# 10 Supplementary material

## 10. 1 Aggression scores

| Uncontrolled Trajectory Approximations | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **N.I.** | **KRAS** | **TP53** | **CycD** | **SMAD** | **T-K** | **C-K** | **S-K** | **T-C** | **T-S** | **C-S** | **T-C-K** | **T-S-K** | **C-S-K** | **T-C-S** | **T-C-S-K** |
| $\text{Aut}_c$ | 0.509 | 0.531 | 0.964 | 0.818 | 0.528 | 0.974 | 0.827 | 0.356 | 0.953 | 0.955 | 0.817 | 0.97 | 0.967 | 0.82 | 0.962 | 0.961 |
| $\text{Apo}_c$ | 0.359 | 0.356 | 0.021 | 0.109 | 0.343 | 0.011 | 0.118 | 0.479 | 0.025 | 0.035 | 0.105 | 0.018 | 0.013 | 0.1 | 0.028 | 0.017 |
| $\text{Pro}_c$ | 0.213 | 0.209 | 0.56 | 0.017 | 0.211 | 0.555 | 0.013 | 0.24 | 0.018 | 0.92 | 0.012 | 0.015 | 0.944 | 0.018 | 0.01 | 0.014 |

Table 1: *Expression Approximations*. This table records the approximate phenotype expressions for the PCC in Figure 10. Given 1,000 random initial states, these results show trajectory approximations after 300 time steps (i.e. function updates) with $1\%$ noise.

We derived aggressiveness scores for each mutation combination using long-term trajectory approximations. Simulations were run using 1000 random initializations, 300 time steps, and $1\%$ noise to achieve an approximate probability of phenotype expression. In Table, 1, we see that the non-induced (N.I.) system showed levels of $51\%$ autophagy, $36\%$ apoptosis, and $21\%$ proliferation. The heat maps in Figure 3 are sorted with column-wise mutation groups and used to compare cancer cell autophagy and proliferation while giving a negative weight ($\omega = -1$) to apoptosis. The row label "Same" indicates that the same weight was given to both autophagy and proliferation (used value $\omega = 2$ for both), "High/Low" indicates a high weight for autophagy ($\omega = 10$) but a low weight for proliferation ($\omega = 2$), and "Low/High" indicates a low weight for autophagy ($\omega = 2$) but a high weight for proliferation ($\omega = 10$). Thus, scores were calculated using:

$$\text{Score} = \text{Aut}_c \times \omega_1 + \text{Pro}_c \times \omega_2 + \text{Apop}_c \times (-1) \quad \text{where} \quad \omega_{1,2} \in \{2, 10\}$$

Scaling of the heat map ranges orange (low score) to red (high score) based on the maximum and minimum values in each table. However, blue shading (i.e. cold) indicates a negative score, which is interpreted as successful depletion of aggression. See [23] sections 2.3 and 4.4 for more details.

Lastly, we justify the positive weight given to autophagy, which is a natural process where cells heal themselves. The cell will break down any damaged or unnecessary components, and it will reallocate the nutrients from these processes to those that are essential. However, studies have shown that autophagy is required for pancreatic tumor growth [39]. Autophagy can help tumors overcome conditions such as hypoxia and nutrient deprivation. Within tumors, cells can exist under hypoxic conditions. If activated autophagy is then suppressed by deletion of Beclin 1, studies have shown increased cell death. It has also been observed that autophagy is increased in KRAS mutated cells, and aids in survival of the cancer cells while experiencing nutrient starvation. Further, animal studies have shown that autophagy contributes to tumor-cell survival by enhancing stress tolerance and supplying nutrients to meet the metabolic demands of tumors. Once suppression of autophagy occurred, there was an observance of tumor-cell death [40].

Note: our aggression scores are based on combinations autophagy, apoptosis, and proliferation, merely one method among many for estimating aggression. Moreover, the attractor analysis (see [11, 23] indicated that certain mutation combinations yield a large basin for attractors with both autophagy and proliferation expression. It is likely that modular structure alone is not enough to determine aggression and target cardinality. Rather, it should be used alongside other analyses.

## 10. 2 Boolean pancreatic cancer model and functions

| Cytokines | (Cyan) |
|---|---|
| VEGF | NF$\kappa$Bs \| STATp \| NF$\kappa$Bp |
| EGF | cJUNp |
| bFGF | ERKs \| ERKp |
| TNF$\alpha$ | TNF$\alpha$ |
| PDGFBB | SMADs \| SMADp |
| Thiazolidinedione | Thiazolidinedione |
| TGF$\beta$1 | SMADs \| SMADp |
| IFN$\gamma$ | IFN$\gamma$ |
| Pancreatic Stellate Cell | (Green) |
| VEGFRs | VEGF |
| EGFRs | EGF |
| FGFRs | bFGF |
| TNFRs | TNF$\alpha$ |
| PDGFBBRs | PDGFBB |
| PPAR$\gamma$s | Thiazolidinedione |
| TGFRs | TGF$\beta$1 |
| IFNGRs | IFN$\gamma$ |
| RASs | ( ( VEGFRs ) \| ( EGFRs ) \| ( FGFRs ) ) |
| PIK3s | ( ( PDGFBBRs ) \| ( RASs ) ) |
| SMADs | TGFRs |
| STATs | IFNGRs |
| RAFs | RASs |
| NF$\kappa$Bs | ( ( TNFRs ) \| ( AKTs ) ) |
| P38s | ( ( MEKs ) \| ( P53s ) ) |
| MEKs | RAFs |
| PIP3s | ( $\sim$ PTENs ) & ( PIK3s ) |
| P53s | ( $\sim$ MDM2s ) & ( P38s ) |
| PTENs | P53s |
| AKTs | PIP3s |
| ERKs | ( PDGFBBRs ) \| ( MEKs ) |
| AP1s | ERKs |
| P21s | P53s |
| MDM2s | ( ( AKTs ) \| ( P53s ) ) |
| Apos | P53s |
| Pros | ( $\sim$ P21s ) & ( AP1s ) |
| Migs | ( AKTs ) & ( AP1s ) & ( Acts ) |
| Acts | SMADs & (($\sim$STATs ) \| ( $\sim$PPAR$\gamma$s )) & ( NF$\kappa$Bs \| AP1s ) |
| Pancreatic Cancer Cell | (Purple) |
| EGFRc | EGF \| HER2c |
| FGFRc | bFGF |
| TGFRc | TGF$\beta$1 |
| HER2c | HER2c |
| JAK1c | HER2c |

| | |
|---|---|
| PIK3c | ( EGFRc ) \| ( RASc ) |
| RASc | ( EGFRc ) \| ( FGFRc ) |
| SMADc | TGFRc |
| STATc | JAK1c |
| PIP3c | ( $\sim$ PTENc ) & ( PIK3c ) |
| RAFc | RASc |
| P21c | ( $\sim$ STATc ) & ( ( SMADc ) \| ( P53c ) ) |
| MEKc | RAFc |
| NF$\kappa$Bc | AKTc |
| AKTc | PIP3c |
| PTENc | P53c |
| ERKc | MEKc |
| E2Fc | ( $\sim$ RBc ) |
| cJUNc | ( ERKc ) \| ( JNKc ) |
| CyclinDc | ( $\sim$ P21c ) & ( NF$\kappa$Bc ) |
| RBc | ( $\sim$ CyclinDc ) |
| BCLXLc | ($\sim$ P53c) & ( (NF$\kappa$Bc) \| (STATc) \| (AKTc) \| (JNKc) ) |
| JNKc | MEKc |
| mTORc | ( $\sim$ cJUNc ) & ( AKTc ) |
| BAXc | ( $\sim$ BCLXLc ) |
| Beclin1c | ( $\sim$ BCLXLc ) & ( $\sim$ CASPc ) |
| MDM2c | ( $\sim$ E2Fc ) & ( AKTc \| P53c ) |
| P53c | ( $\sim$ MDM2c ) |
| CyclinEc | ( $\sim$ P21c ) & ( E2Fc ) |
| CASPc | ( $\sim$ NF$\kappa$Bc ) & ( ( P53c ) \| ( Beclin1c ) \| ( BAXc ) ) |
| Autc | ( $\sim$ mTORc ) & ( ( NF$\kappa$Bc ) \| ( Beclin1c ) ) |
| Apoc | CASPc |
| Proc | ( CyclinEc ) & (( JNKc ) \| ( cJUNc )) |

Table 2: *Boolean functions for the whole pancreatic cancer model.* Each function indicates the next state of the node in terms of the current states of said nodes' regulators. Activation is written as OR statements, while suppression is written as AND NOT. The exception to this rule is PCC proliferation, because of its upstream signaling.

| Cytokines | (Cyan) |
|---|---|
| TNF$\alpha$ | TNF$\alpha$ |
| Thiazolidinedione | Thiazolidinedione |
| TGF$\beta$1 | TGF$\beta$1 |
| IFN$\gamma$ | IFN$\gamma$ |
| Stellate Cell | (Green) |
| RASs | ( ( NF$\kappa$Bs ) \| ( STATp ) \| ( PIP3p ) \| ( RASp ) \| ( ERKs ) ) |
| NF$\kappa$Bs | ( ( TNF$\alpha$ ) \| ( PIP3s ) ) |
| PIP3s | ( $\sim$ P53s ) & ( ( TGF$\beta$1 ) \| ( RASs ) ) |
| P53s | ( $\sim$ PIP3s ) & ( $\sim$ P53s ) & ( RASs ) |
| ERKs | ( TGF$\beta$1 ) \| ( RASs ) |
| Pros | ( $\sim$ P53s ) & ( ERKs ) |
| Migs | ( PIP3s ) & ( ERKs ) & ( Acts ) |
| Acts | ( TGF$\beta$1 ) & ( ( $\sim$ IFN$\gamma$ ) \| ( $\sim$ Thiaz. ) ) & ( ( NF$\kappa$Bs ) \| ( ERKs ) ) |
| Pancreatic Cell | (Purple) |
| HER2p | HER2p |
| RASp | ( RASp ) \| ( HER2p ) \| ( ERKs ) |
| STATp | HER2p |
| PIP3p | ( $\sim$ P53p ) & ( ( RASp ) \| ( HER2p ) ) |
| P21p | ( $\sim$ STATp ) & ( ( TGF$\beta$1 ) \| ( P53p ) ) |
| BCLXLp | ( $\sim$ P53p ) & ( ( PIP3p ) \| ( STATp ) \| ( RASp ) ) |
| P53p | $\sim$ ( ( ( P21p ) \| ( $\sim$ PIP3p ) ) & ( ( PIP3p ) \| ( P53p ) ) ) |
| CASPp | ( $\sim$ PIP3p ) & ( ( P53p ) \| ( $\sim$ BCLXLp ) ) |
| Autp | ( ( RASp ) \| ( $\sim$ PIP3p ) ) & ( ( PIP3p ) \| ( ( $\sim$ BCLXLp ) & ($\sim$ CASPp ) ) ) |
| Prop | ( $\sim$ P21p ) & ( PIP3p ) & ( RASp ) |

Table 3: *Boolean functions for the reduced pancreatic cancer model.* Each function indicates the next state of the node in terms of the current states of said nodes' regulators. Activation is written as OR statements, while suppression is written as AND NOT. Functions maintain the rules from the whole model by substituting values from the deleted nodes.

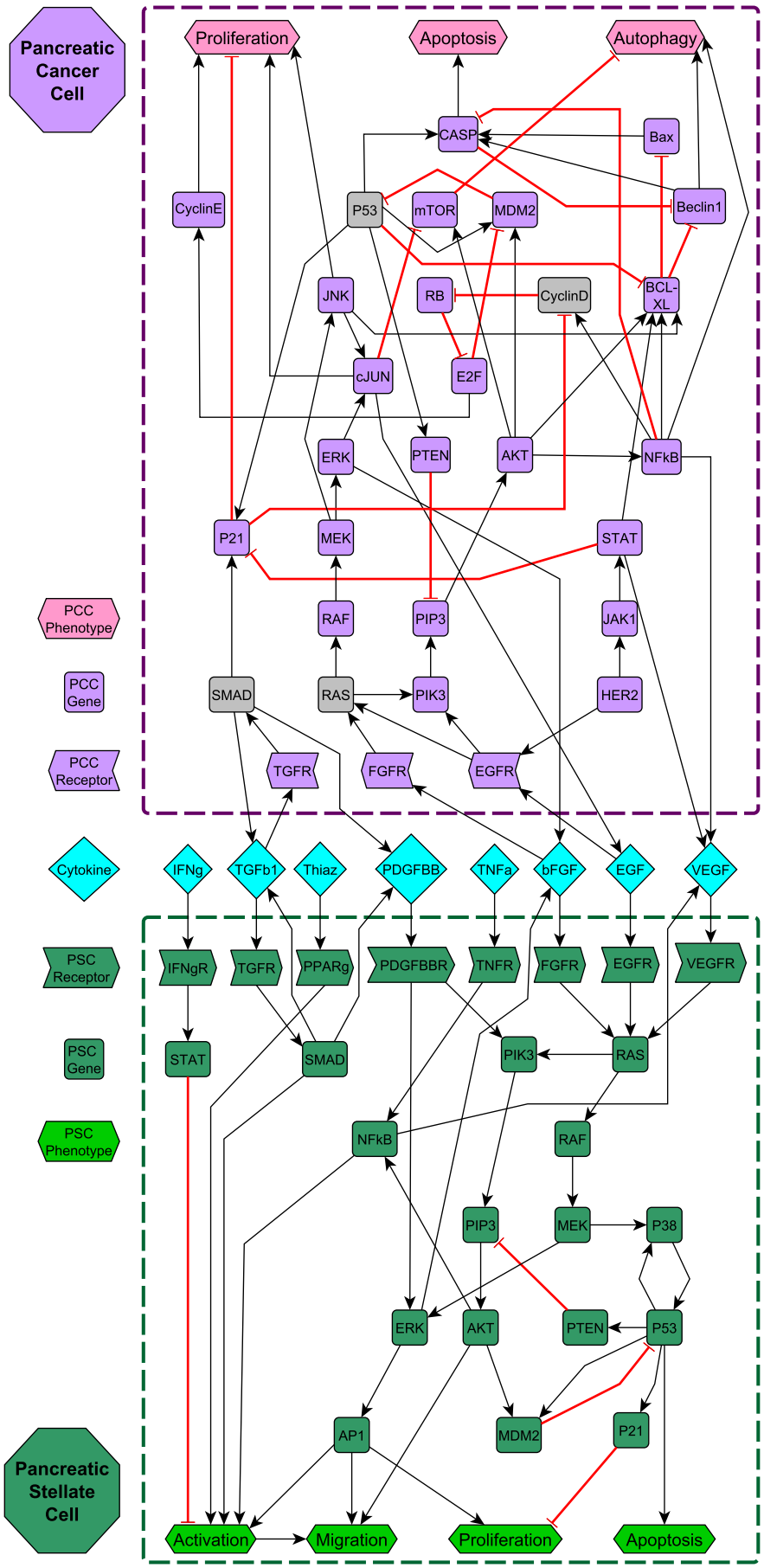
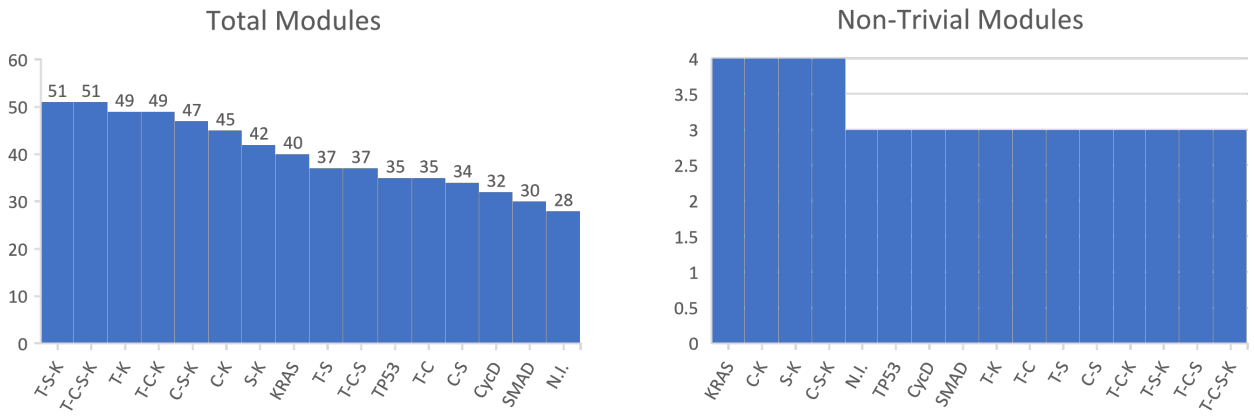



Figure 10:
*Gene regulatory network model of pancreatic cancer.*
Shapes and colors of nodes indicate their function and cell type (respectively), as shown in the legend. Black barbed arrows indicate signal expression, while red bar arrows indicate suppression. Grey nodes located in the PCC indicate prevalent mutant genes [11, 29].

## 10. 3 Tables and Graphs

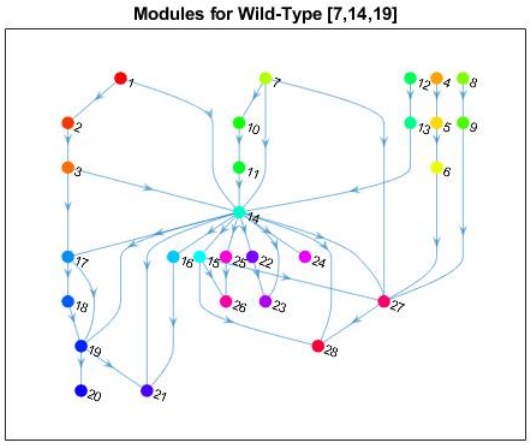



(a) Total module counts

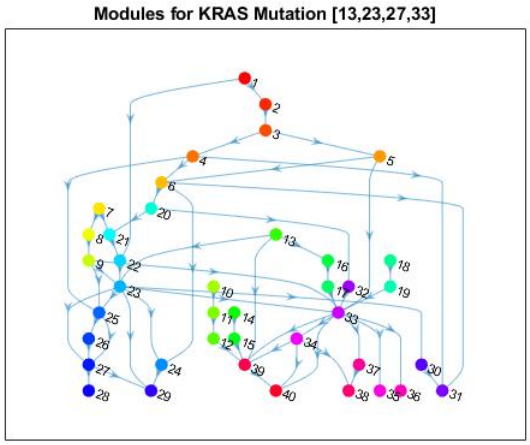
(b) Non-trivial module counts

Figure 11: *Module counts*

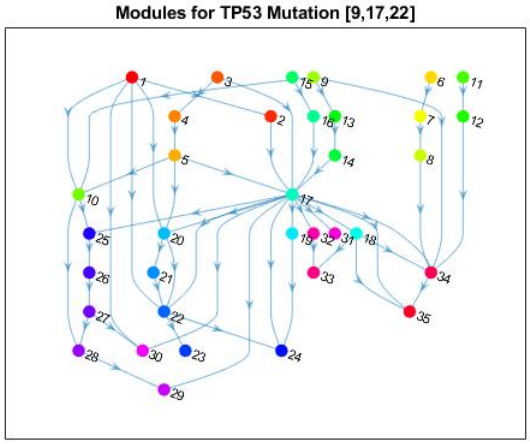
Figure 12: *PC condensation graphs.* Included are all condensation graphs for each mutation combination. These are directed, acyclic graphs that are topologically sorted, and whose nodes represent the strongly connected components of Figure 10. Colors of nodes are based on the components they represent, and node numbers correspond to bin numbers (see supplementary materials through Section 7).



