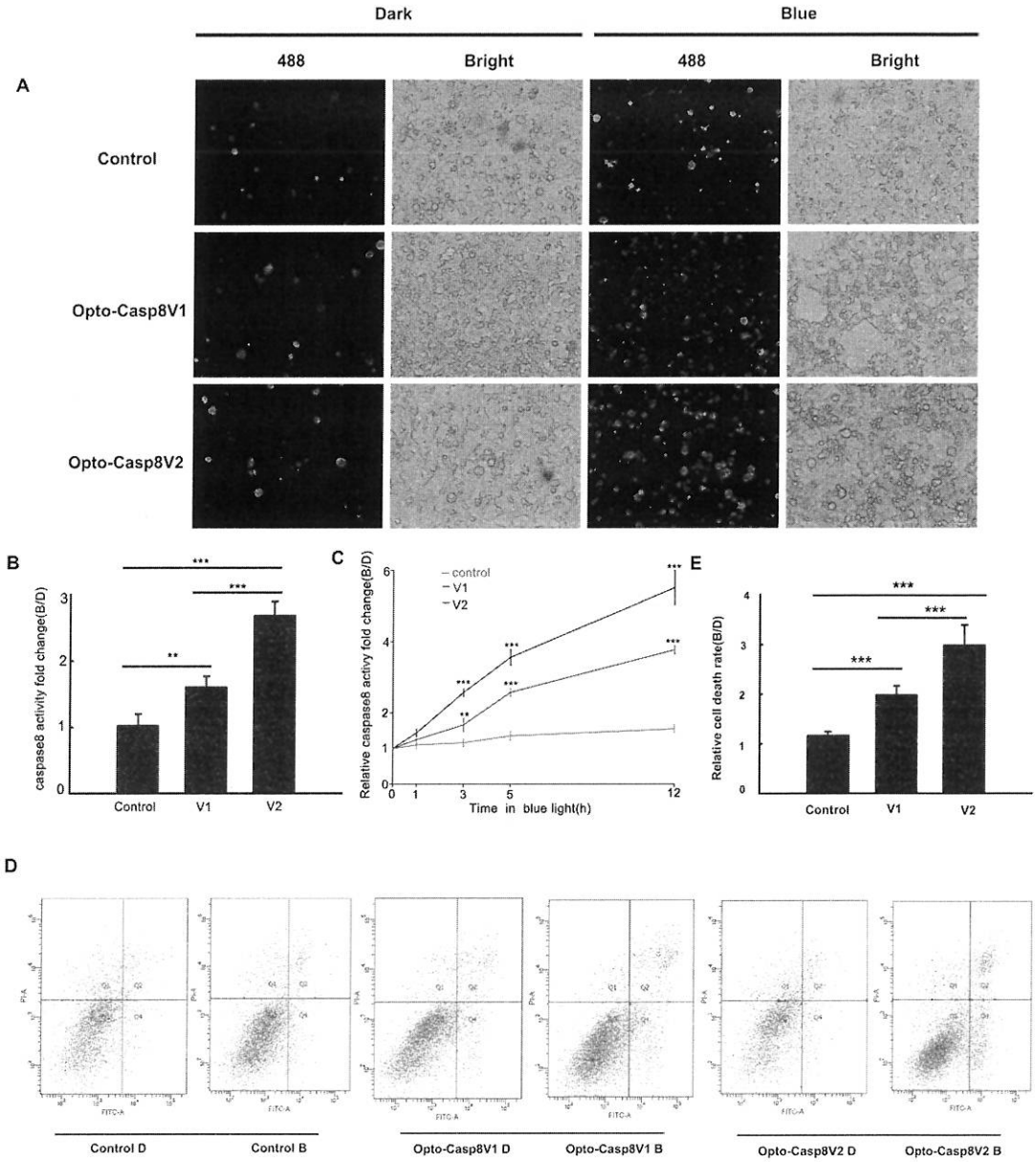

Figure 3. Blue light dependent apoptosis activated by Opto-Caspase8. (**A**) Active caspase8 was detected by fluorescence microscope labeled by FAM-LETD-FMK caspase-8 in HEK293T cells. Blue light (50 μmol m^{-2} s^{-1} for 3 h) treated transfected cells were incubated with FAM-LETD-FMK caspase-8 before capturing pictures, CiB1N-Caspase8 and the empty vector pci myc were co-transfected as control. (**B**) Caspase8 activity analysis in (**A**), activities = fluorescent intensity of caspase8 in blue/fluorescent intensity of caspase8 in dark. Data are presented as mean ± SD (n = 3). Student's t test: ***p < 0.001. (**C**) Time course of FAM-LETD-FMK labeled caspase-8 activity assay in HEK293T cells. Same operation were conducted as showed in (**A,B**). Data are presented as mean ± SD (n = 3). Student's t test: ***p < 0.001. (**D**) Cell viability analysis in HeLa cells expressed Opto-Caspase8 V1 or Opto-Caspase8 V2 by flow cytometry. Alexa Fluor® 488 annexin and PI was used to detect apoptosis cells. The transfected cells were either shielded or exposed to continuous blue light (50 μmol m^{-2} s^{-1}) for 3 h. (**E**) Relative cell death rate analysis in (**D**). Rate = number of apoptosis cells in blue/number of apoptosis cells in dark. Data are presented as mean ± SD (n = 3). Student's t test: ***p < 0.001.

However, our study specifically focuses on Caspase8, an upstream protein in the apoptosis pathway, rather than downstream proteins such as BAX (which regulates the mitochondrial pathway) or Caspase3. Caspase8 mediates cell apoptosis not only through the mitochondrial pathway but also through other caspases, such as Caspase3/7/9, providing a wider regulatory range.

Recent reports suggest that Caspase-8 not only acts as the initiator of extrinsic apoptosis but also as a molecular switch for necroptosis and pyroptosis[13]. Compared to optogenetics tools based on BAX or Caspase3, our