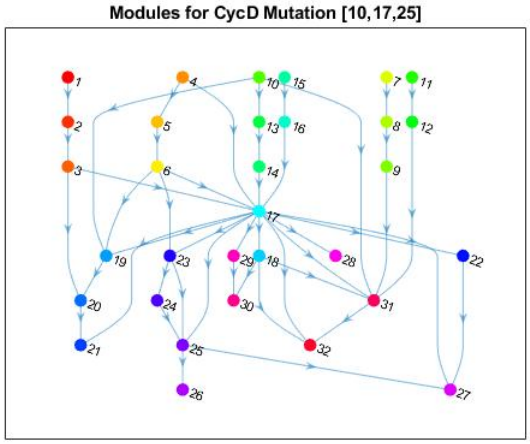

(a) Wild-type condensation graph.



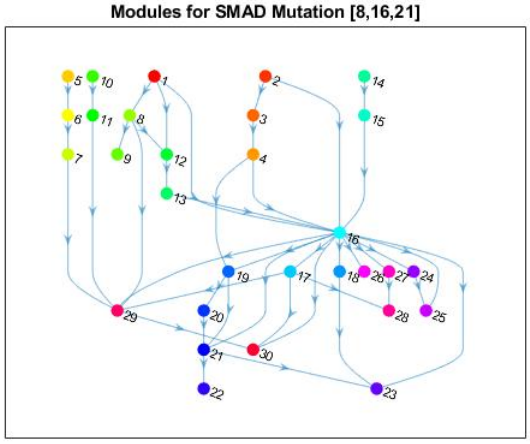

(b) KRAS condensation graph.



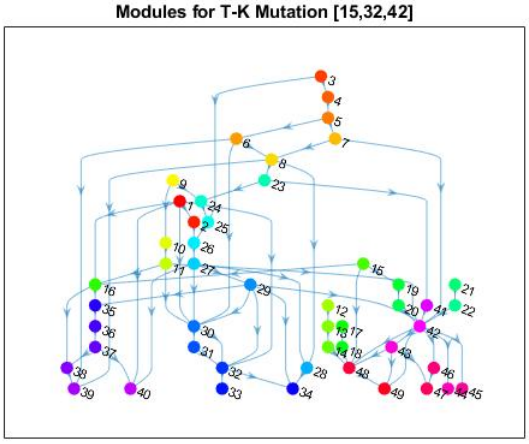

(c) TP53 condensation graph.



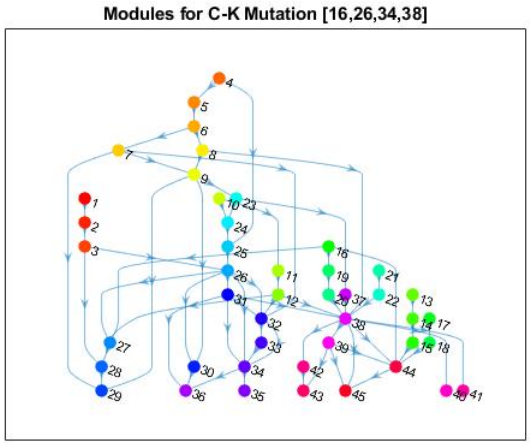

(d) CyclinD condensation graph.



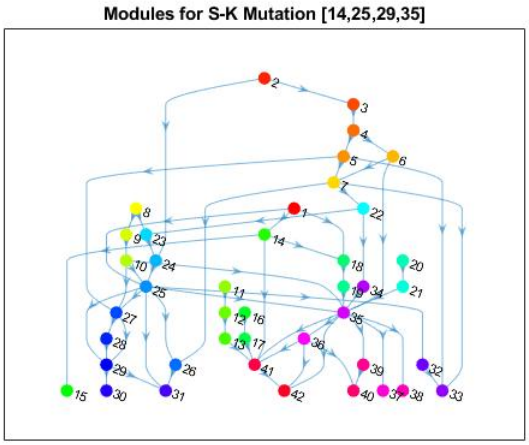

(e) SMAD condensation graph.



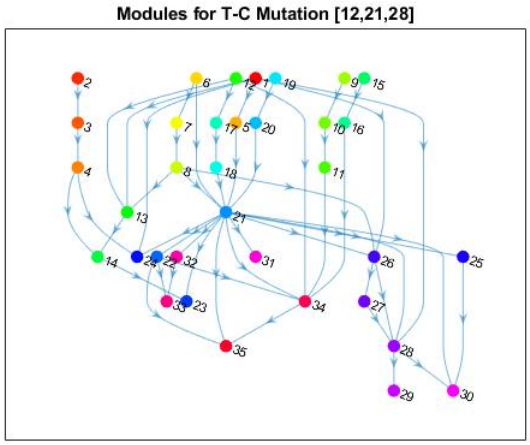

(f) T/K condensation graph.



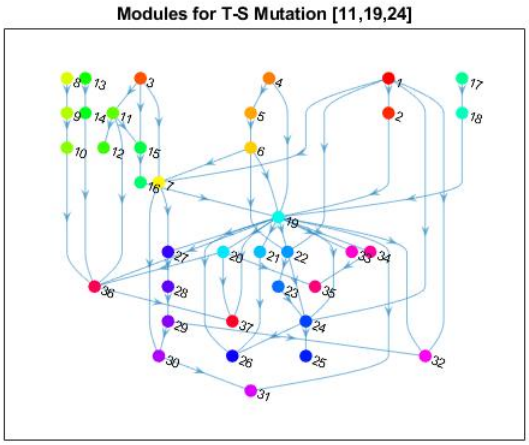

(g) C/K condensation graph.



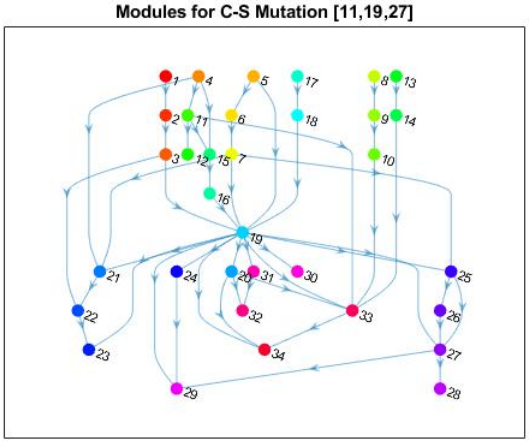

(h) S/K condensation graph.



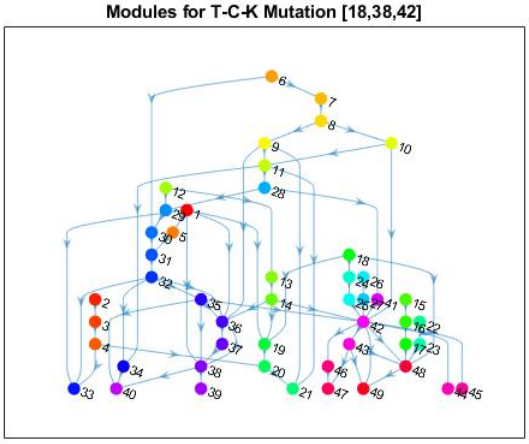

(i) T/C condensation graph.



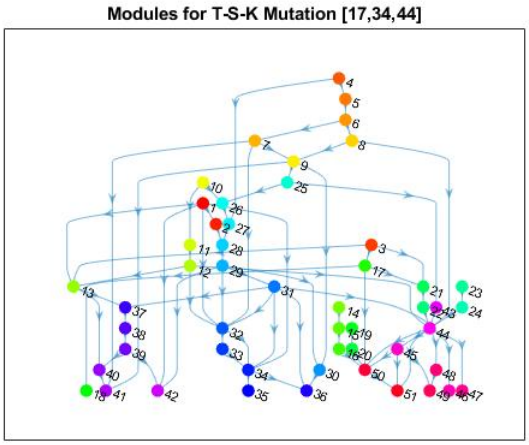

(j) T/S condensation graph.



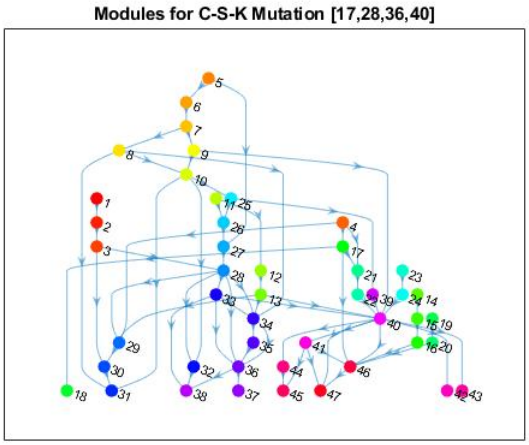

(k) C/S condensation graph.



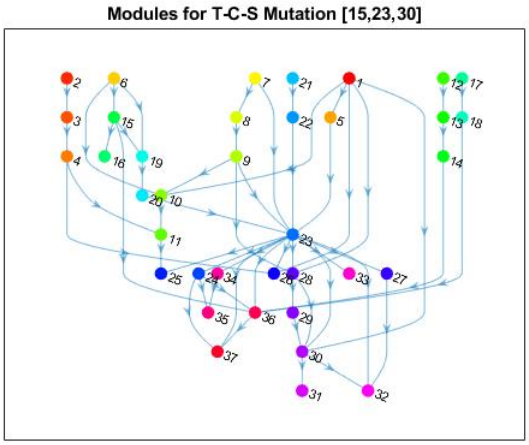

(l) T/C/K condensation graph.



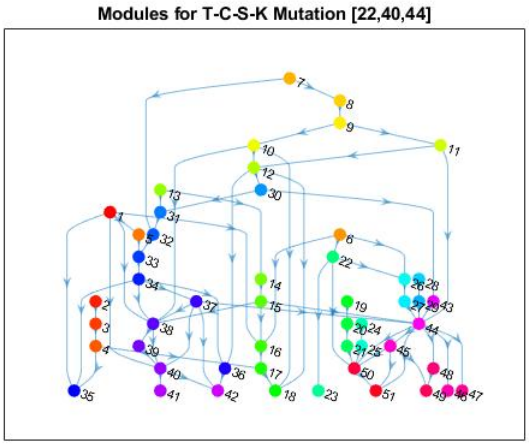

(m) T/S/K condensation graph.



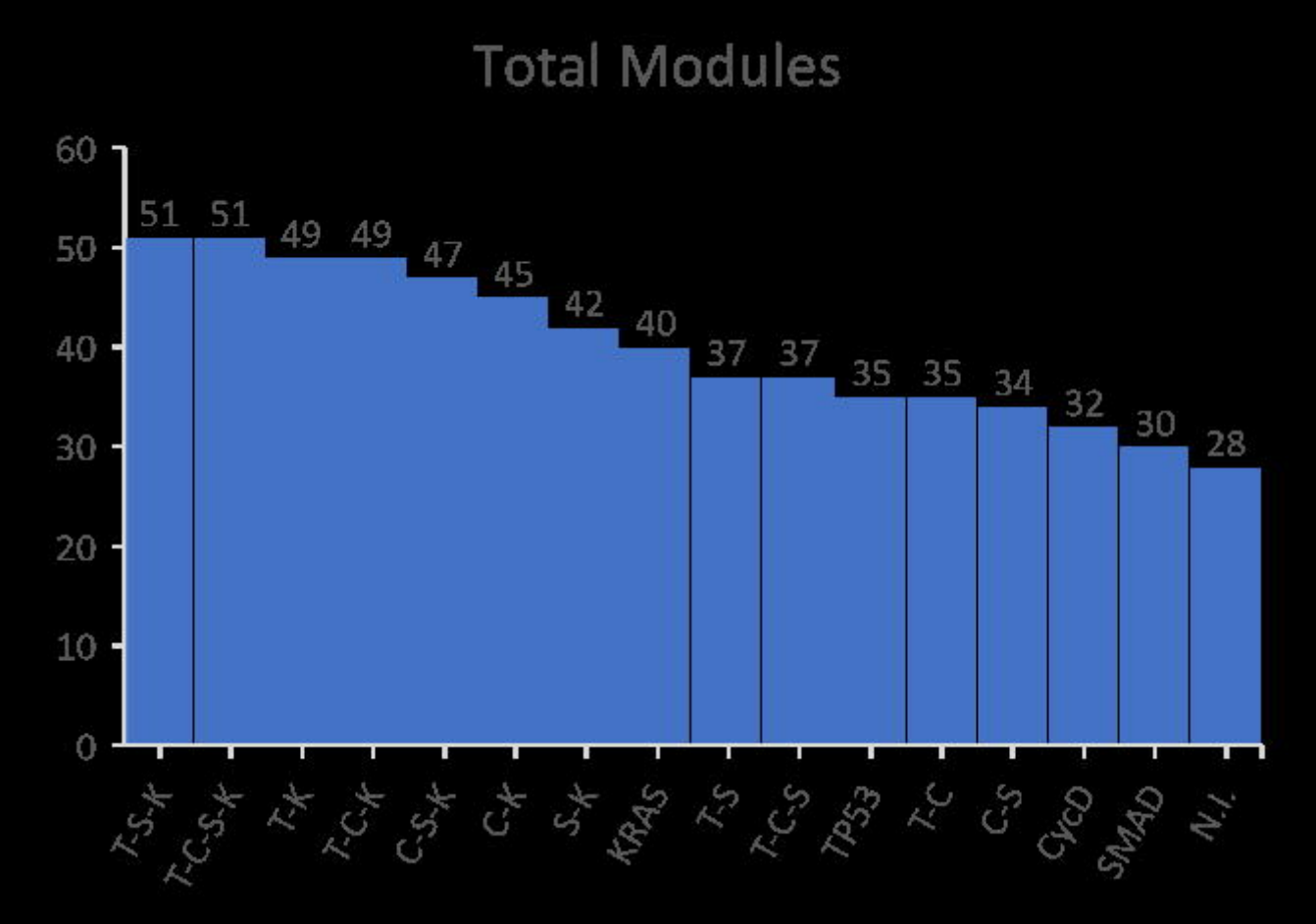

(n) C/S/K condensation graph.

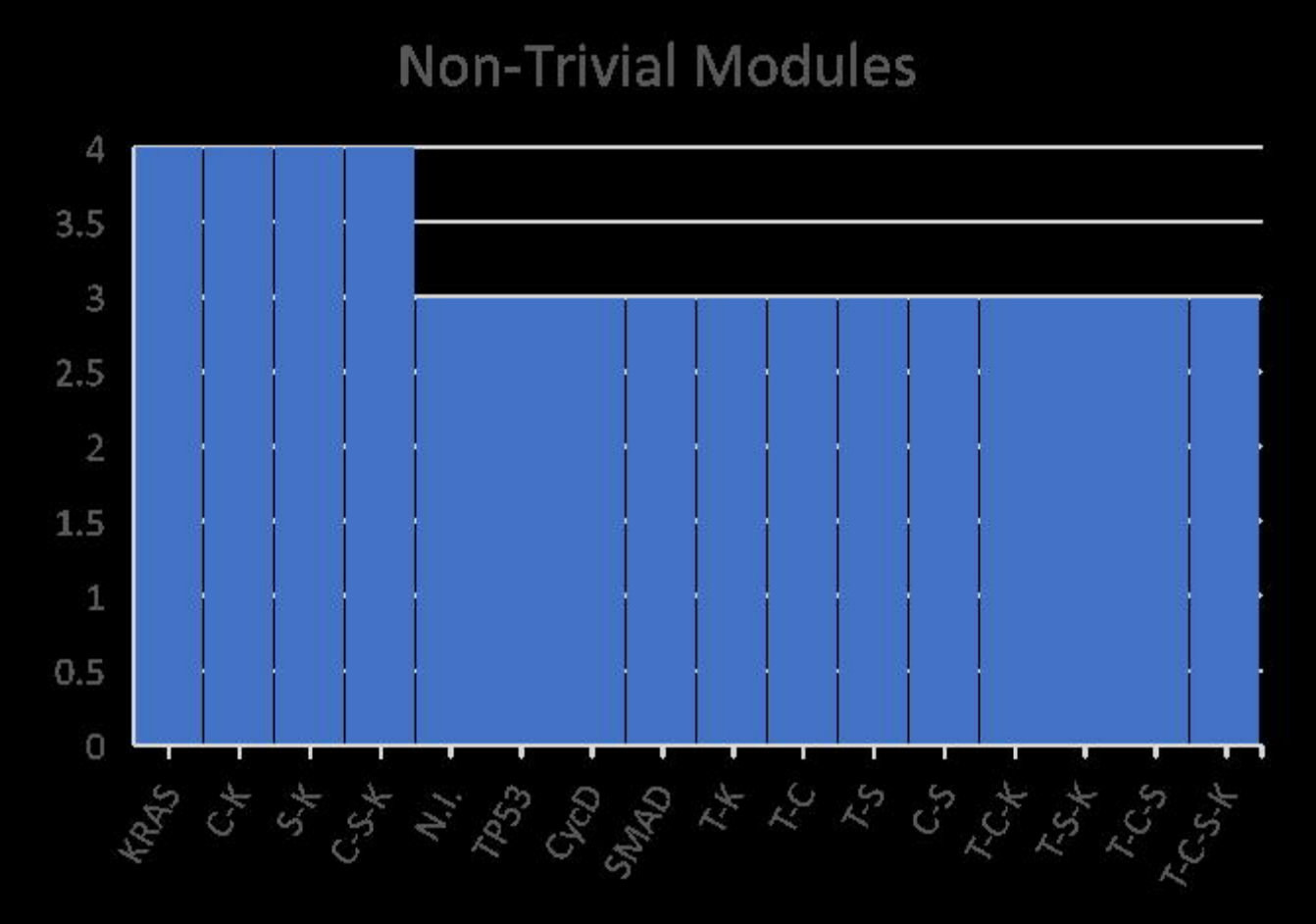


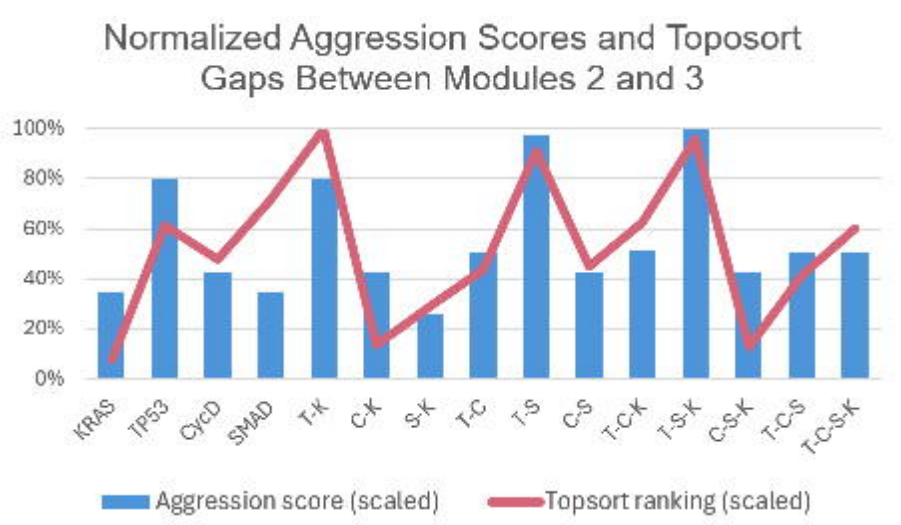

(o) T/C/S condensation graph.

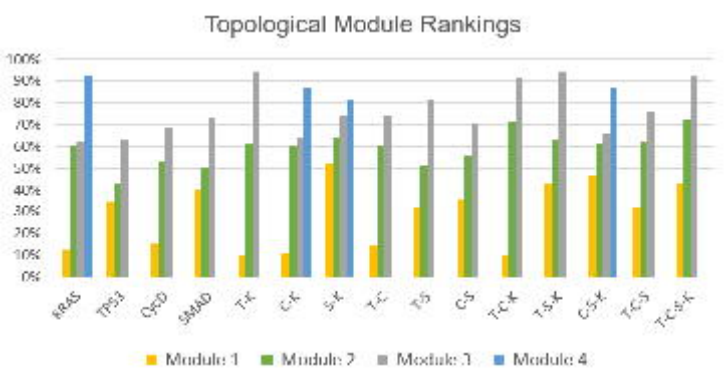



(p) T/C/S/K condensation graph.