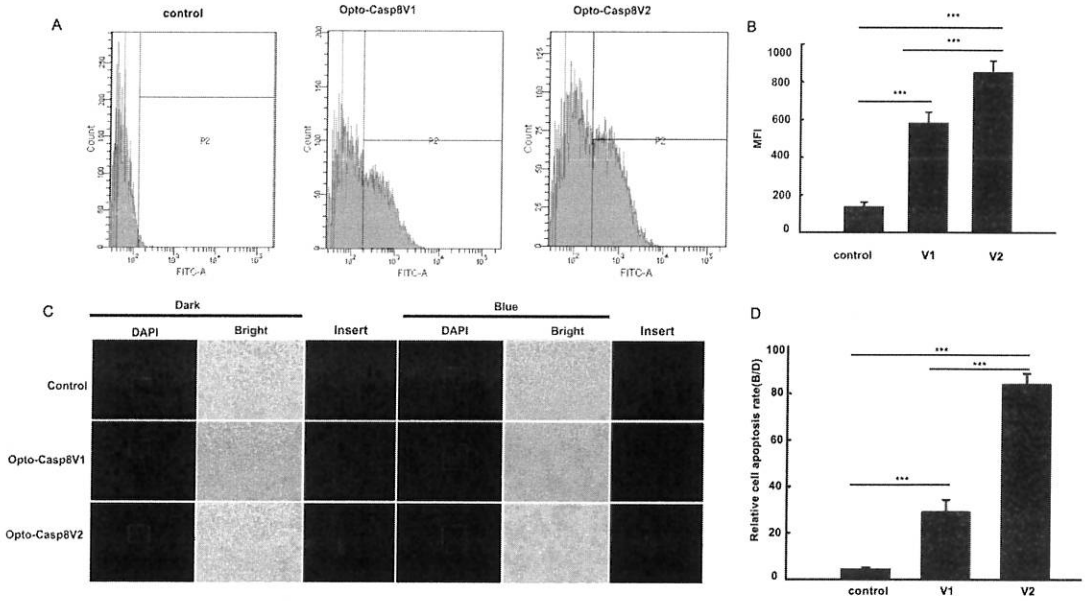

Figure 4. Opto-Caspase8 cause cell death in HeLa cells. (**A**) Detecting the caspase-8 activity with FAM-LETD-FMK with flow cytometry in HeLa cells under blue light, the empty vector was transfected as control. (**B**) Mean fluorescence intensity (NFI) of (**A**). Data are presented as mean ± SD (n = 3). Student's t test: ***$p < 0.001$. (**C**) The transfected cells were either shielded or exposed to pulse blue light (50 μmol m^{-2} s^{-1}) for 12 h and incubated with DAPI, after washing for 3 times, pictures were obtained by Zeiss Axio Observer A1 microscope, CiB1N-Caspase8 and the empty vector pci myc were co-transfected as control. (**D**) Relative cell apoptosis rate analysis of (**A**), rate = number of condensed nuclei in blue/number of condensed nuclei in dark. Data are presented as mean ± SD (n = 3). Student's t test: ***$p < 0.001$.

optogenetics cassette (Opto-Casp8-V1 and Opto-Casp8-V2) has more widespread applications. Our optogenetics tool can precisely control Caspase-8 activity to induce cell apoptosis and utilize blue light to control the activation of the inflammasome and induction of pyroptosis when apoptosis and necroptosis are compromised. In the future, we expect that our optogenetics tools can regulate Caspase-8-mediated signaling pathways to tailor specific immune responses against pathogens and switch different modes of cell death. However, further research is needed to determine the most effective optogenetic tools and conditions and how to apply them to different types of cancer.

In addition, there is research exploring the use of a similar approach, fusing a mutant CRY2PHR oligo with caspase8, thereby regulating cell apoptosis through blue light[15]. What sets our study apart is that we not only validated the feasibility of PHR-caspase8 in HEK293T cells but also in cancer cells, specifically HeLa cells. The results indicated its ability to induce apoptosis in HeLa cells, broadening the potential applications of this tool and may providing an additional option for optogenetic cancer therapy.

Another crucial point of discussion is the safety of optogenetic methods. When using these tools, it is essential to ensure that they do not have adverse effects on healthy cells, and a thorough investigation of potential risks is required. Furthermore, the impact of different types of light sources and illumination conditions on cells needs to be taken into account to determine the optimal experimental conditions. Optogenetics also offers a novel approach to the spatiotemporal control of cell apoptosis research. By adjusting the timing and location of light exposure, researchers can precisely regulate the occurrence of cell apoptosis, contributing to a better understanding of the apoptotic processes in different cell types and tissues. In conclusion, optogenetics, as a novel approach to controlling cell apoptosis, presents exciting opportunities. However, further research is required to address the challenges and safety concerns in its application.

Data availability

The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

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References

1. Shi, Y. Mechanisms of caspase activation and inhibition during apoptosis. *Mol. Cell* **9**, 459–470 (2002).
2. Budihardjo, I., Oliver, H., Lutter, M., Luo, X. & Wang, X. Biochemical pathways of caspase activation during apoptosis. *Annu. Rev. Cell Dev. Biol.* **15**, 269–290 (1999).
3. Crawford, E. D. & Wells, J. A. Caspase substrates and cellular remodeling. *Annu. Rev. Biochem.* **80**, 1055–1087 (2011).