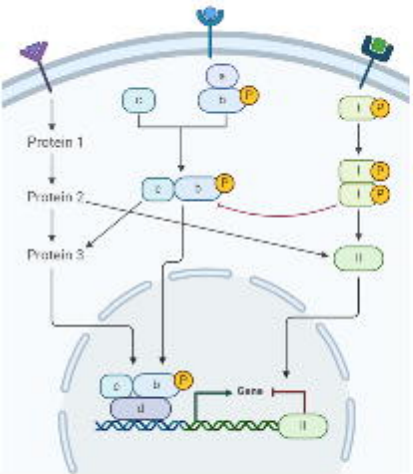

Total Modules
60
50
40
30
20
10
0
51
51
49
49
47
45
42
40
37
37
35
35
34
32
30
28
T-S-K
T-C-S-K
T-K
T-C-K
C-S-K
C-K
S-K
KRAS
T-S
T-C-S
TP53
T-C
C-S
CycD
SMAD
N.I.


Non-Trivial Modules
4
3.5
3
2.5
2
1.5
1
0.5
0
KRAS
C-K
S-K
C-S-K
N.I.
TP53
CycD
SMAD
T-K
T-C
T-S
C-S
T-C-K
T-S-K
T-C-S
T-C-S-K

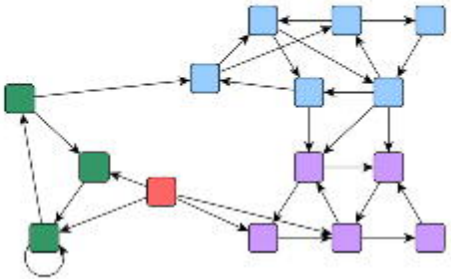

Normalized Aggression Scores and Toposort Gaps Between Modules 2 and 3
100%
80%
60%
40%
20%
0%
KRAS
TP53
CycD
SMAD
T-K
C-K
S-K
T-C
T-S
C-S
T-C-K
T-S-K
C-S-K
T-C-S
T-C-S-K
Aggression score (scaled)
Topsort ranking (scaled)

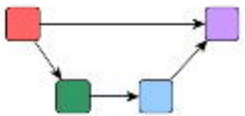

Topological Module Rankings
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
RAS
TP53
CyCD
SMAD
T-K
C-K
S-K
T-C
T-S
C-S
T-C-K
T-S-K
C-S-K
T-C-S
T-C-S-K
Module 1
Module 2
Module 3
Module 4

## Network Analysis

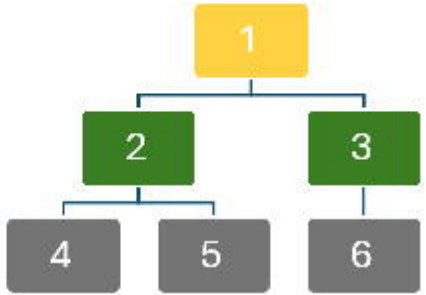



## Determine Modules

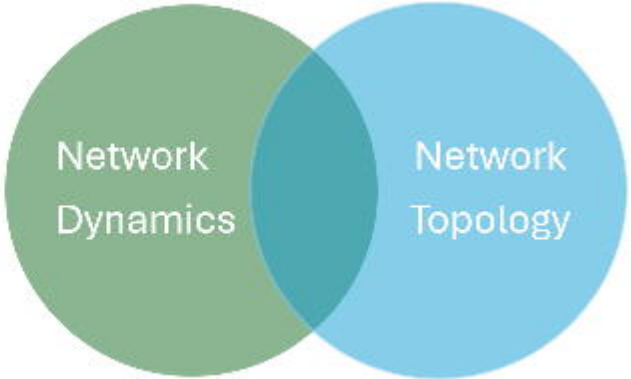




## Rank Modules



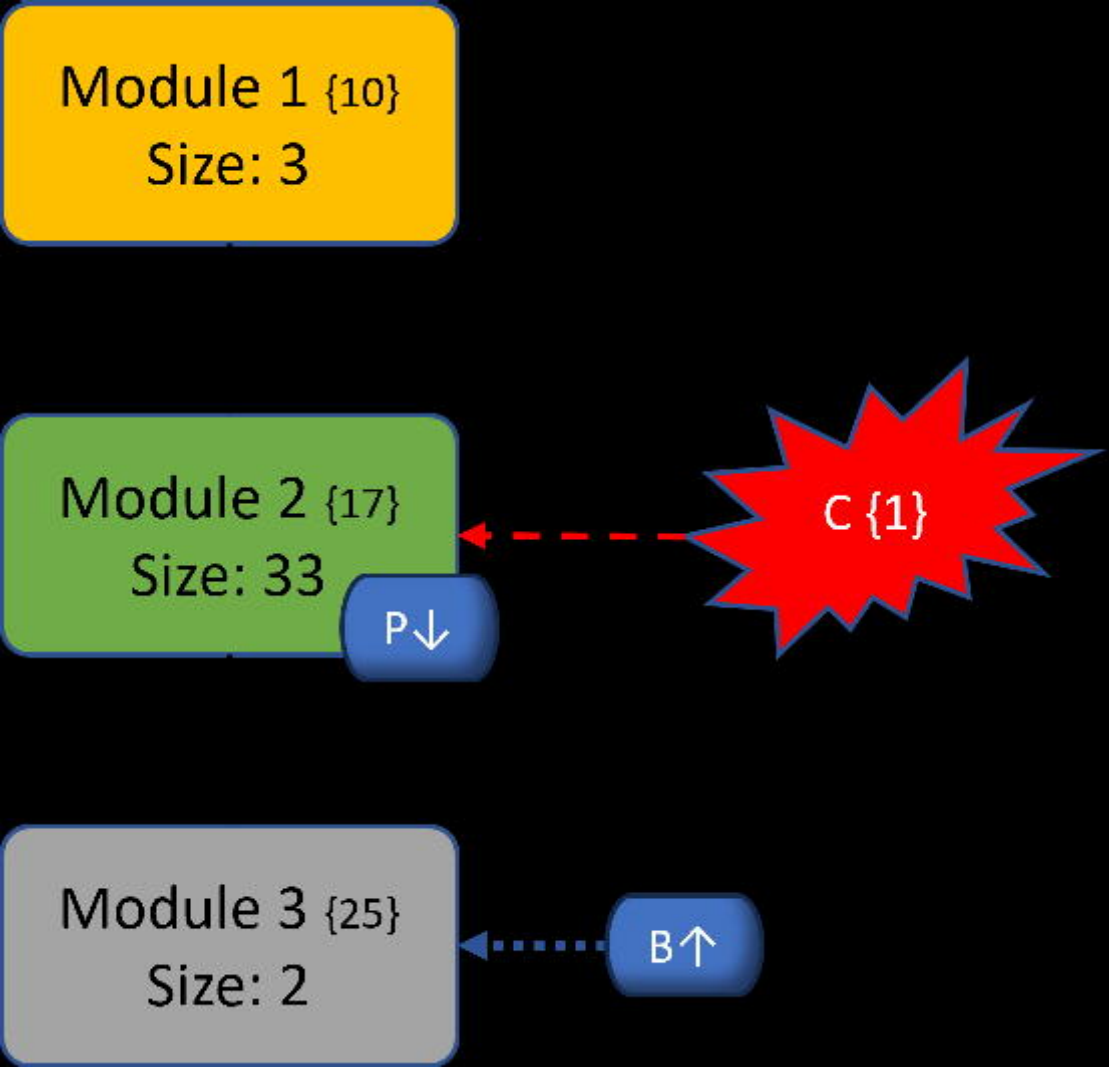

## Comparative Analysis

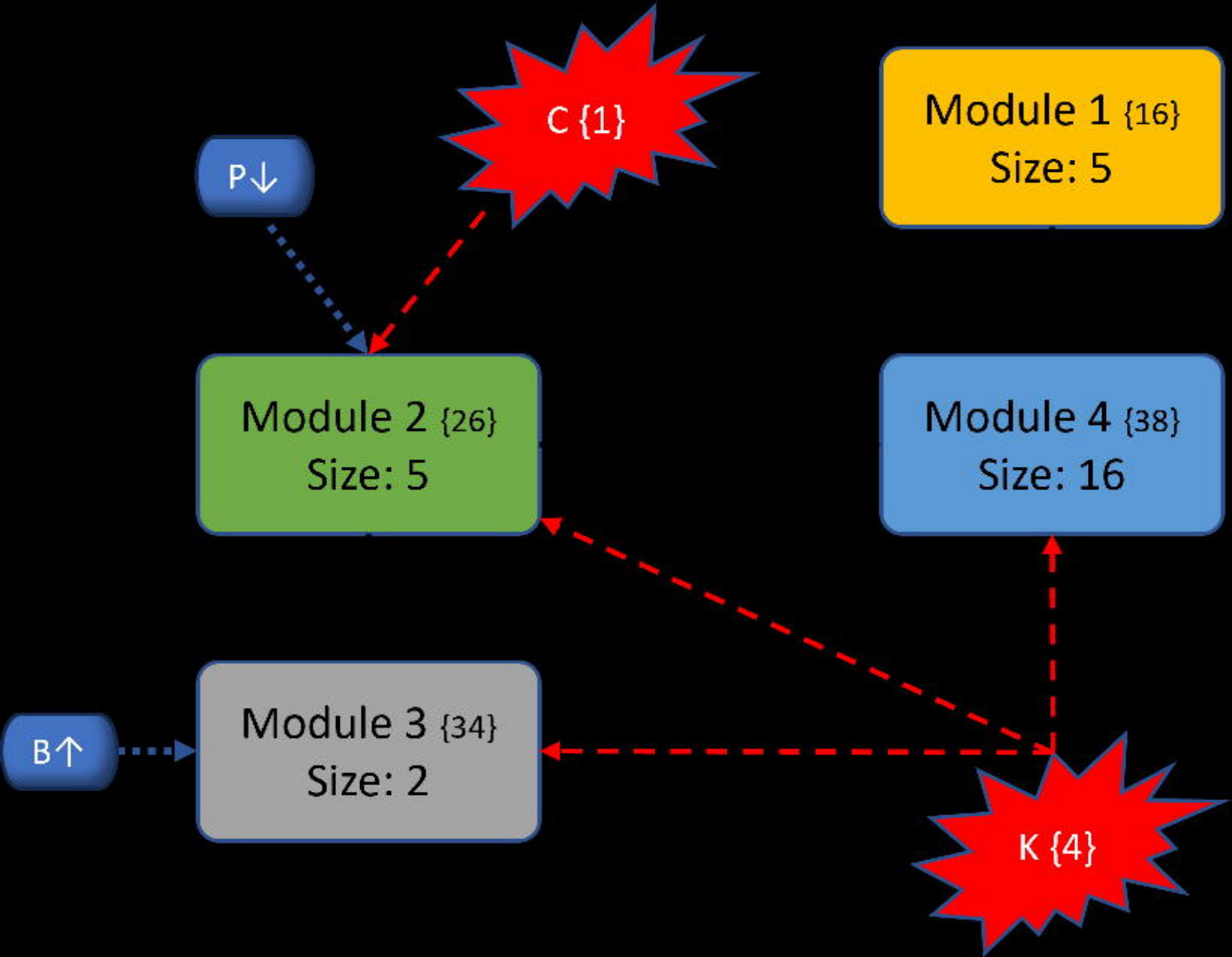


Errors of Normalized Toposort Gap Rankings

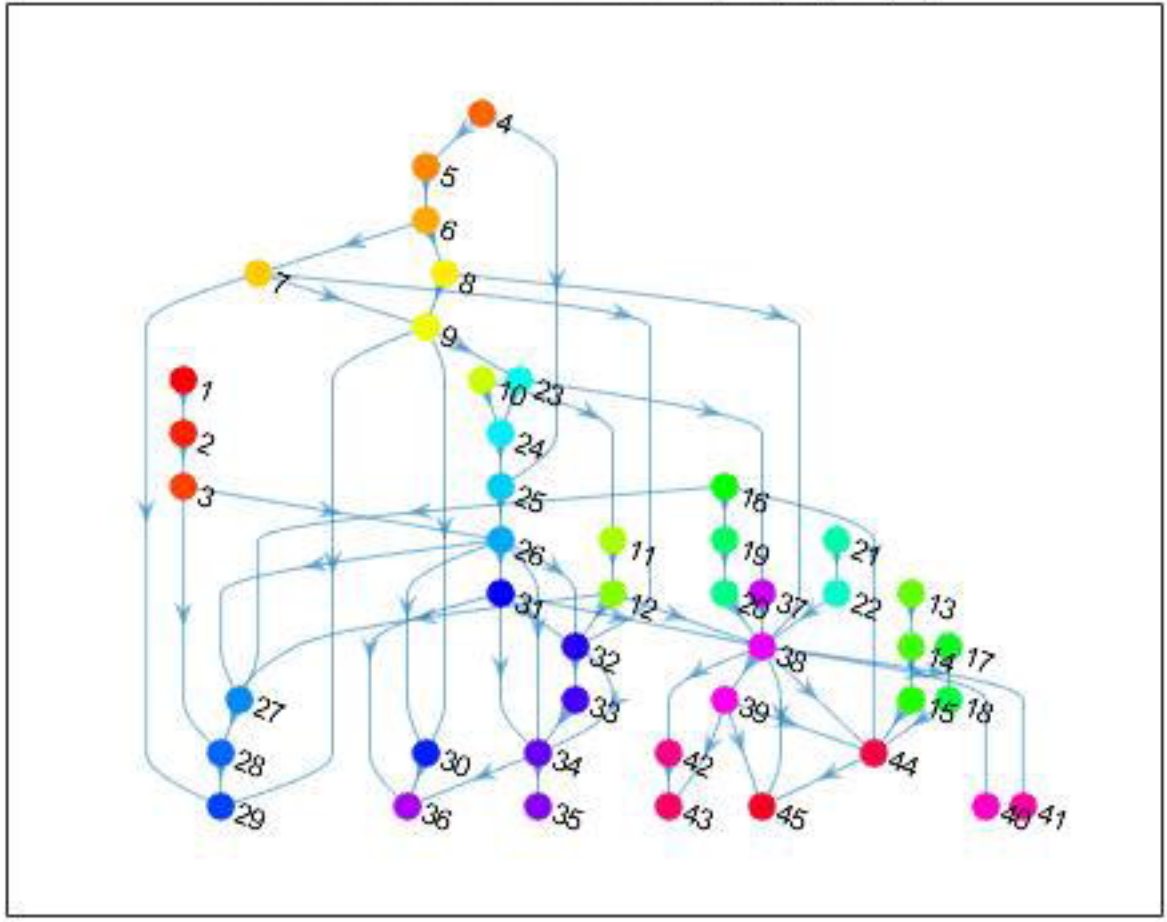
## Uncontrolled Phenotype Aggression Scores

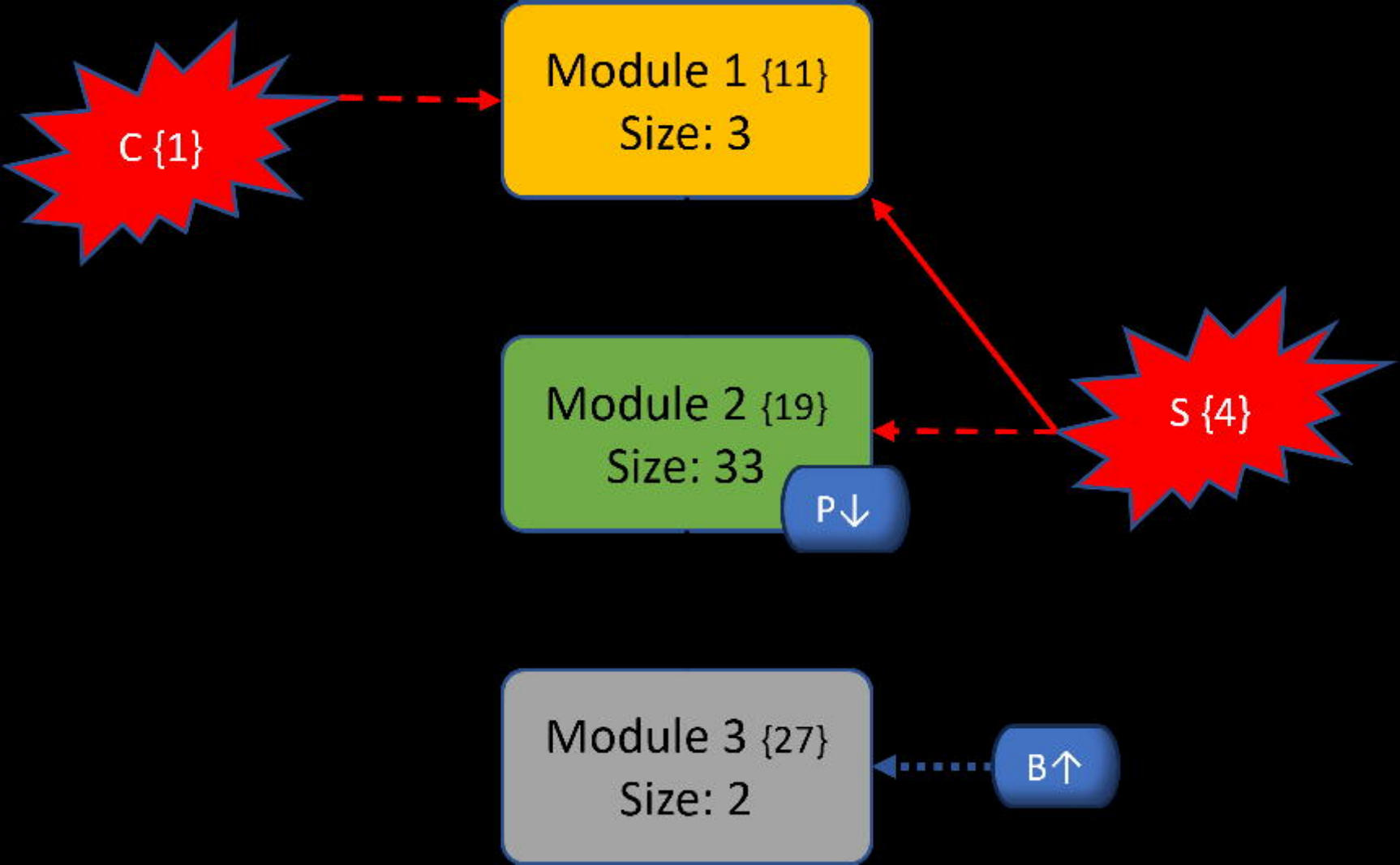
| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1.09 | 1.12 | 3.03 | 1.56 | 1.14 | 3.05 | 1.56 | 0.71 | 1.92 | 3.72 | 1.55 | 1.95 | 3.81 | 1.58 | 1.92 | 1.93 |
| 5.16 | 5.37 | 10.74 | 8.11 | 5.36 | 10.84 | 8.18 | 3.56 | 9.54 | 11.36 | 8.09 | 9.71 | 11.55 | 8.14 | 9.61 | 9.62 |
| 2.79 | 2.80 | 7.51 | 1.70 | 2.82 | 7.49 | 1.67 | 2.63 | 2.06 | 11.08 | 1.65 | 2.07 | 11.36 | 1.72 | 2.00 | 2.05 |
| 3.01 | 3.10 | 7.09 | 3.79 | 3.11 | 7.12 | 3.80 | 2.30 | 4.51 | 8.72 | 3.76 | 4.58 | 8.91 | 3.81 | 4.51 | 4.53 |
| **PI3K↓** | | | | | | | | | | | | | | | |
| -0.80 | -0.78 | 0.08 | -0.87 | -0.82 | 0.07 | -0.84 | -0.78 | 0.04 | 0.09 | -0.83 | 0.06 | 0.07 | -0.83 | 0.04 | 0.04 |
| -0.68 | -0.60 | 0.28 | -0.78 | -0.67 | 0.20 | -0.70 | -0.63 | 0.18 | 0.21 | -0.69 | 0.22 | 0.22 | -0.71 | 0.15 | 0.18 |
| -0.60 | -0.51 | 0.24 | -0.76 | -0.65 | 0.30 | -0.74 | -0.47 | 0.16 | 0.41 | -0.71 | 0.21 | 0.31 | -0.71 | 0.20 | 0.15 |
| -0.70 | -0.63 | 0.20 | -0.80 | -0.71 | 0.19 | -0.76 | -0.62 | 0.12 | 0.24 | -0.75 | 0.16 | 0.20 | -0.75 | 0.13 | 0.13 |
| **BAX↑** | | | | | | | | | | | | | | | |
| 1.06 | 1.11 | 2.98 | 1.51 | 0.69 | 3.08 | 1.58 | 0.60 | 1.94 | 3.74 | 1.53 | 1.95 | 3.75 | 1.58 | 1.94 | 1.92 |
| 5.23 | 5.36 | 10.70 | 8.04 | 3.60 | 10.85 | 8.26 | 3.34 | 9.70 | 11.43 | 8.13 | 9.70 | 11.44 | 8.28 | 9.73 | 9.60 |
| 2.74 | 2.85 | 7.33 | 1.64 | 2.74 | 7.77 | 1.71 | 2.51 | 2.06 | 11.16 | 1.62 | 2.09 | 11.24 | 1.73 | 2.02 | 2.11 |
| 3.01 | 3.11 | 7.00 | 3.73 | 2.34 | 7.23 | 3.85 | 2.15 | 4.57 | 8.78 | 3.76 | 4.58 | 8.81 | 3.86 | 4.57 | 4.55 |
| **PI3K↓ / BAX↑** | | | | | | | | | | | | | | | |
| -0.86 | -0.86 | -0.88 | -0.90 | -0.86 | -0.91 | -0.88 | -0.91 | -0.91 | -0.88 | -0.87 | -0.89 | -0.87 | -0.89 | -0.90 | -0.91 |
| -0.65 | -0.65 | -0.75 | -0.68 | -0.60 | -0.82 | -0.66 | -0.76 | -0.75 | -0.72 | -0.63 | -0.73 | -0.69 | -0.70 | -0.80 | -0.76 |
| -0.65 | -0.62 | -0.67 | -0.80 | -0.66 | -0.76 | -0.75 | -0.76 | -0.80 | -0.65 | -0.71 | -0.73 | -0.64 | -0.76 | -0.74 | -0.82 |
| -0.72 | -0.71 | -0.77 | -0.80 | -0.71 | -0.83 | -0.76 | -0.81 | -0.82 | -0.75 | -0.74 | -0.78 | -0.73 | -0.78 | -0.82 | -0.83 |