4. Fussenegger, M., Bailey, J. E. & Varner, J. A mathematical model of caspase function in apoptosis. *Nat. Biotechnol.* **18**, 768–774 (2000).
5. Fiandalo, M. V. & Kyprianou, N. Caspase control: Protagonists of cancer cell apoptosis. *Exp. Oncol.* **34**, 165–175 (2012).
6. Fulda, S. Caspase-8 in cancer biology and therapy. *Cancer Lett.* **281**, 128–133 (2009).
7. Muzio, M., Stockwell, B. R., Stennicke, H. R., Salvesen, G. S. & Dixit, V. M. An induced proximity model for caspase-8 activation. *J. Biol. Chem.* **273**, 2926–2930 (1998).
8. Shi, Y. Caspase activation: Revisiting the induced proximity model. *Cell* **117**, 855–858 (2004).
9. Salvesen, G. S. & Dixit, V. M. Caspase activation: The induced-proximity model. *Proc. Natl. Acad. Sci. U.S.A.* **96**, 10964–10967 (1999).
10. Mandal, R. *et al.* pERK 1/2 inhibit Caspase-8 induced apoptosis in cancer cells by phosphorylating it in a cell cycle specific manner. *Mol. Oncol.* **8**, 232–249 (2014).
11. Ozören, N. & El-Deiry, W. S. Defining characteristics of types I and II apoptotic cells in response to TRAIL. *Neoplasia* **4**, 551–557 (2002).
12. Chang, D. W., Xing, Z., Capacio, V. L., Peter, M. E. & Yang, X. Interdimer processing mechanism of procaspase-8 activation. *EMBO J.* **22**, 4132–4142 (2003).
13. Mills, E., Chen, X., Pham, E., Wong, S. & Truong, K. Engineering a photoactivated caspase-7 for rapid induction of apoptosis. *ACS Synth. Biol.* **1**, 75–82 (2012).
14. Smart, A. D. *et al.* Engineering a light-activated caspase-3 for precise ablation of neurons in vivo. *Proc. Natl. Acad. Sci. U.S.A.* **114**, E8174–E8183 (2017).
15. Shkarina, K. *et al.* Optogenetic activators of apoptosis, necroptosis, and pyroptosis. *J. Cell Biol.* **221**, 38 (2022).
16. Liu, H. *et al.* Photoexcited CRY2 interacts with CIB1 to regulate transcription and floral initiation in Arabidopsis. *Science* **322**, 1535–1539 (2008).
17. Duan, L. *et al.* Understanding CRY2 interactions for optical control of intracellular signaling. *Nat. Commun.* **8**, 547 (2017).
18. Bugaj, L. J., Choksi, A. T., Mesuda, C. K., Kane, R. S. & Schaffer, D. V. Optogenetic protein clustering and signaling activation in mammalian cells. *Nat. Methods* **10**, 249–252 (2013).
19. Nagata, S. Apoptosis and clearance of apoptotic cells. *Annu. Rev. Immunol.* **36**, 489–517 (2018).
20. Wuest, M., Perreault, A., Richter, S., Knight, J. C. & Wuest, F. Targeting phosphatidylserine for radionuclide-based molecular imaging of apoptosis. *Apoptosis* **24**, 221–244 (2019).
21. Majtnerová, P. & Roušar, T. An overview of apoptosis assays detecting DNA fragmentation. *Mol. Biol. Rep.* **45**, 1469–1478 (2018).
22. D'Arcy, M. S. Cell death: A review of the major forms of apoptosis, necrosis and autophagy. *Cell Biol. Int.* **43**, 582–592 (2019).
23. Godwin, W. C., Hoffmann, G. F., Gray, T. J. & Hughes, R. M. Imaging of morphological and biochemical hallmarks of apoptosis with optimized optogenetic tools. *J. Biol. Chem.* **294**, 16918–16929 (2019).
24. Rogalińska, M. Alterations in cell nuclei during apoptosis. *Cell. Mol. Biol. Lett.* **7**, 995–1018 (2002).
25. Hanahan, D. & Weinberg, R. A. Hallmarks of cancer: The next generation. *Cell* **144**, 646–674 (2011).
26. Pfeffer, C. M. & Singh, A. T. K. Apoptosis: A target for anticancer therapy. *Int. J. Mol. Sci.* **19**, 448 (2018).

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Author contributions

R.Z. and Z.Z. conceived and designed the study. W.M. and R.S. conducted Co-IP and western blot assays, X.Z., L.Y. and C.W. conducted microscope imaging, and L.W. conducted flow cytometry assays. The other authors were involved in plasmid construction, cell culture, and figure preparation. W.M. and Z.Z. wrote the manuscript. All authors have read and agreed to the final version of the manuscript.

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Competing interests

The authors declare no competing interests.

Additional information

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