Module 1 {10}
Size: 3
Module 2 {17}
Size: 33
P↓
C {1}
Module 3 {25}
Size: 2
B↑

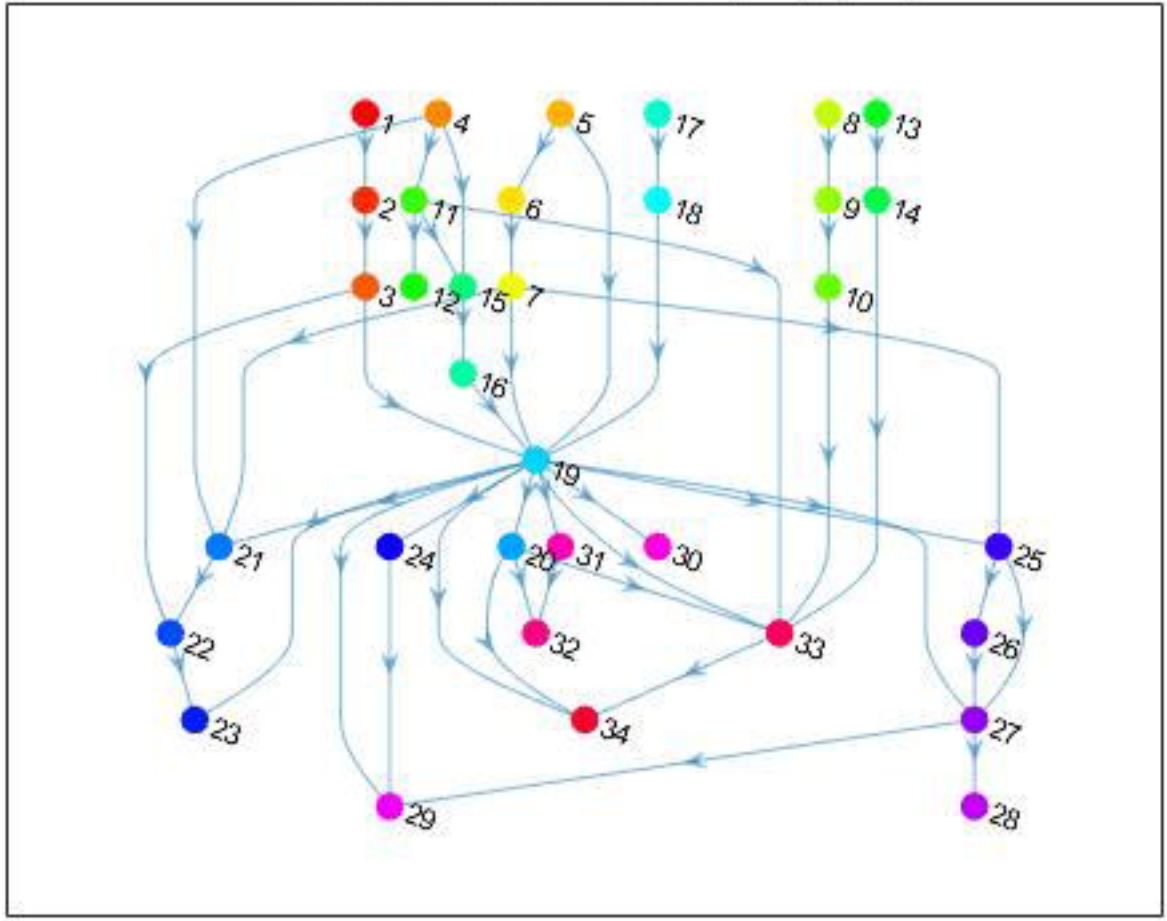

C {1}
Module 1 {16}
Size: 5
P↓
Module 2 {26}
Size: 5
Module 4 {38}
Size: 16
B↑
Module 3 {34}
Size: 2
K {4}

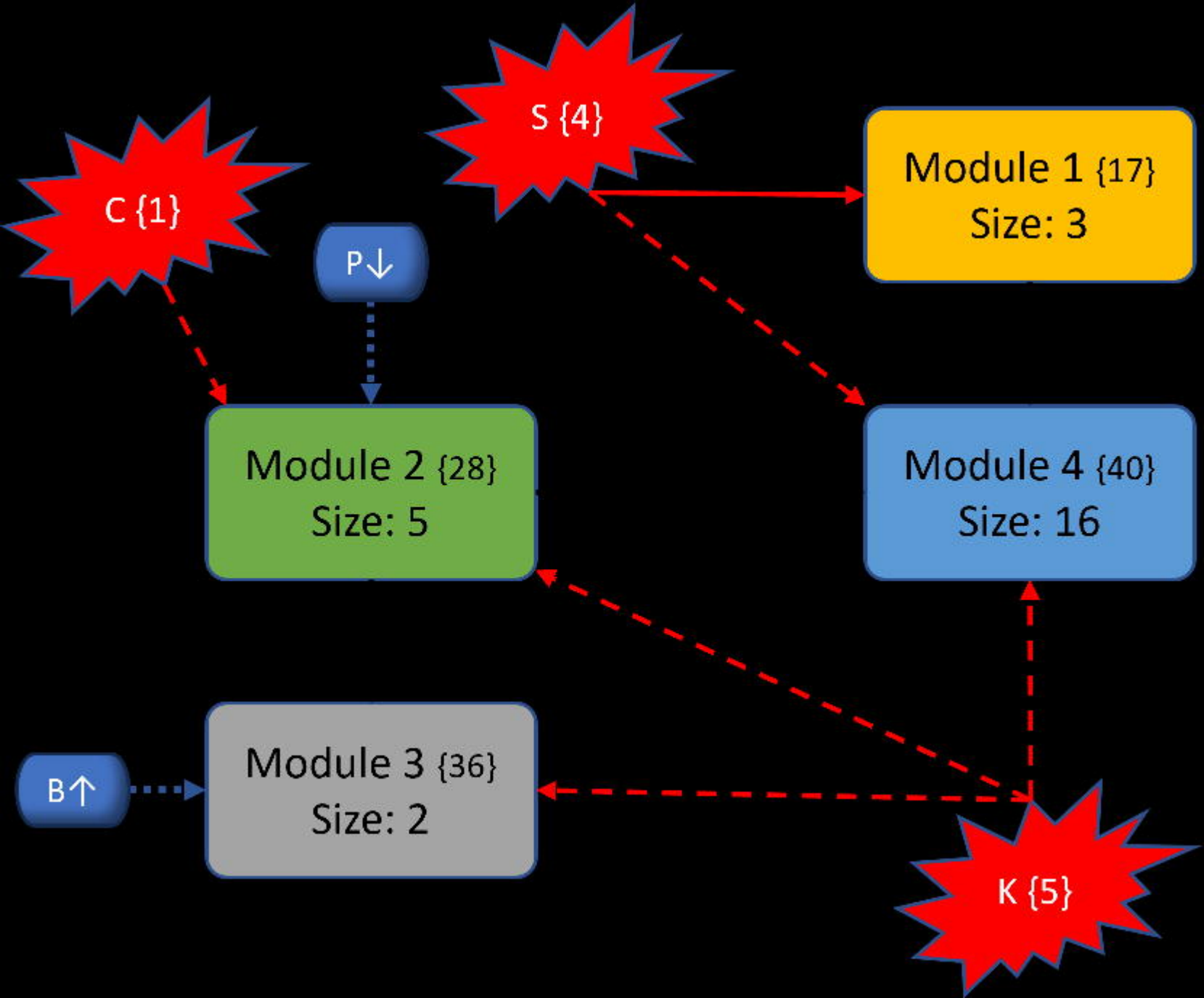
# Modules for C-K Mutation [16,26,34,38]



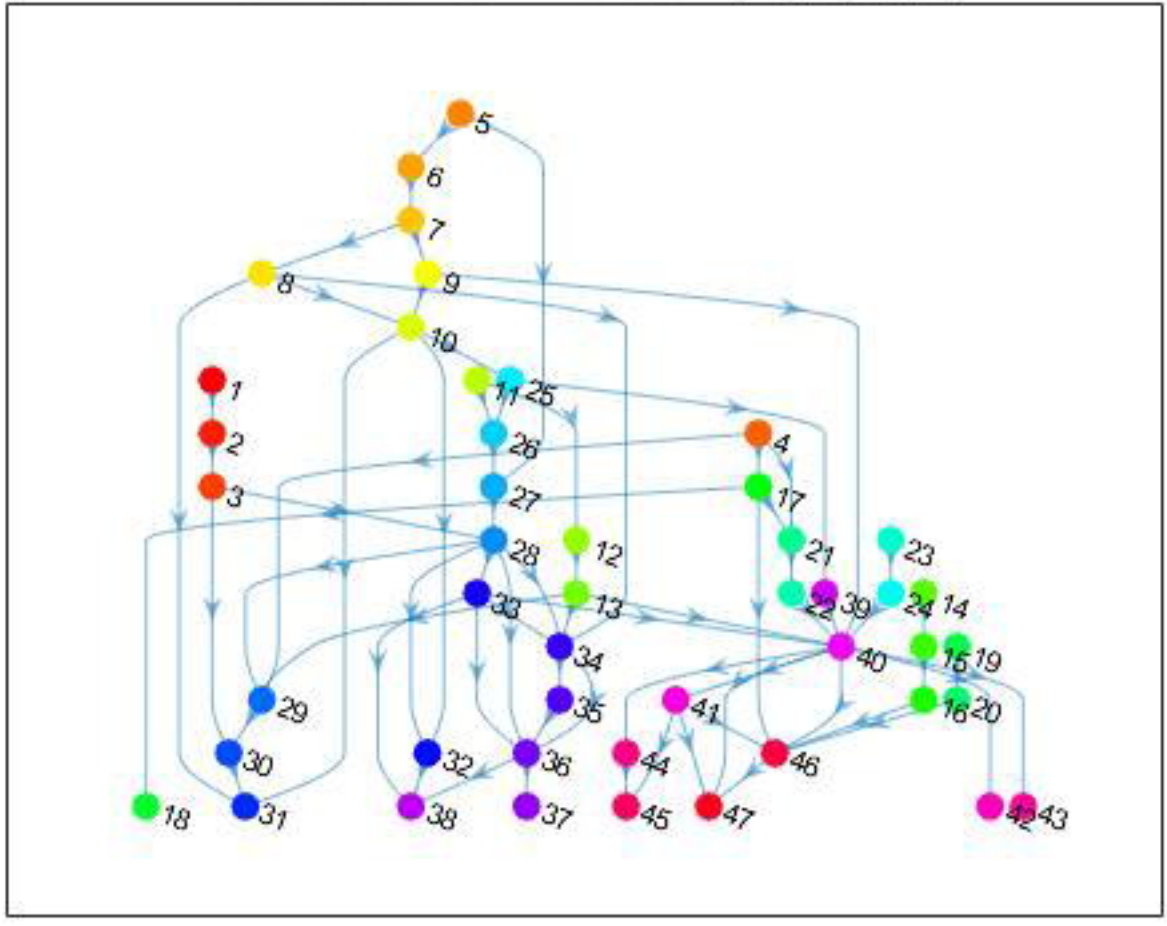

C {1}
Module 1 {11}
Size: 3
Module 2 {19}
Size: 33
P↓
S {4}
Module 3 {27}
Size: 2
B↑

**Modules for C-S Mutation [11,19,27]**

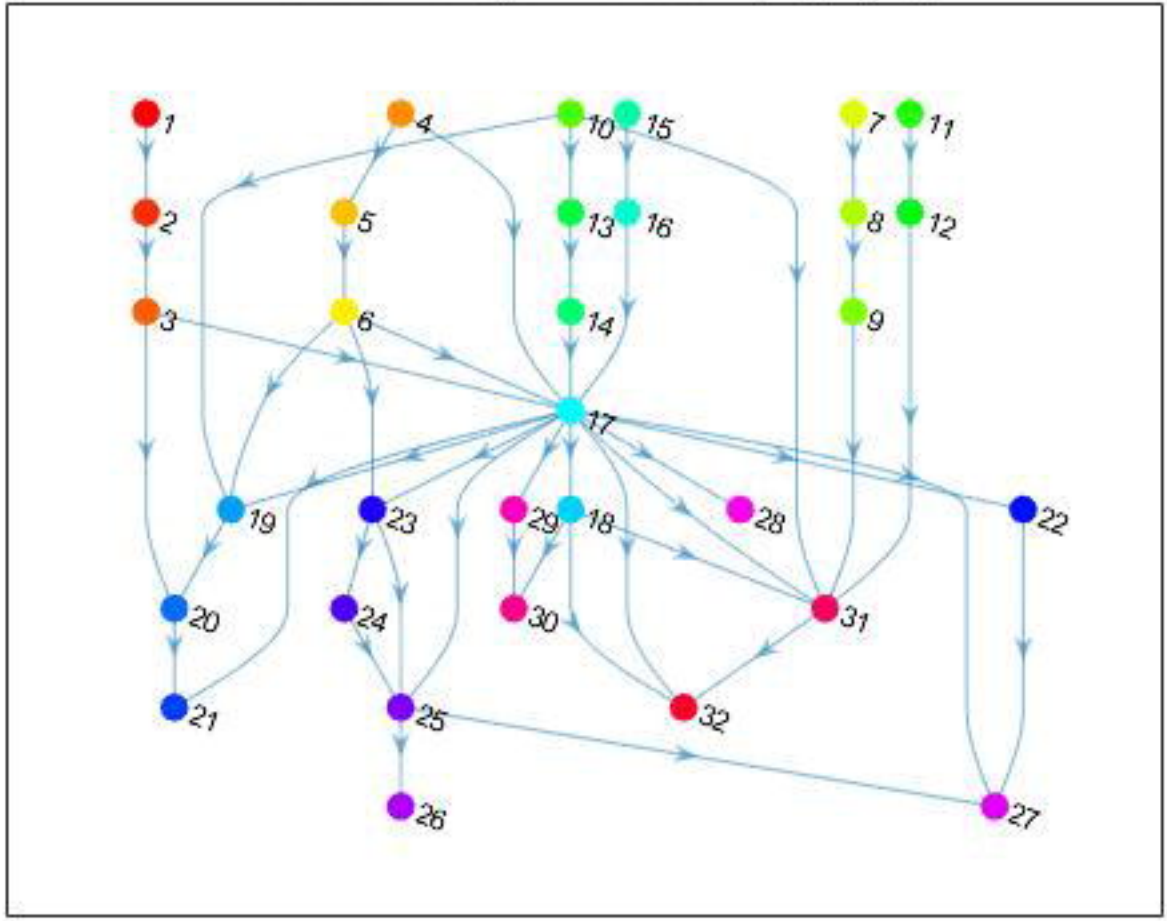


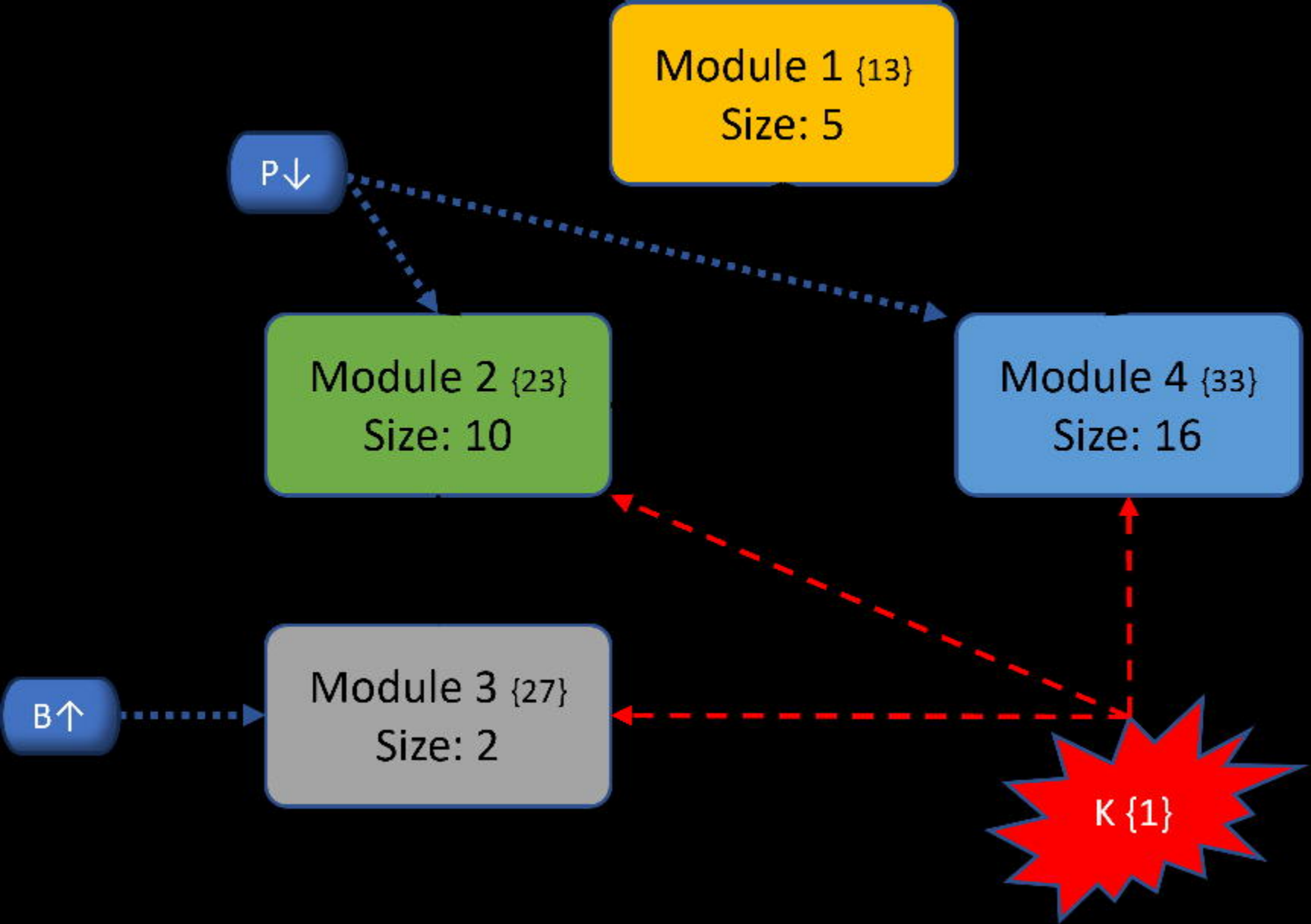

S {4}
C {1}
P↓
Module 1 {17}
Size: 3
Module 2 {28}
Size: 5
Module 4 {40}
Size: 16
B↑
Module 3 {36}
Size: 2
K {5}

Modules for C-S-K Mutation [17,28,36,40]

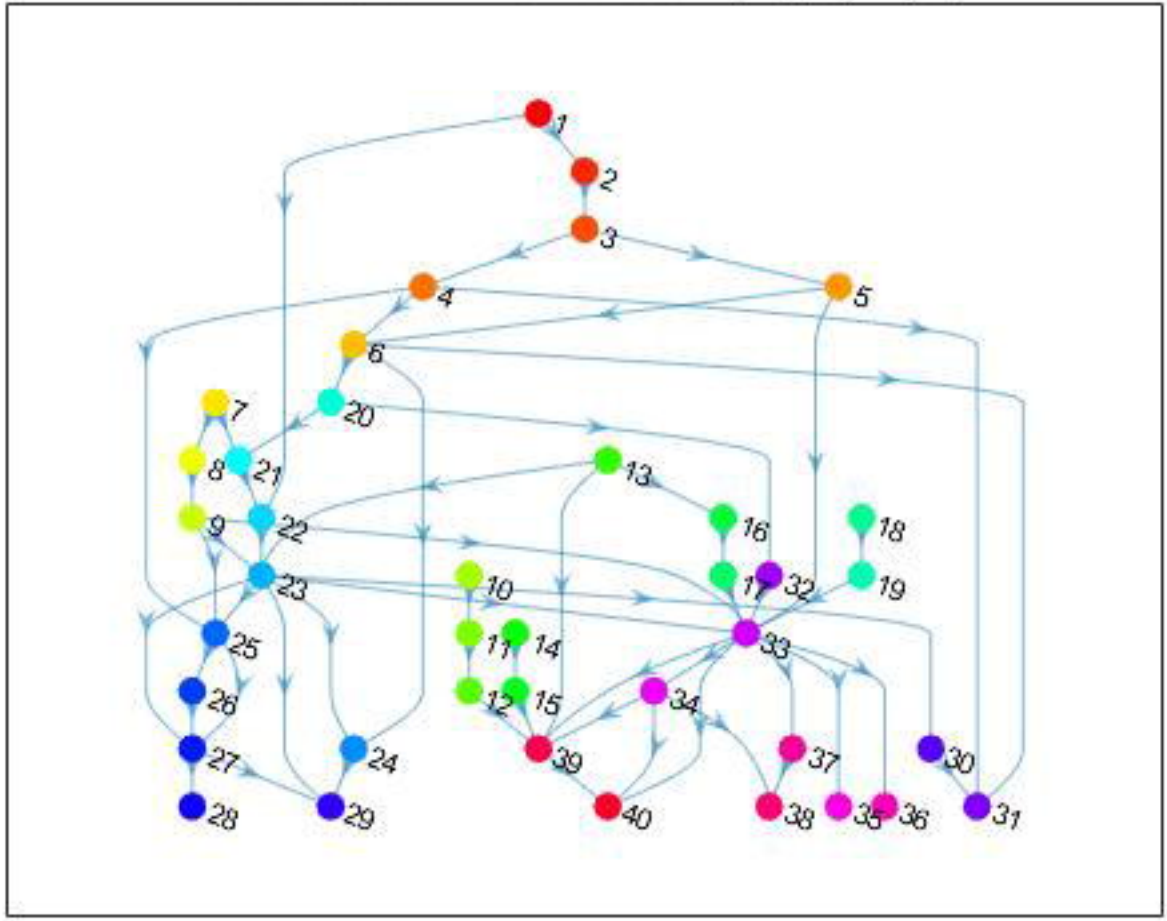


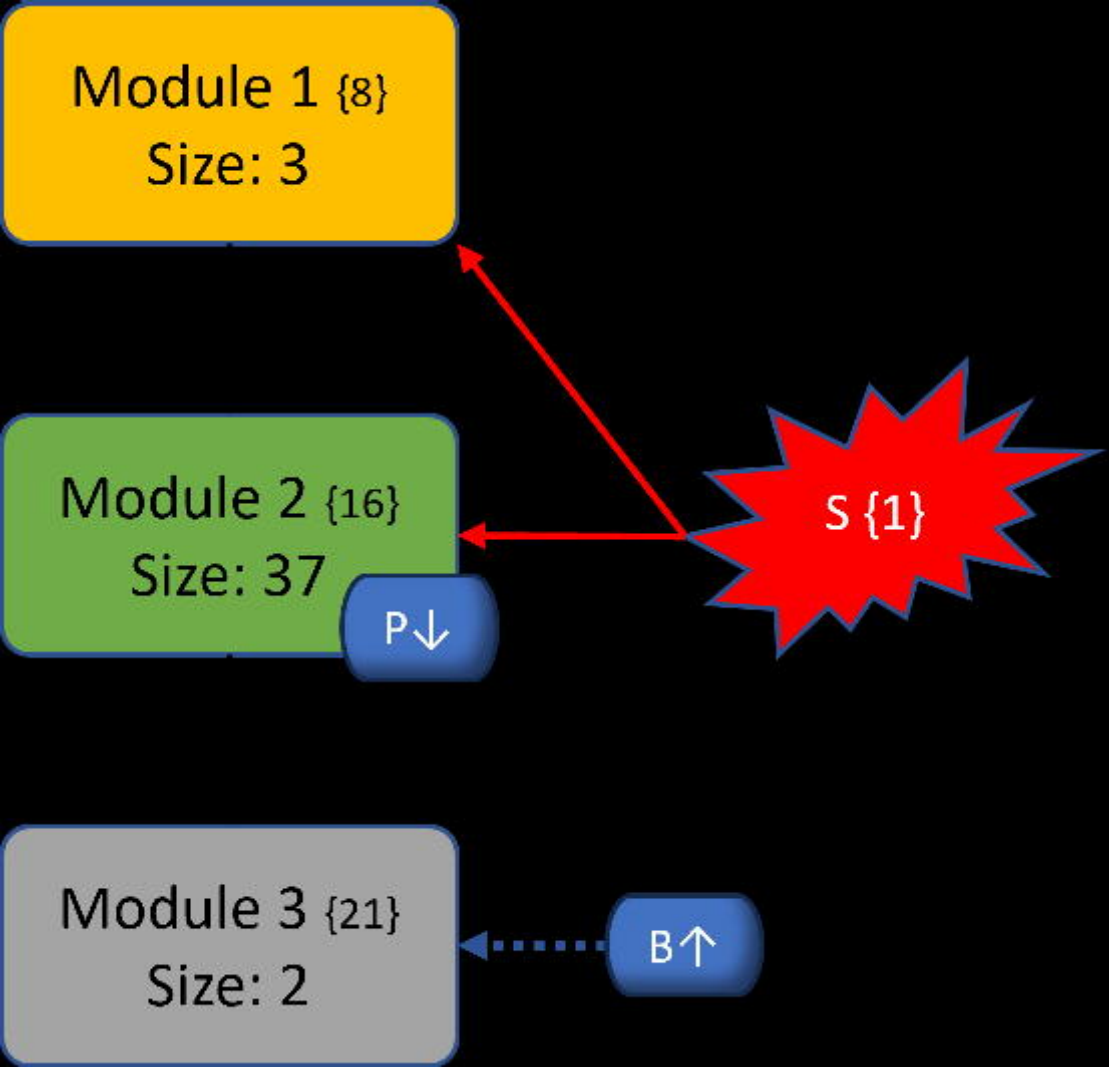
# Modules for CycD Mutation [10,17,25]

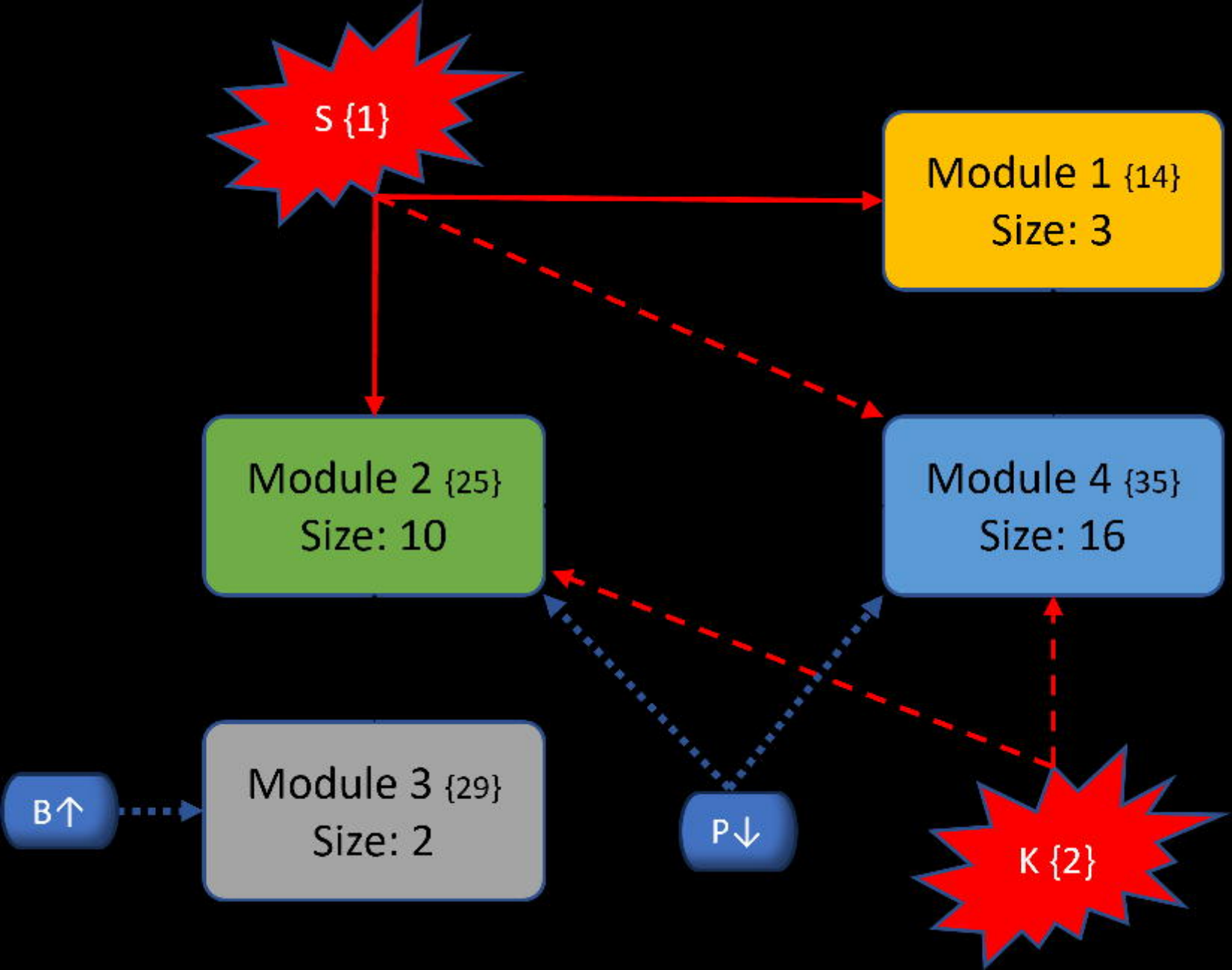



Module 1 {13}
Size: 5
P↓
Module 2 {23}
Size: 10
Module 4 {33}
Size: 16
B↑
Module 3 {27}
Size: 2
K {1}

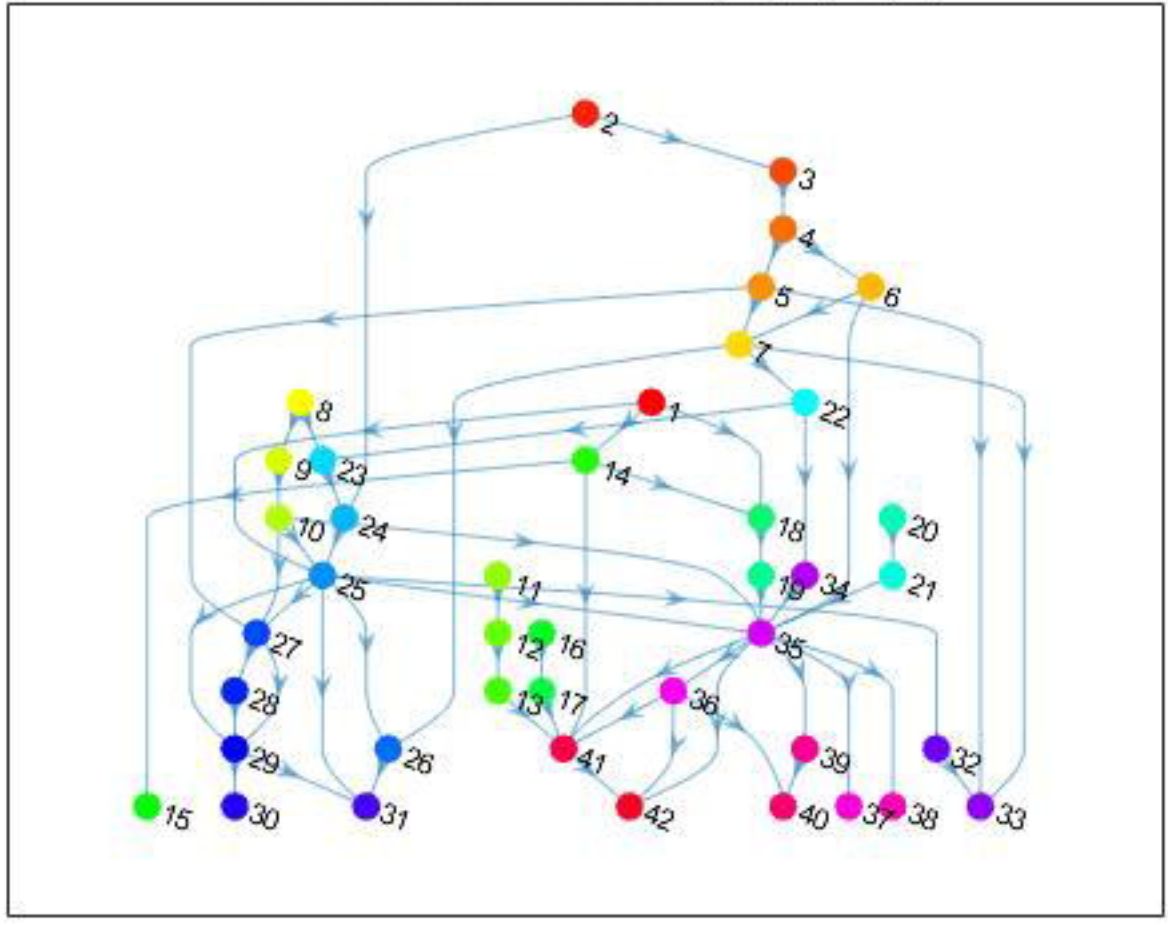
# Modules for KRAS Mutation [13,23,27,33]



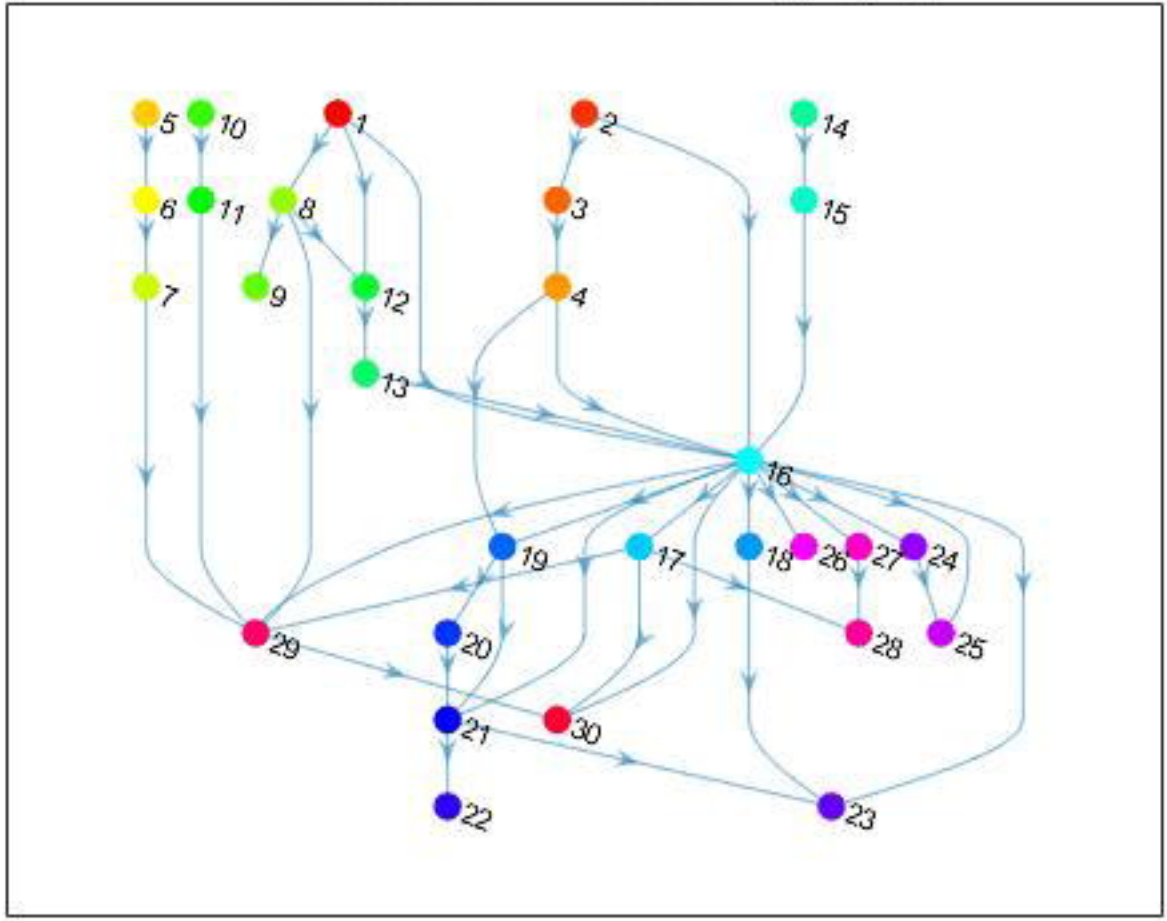

Module 1 {8}
Size: 3
Module 2 {16}
Size: 37
P↓
S {1}
Module 3 {21}
Size: 2
B↑

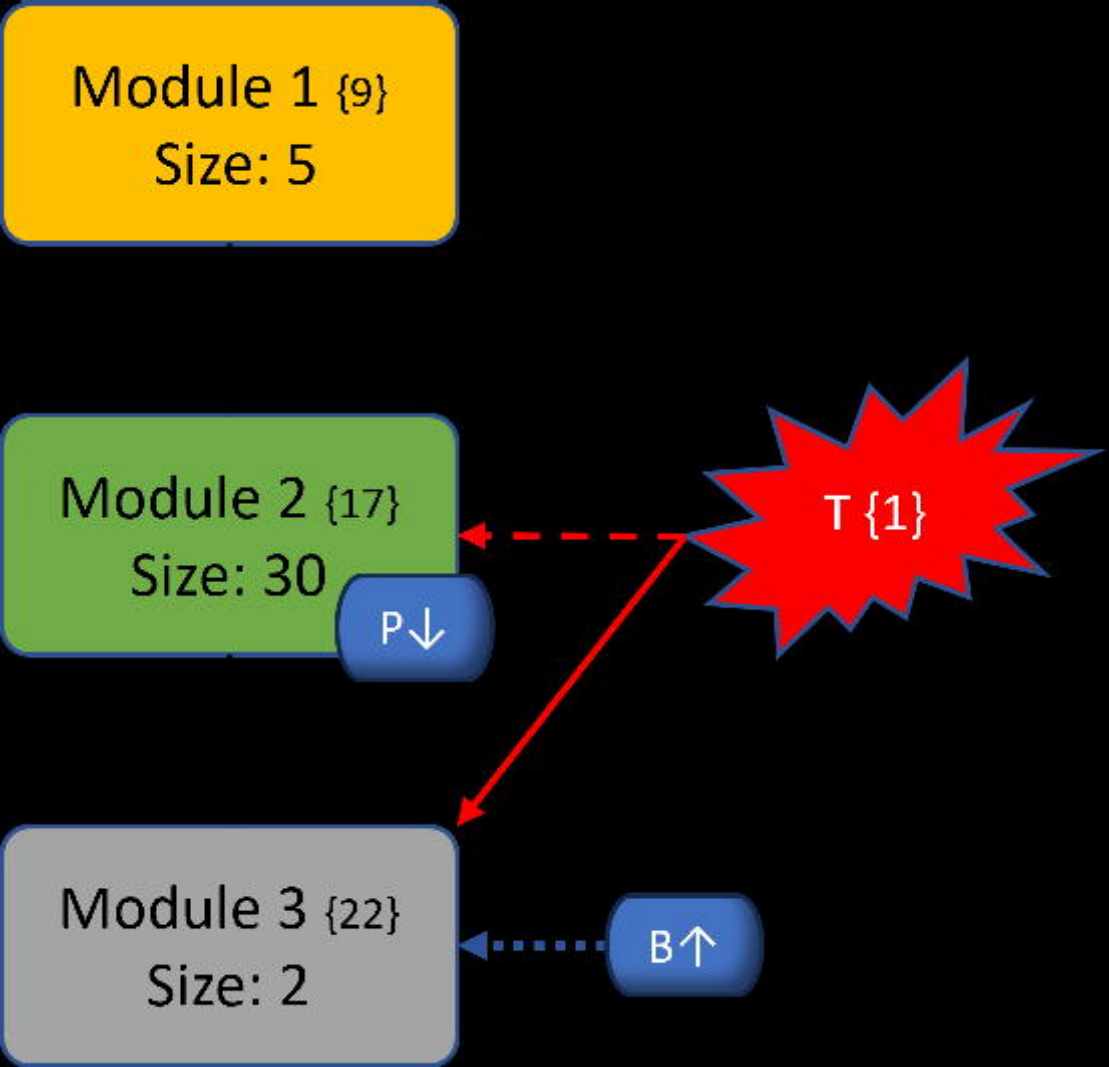

S {1}
Module 1 {14}
Size: 3
Module 2 {25}
Size: 10
Module 4 {35}
Size: 16
Module 3 {29}
Size: 2
B↑
P↓
K {2}

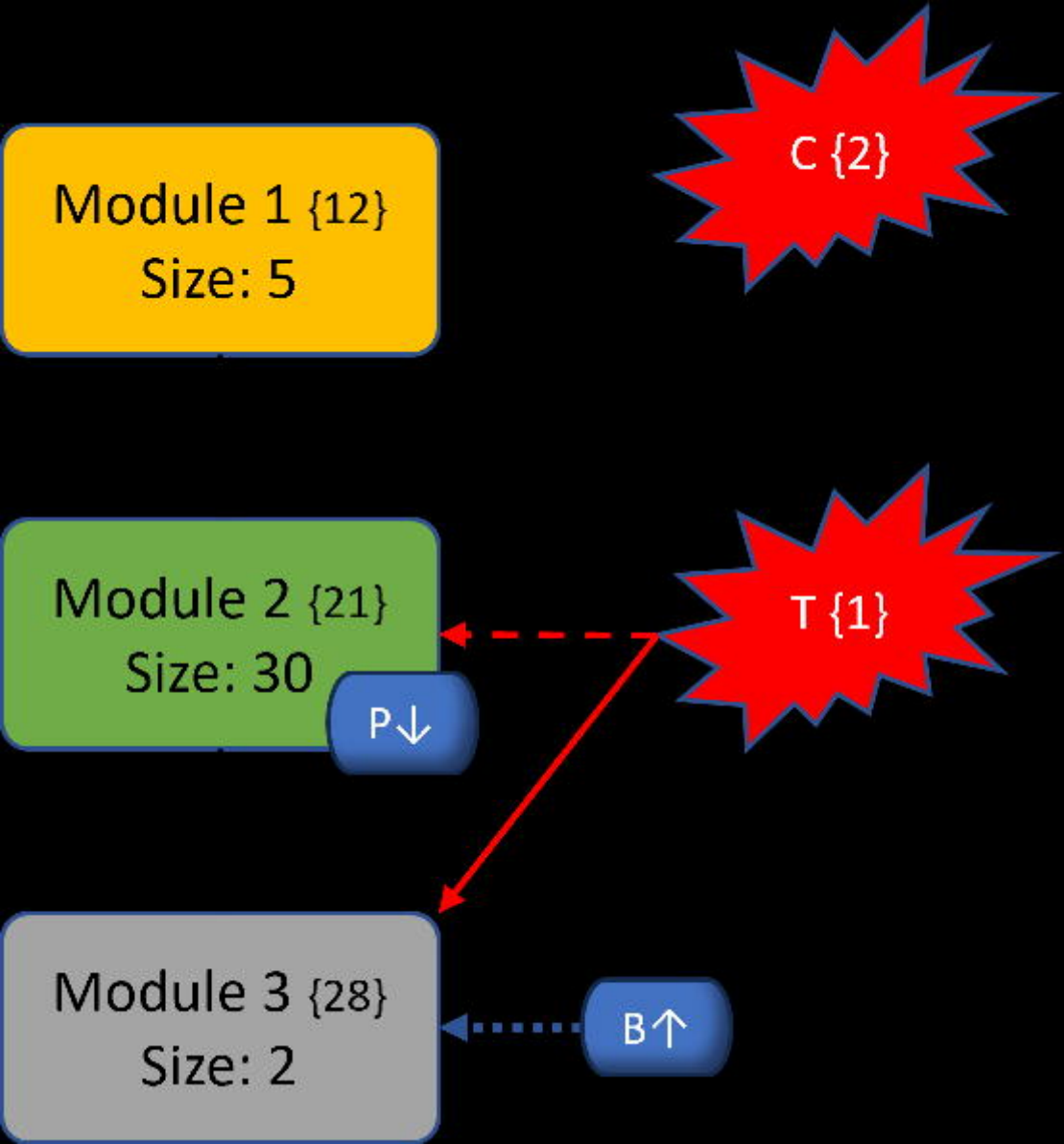
# Modules for S-K Mutation [14,25,29,35]



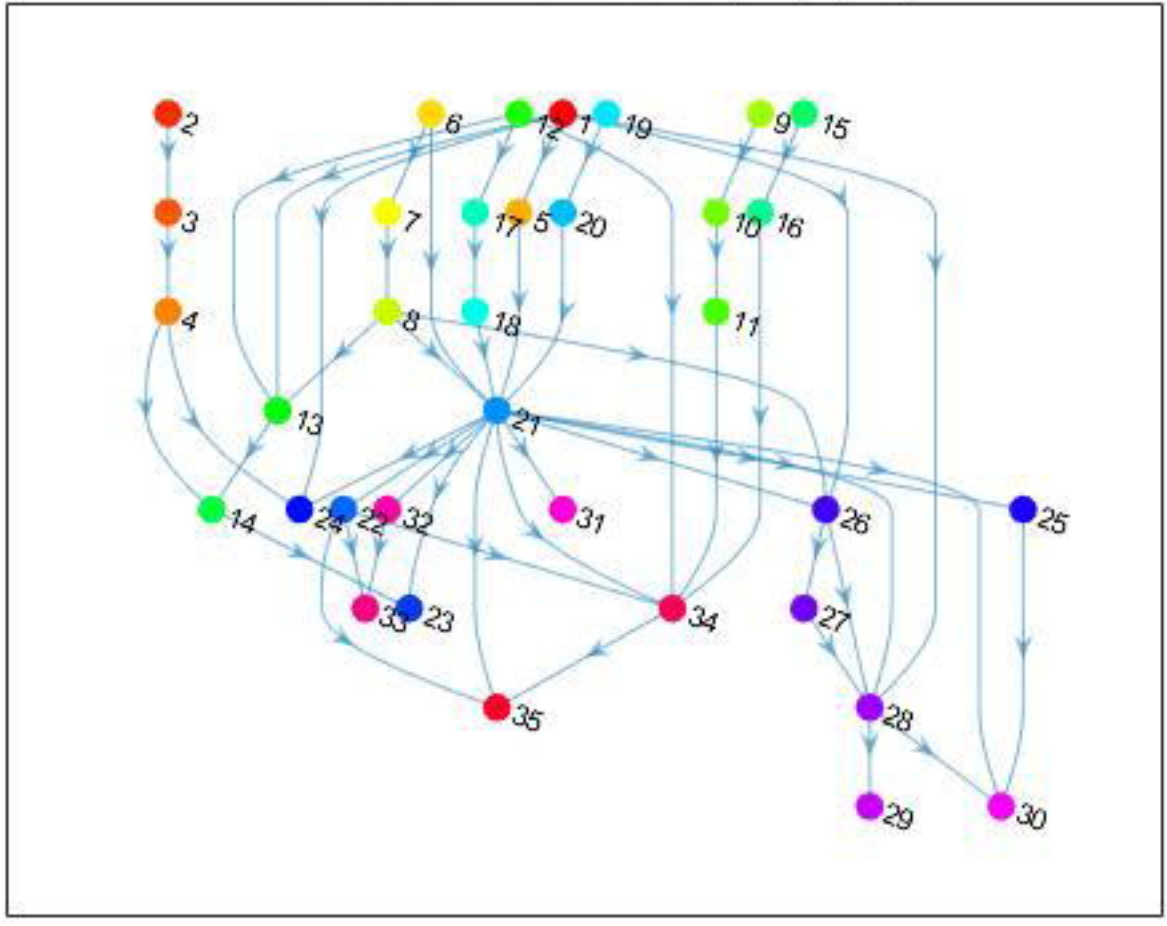
# Modules for SMAD Mutation [8,16,21]

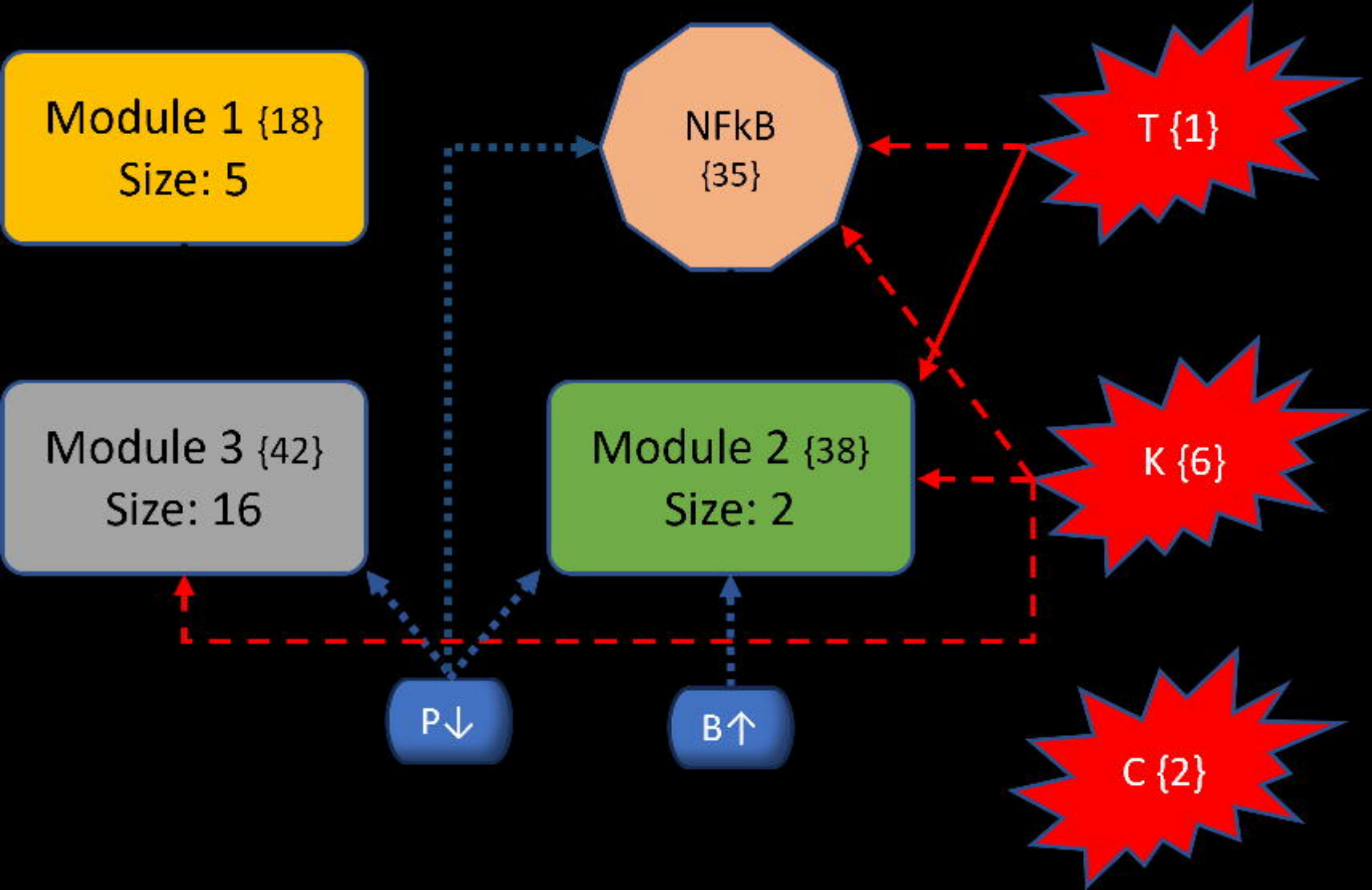



Module 1 {9}
Size: 5
Module 2 {17}
Size: 30
P↓
T {1}
Module 3 {22}
Size: 2
B↑

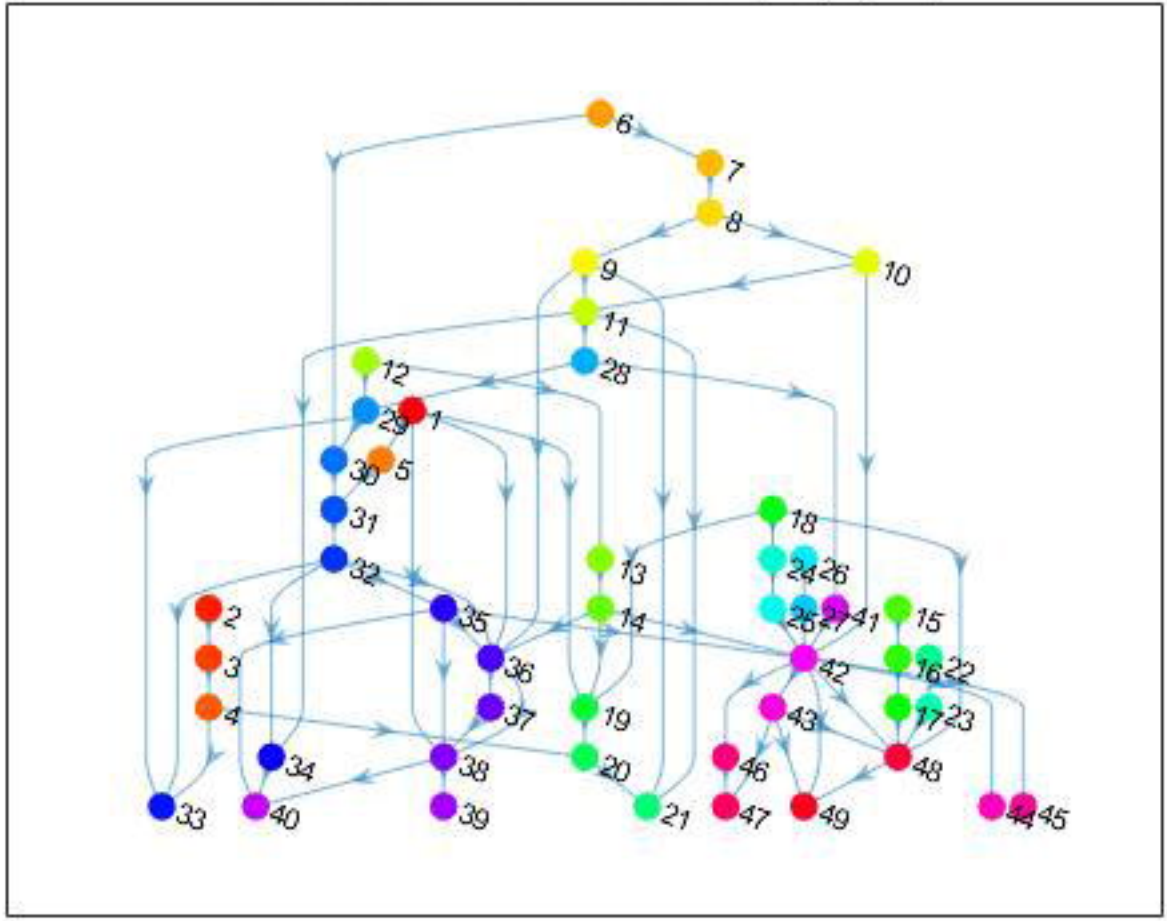

Module 1 {12}
Size: 5
C {2}
Module 2 {21}
Size: 30
P↓
T {1}
Module 3 {28}
Size: 2
B↑

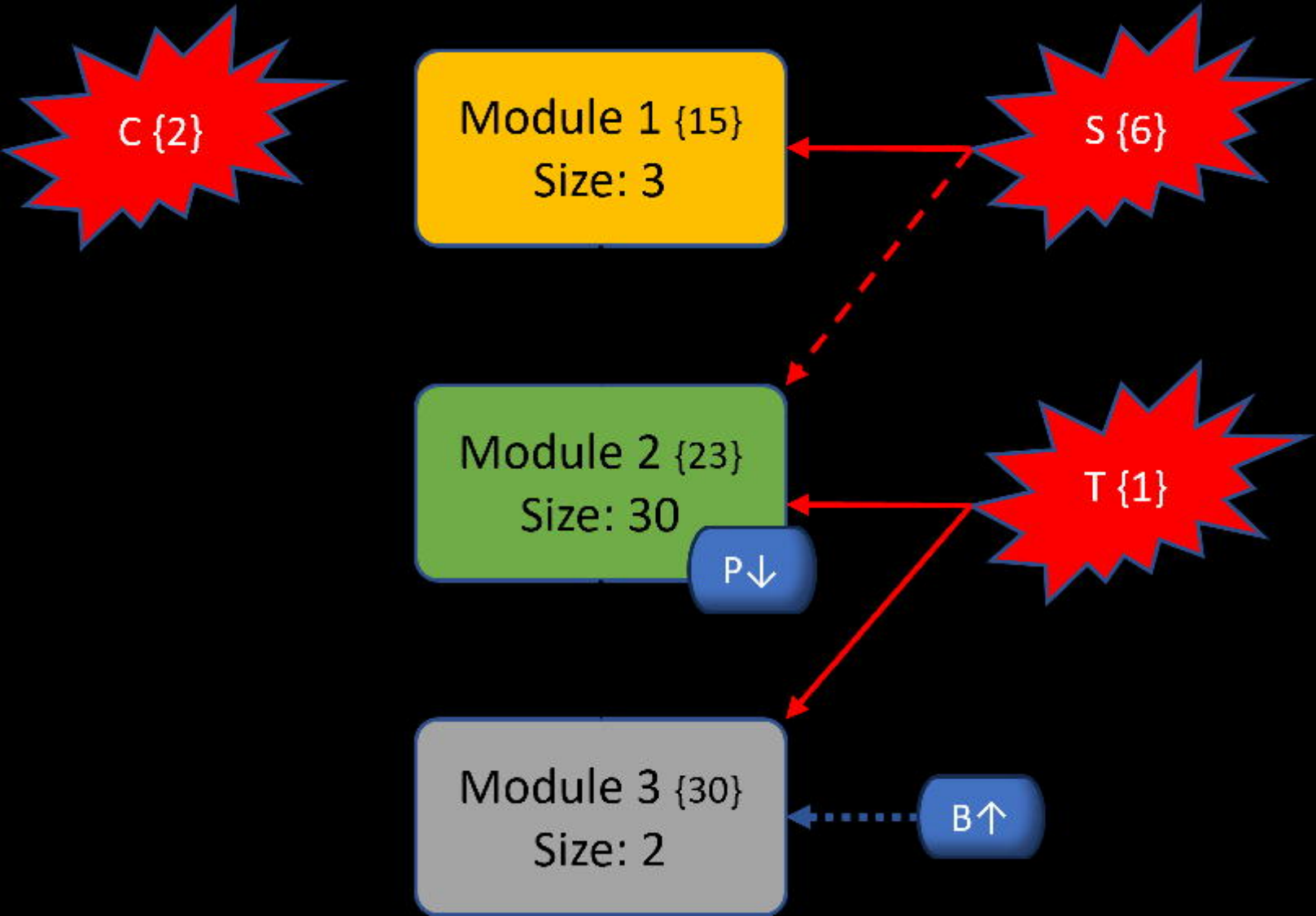
# Modules for T-C Mutation [12,21,28]



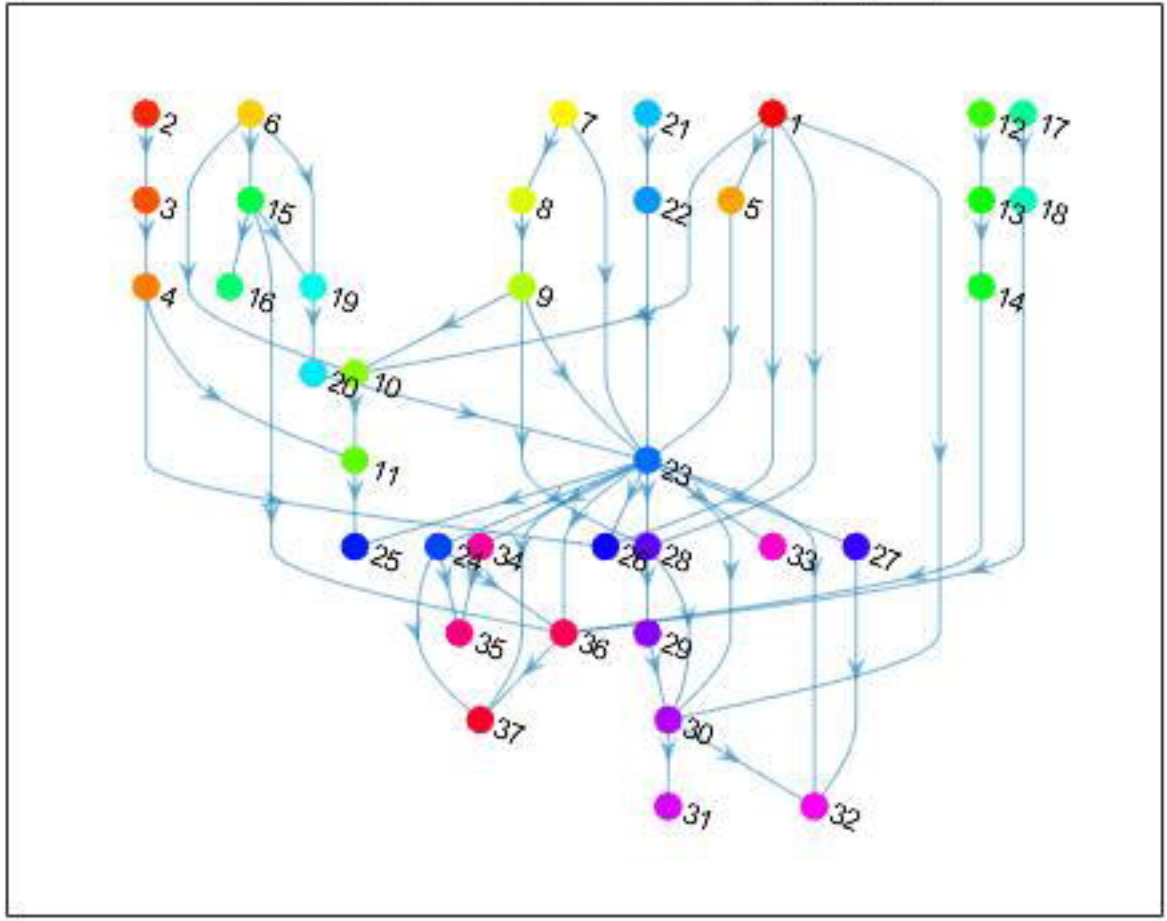

Module 1 {18}
Size: 5
NFkB
{35}
T {1}
Module 3 {42}
Size: 16
Module 2 {38}
Size: 2
K {6}
P↓
B↑
C {2}

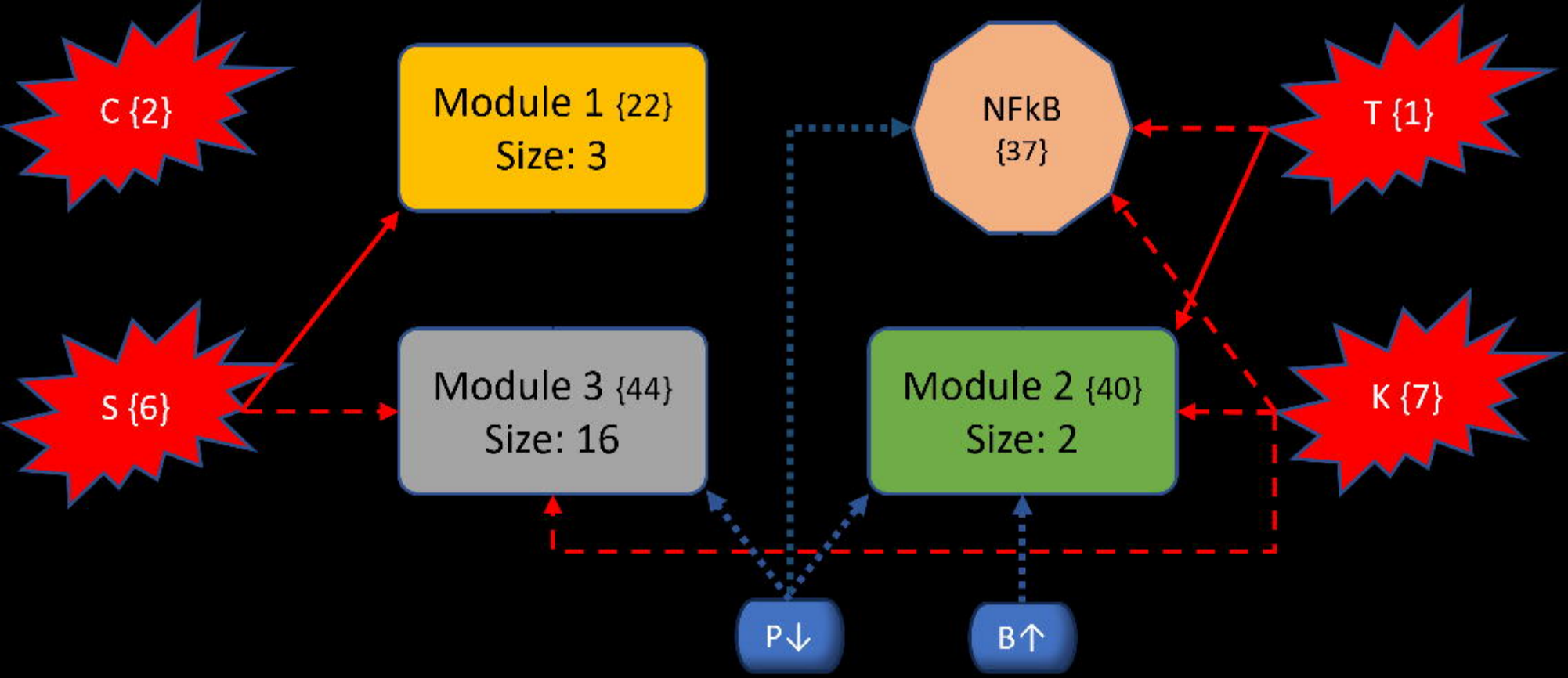
# Modules for T-C-K Mutation [18,38,42]



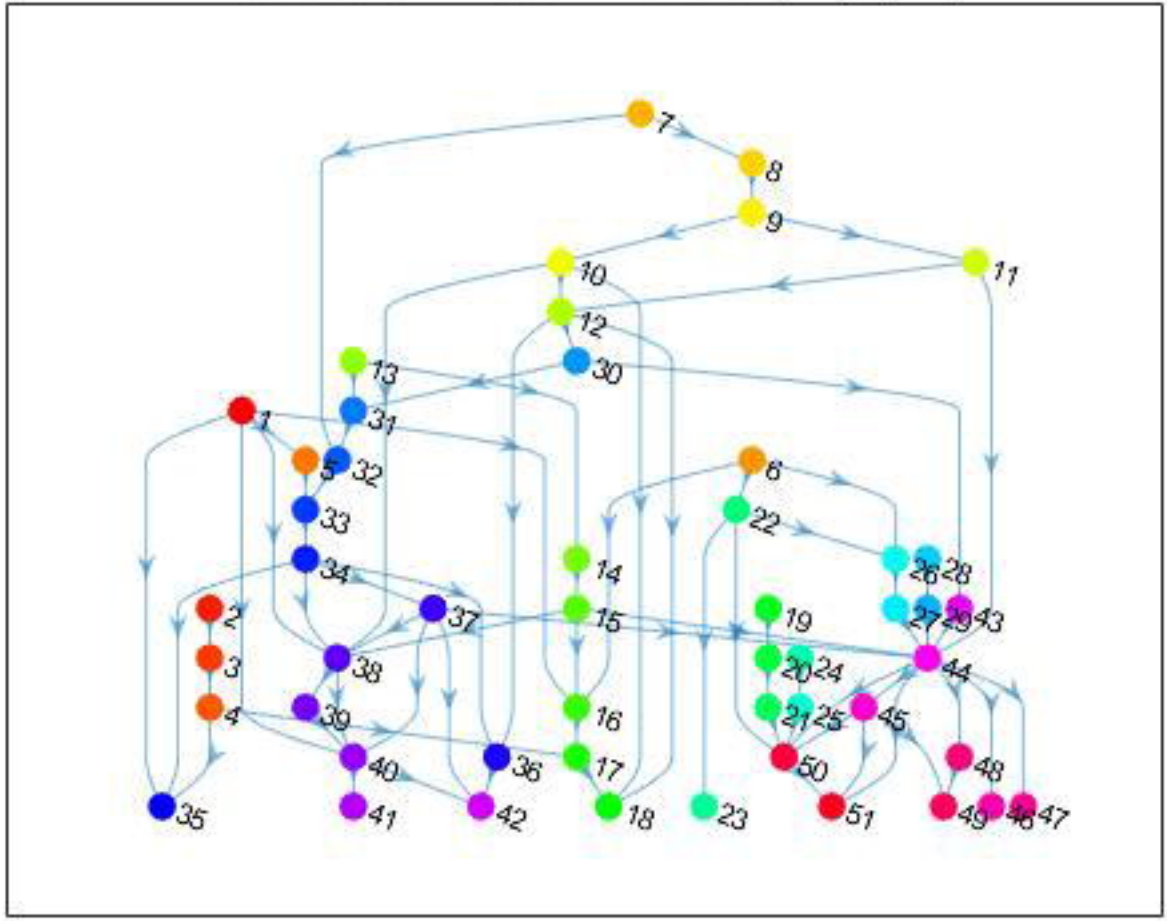

C {2}
Module 1 {15}
Size: 3
S {6}
Module 2 {23}
Size: 30
P↓
T {1}
Module 3 {30}
Size: 2
B↑

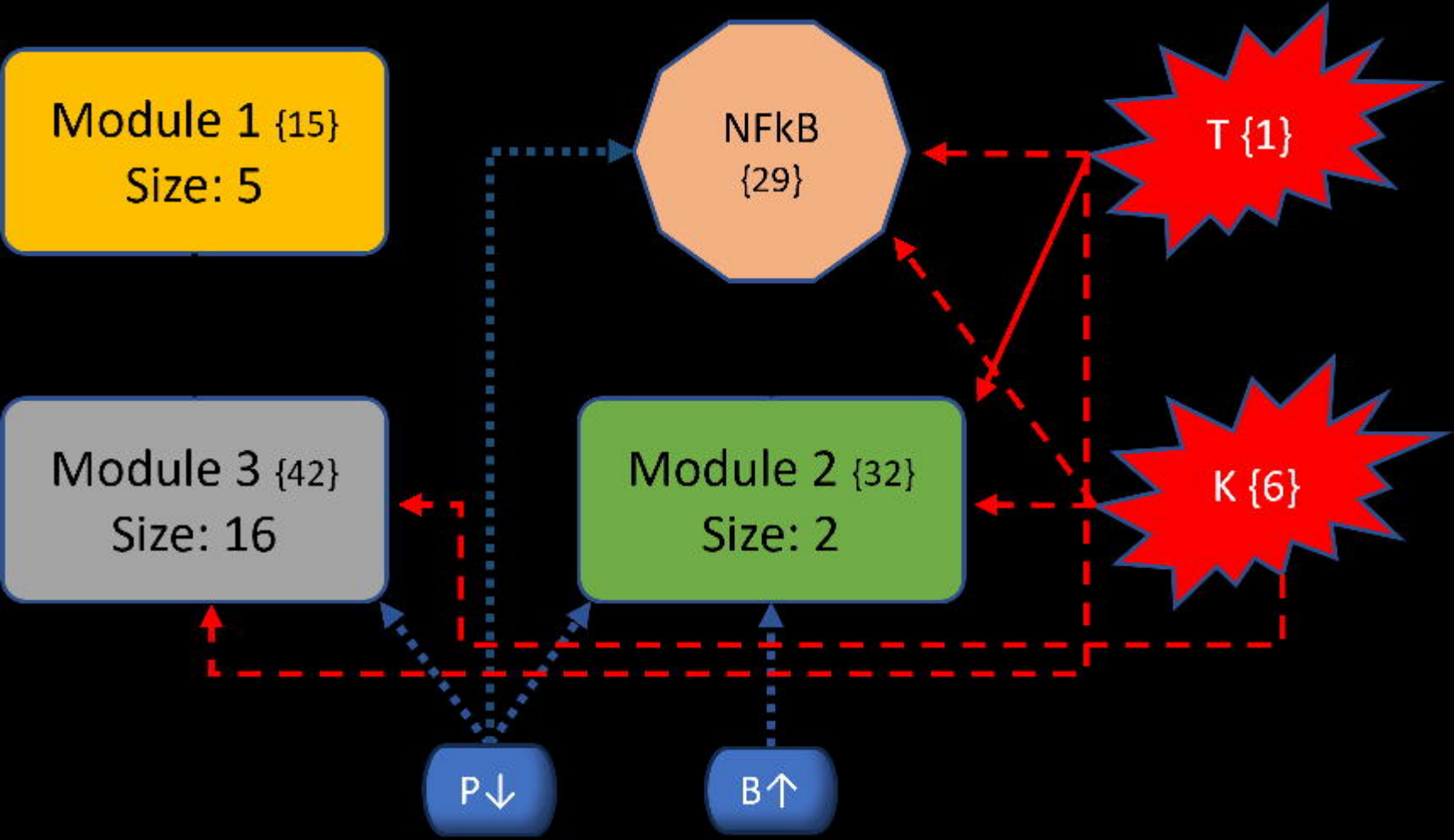
# Modules for T-C-S Mutation [15,23,30]



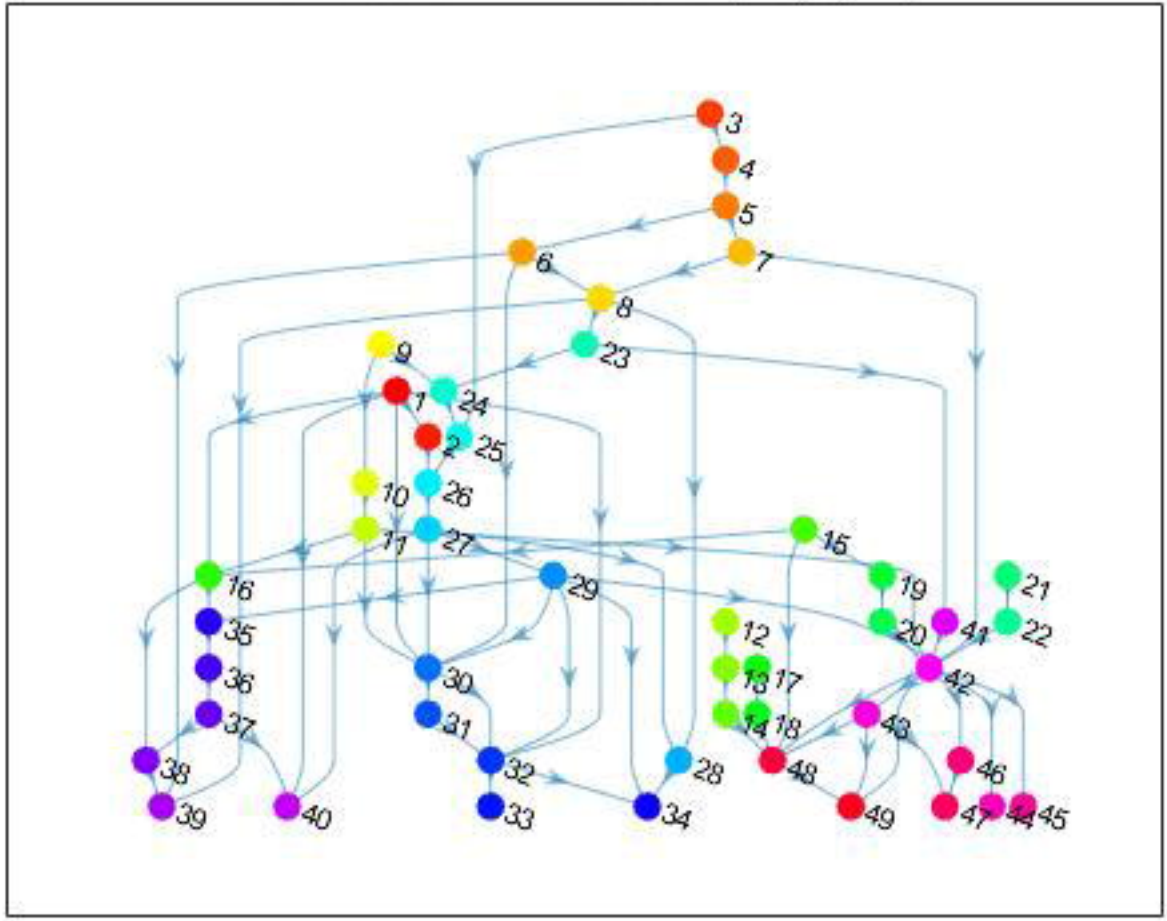

C {2}
Module 1 {22}
Size: 3
NFkB
{37}
T {1}
S {6}
Module 3 {44}
Size: 16
Module 2 {40}
Size: 2
K {7}
P↓
B↑

# Modules for T-C-S-K Mutation [22,40,44]

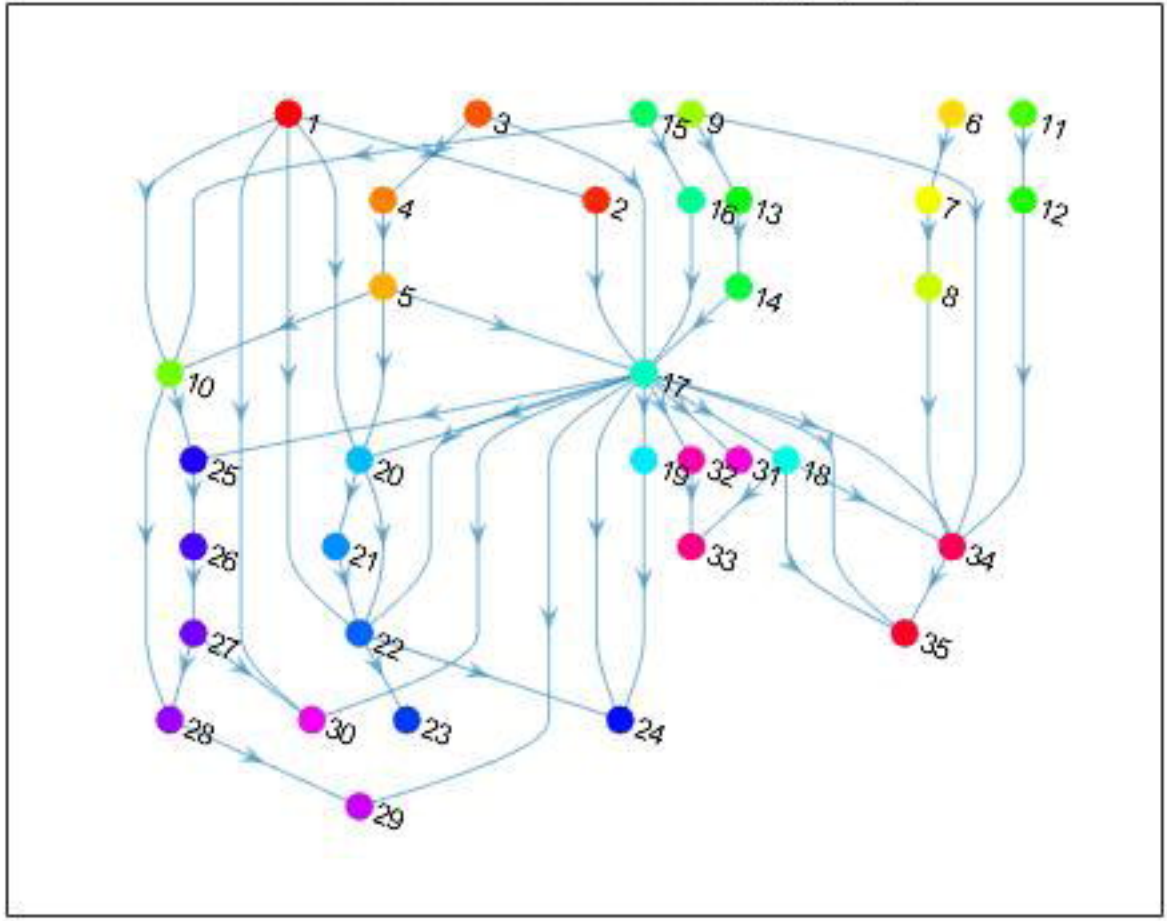


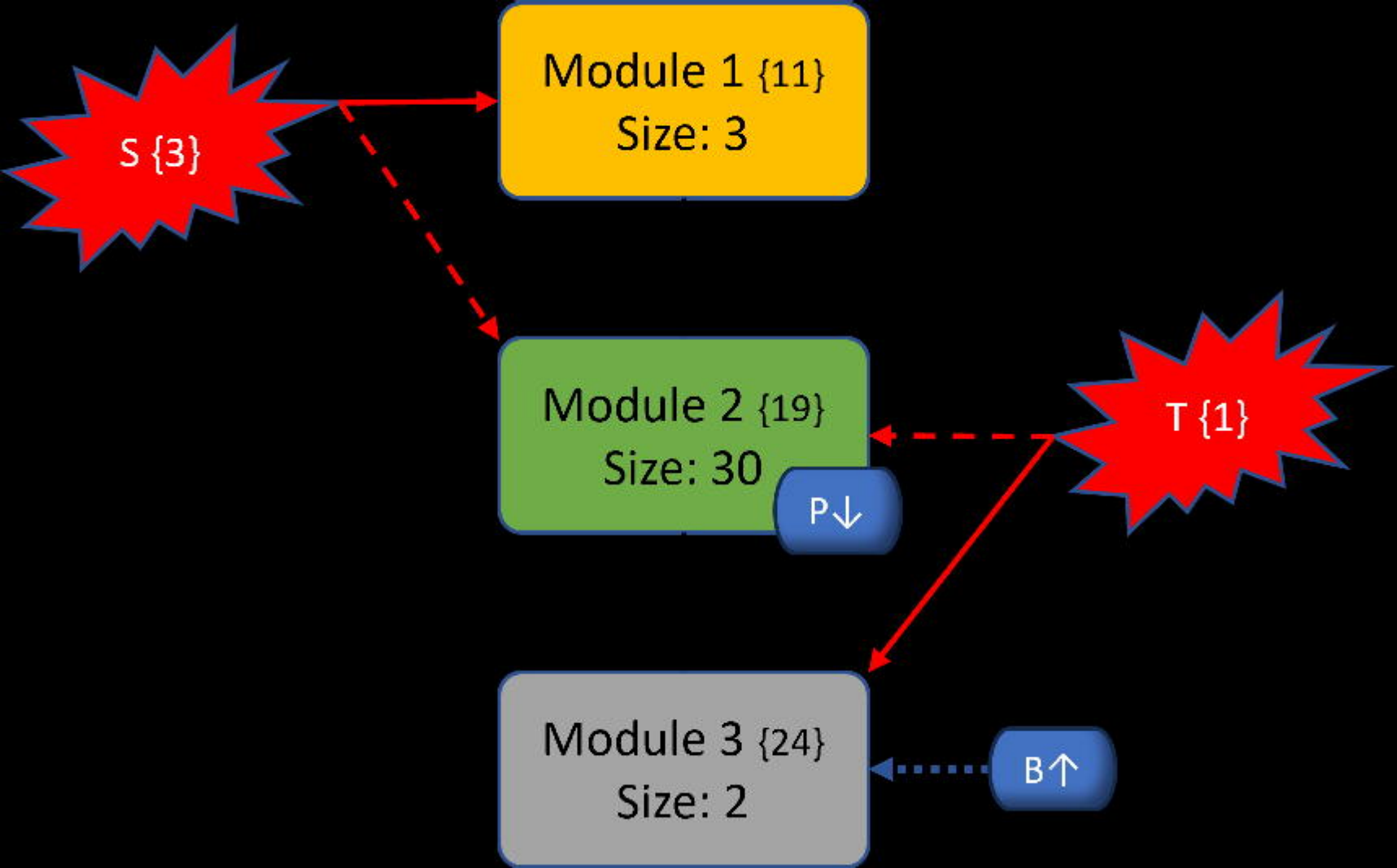

Module 1 {15}
Size: 5
NFkB
{29}
T {1}
Module 3 {42}
Size: 16
Module 2 {32}
Size: 2
K {6}
P↓
B↑

# Modules for T-K Mutation [15,32,42]

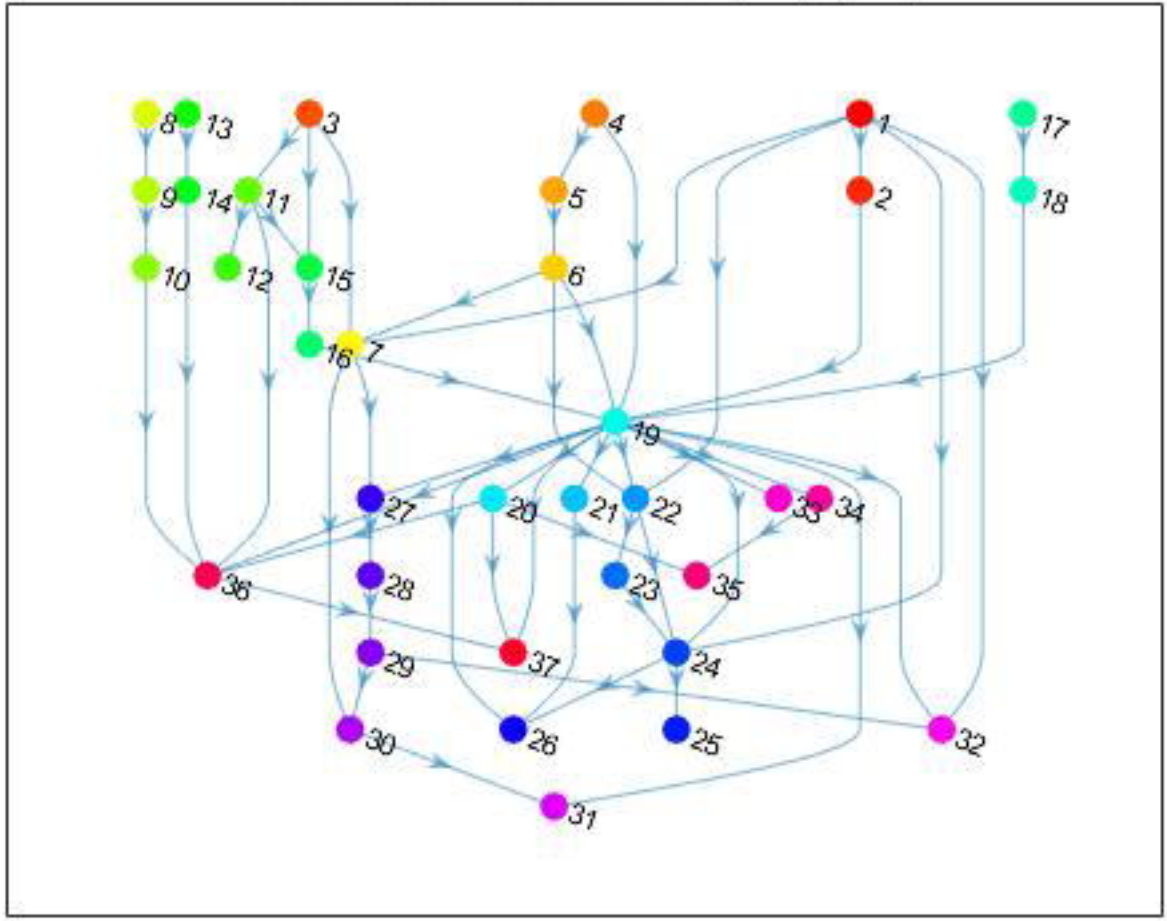


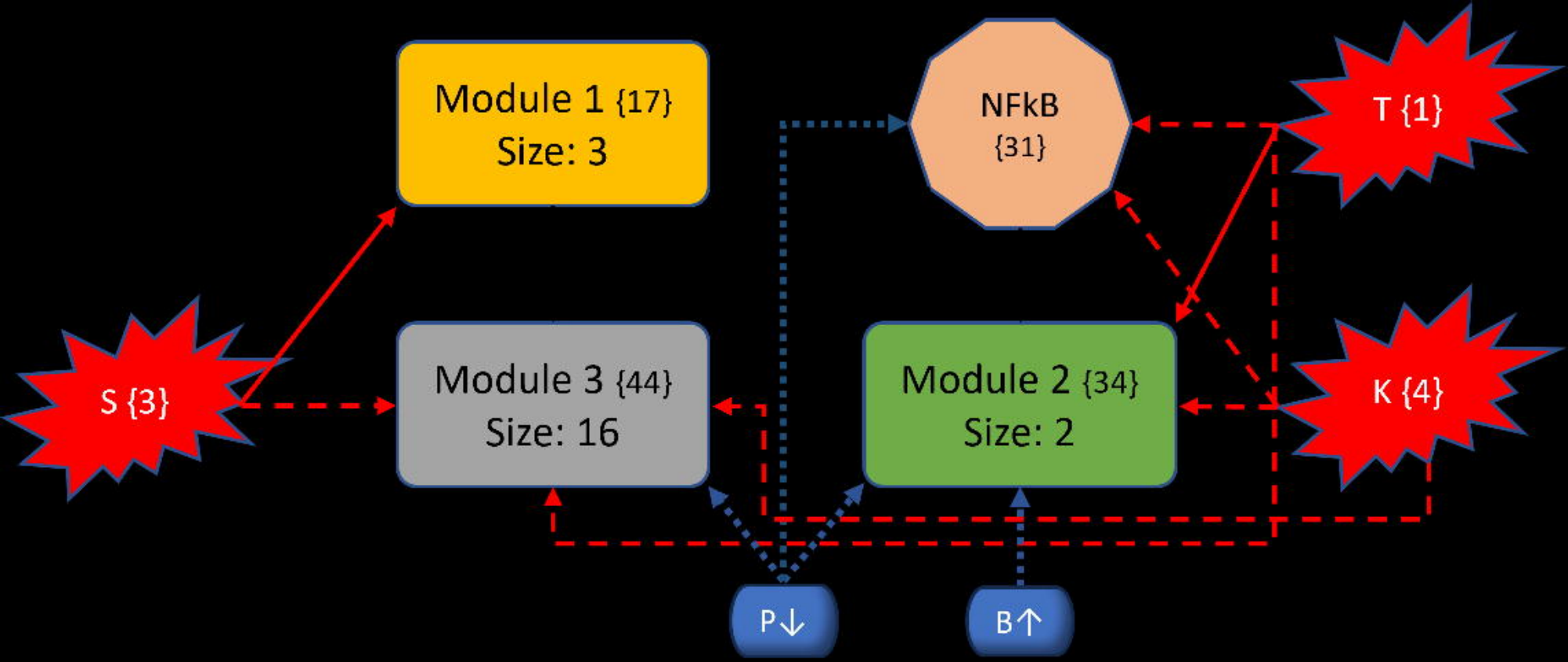
# Modules for TP53 Mutation [9,17,22]



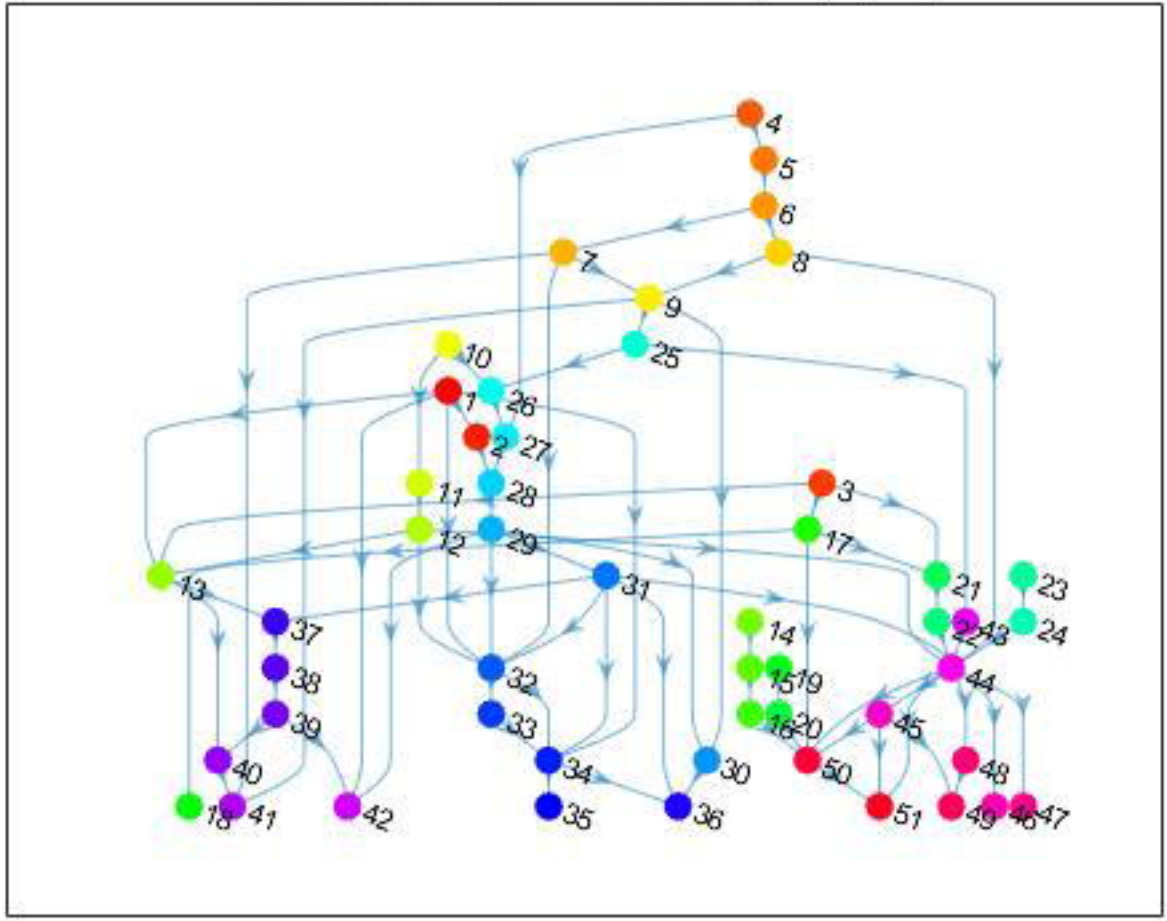

S {3}
Module 1 {11}
Size: 3
Module 2 {19}
Size: 30
P↓
T {1}
Module 3 {24}
Size: 2
B↑

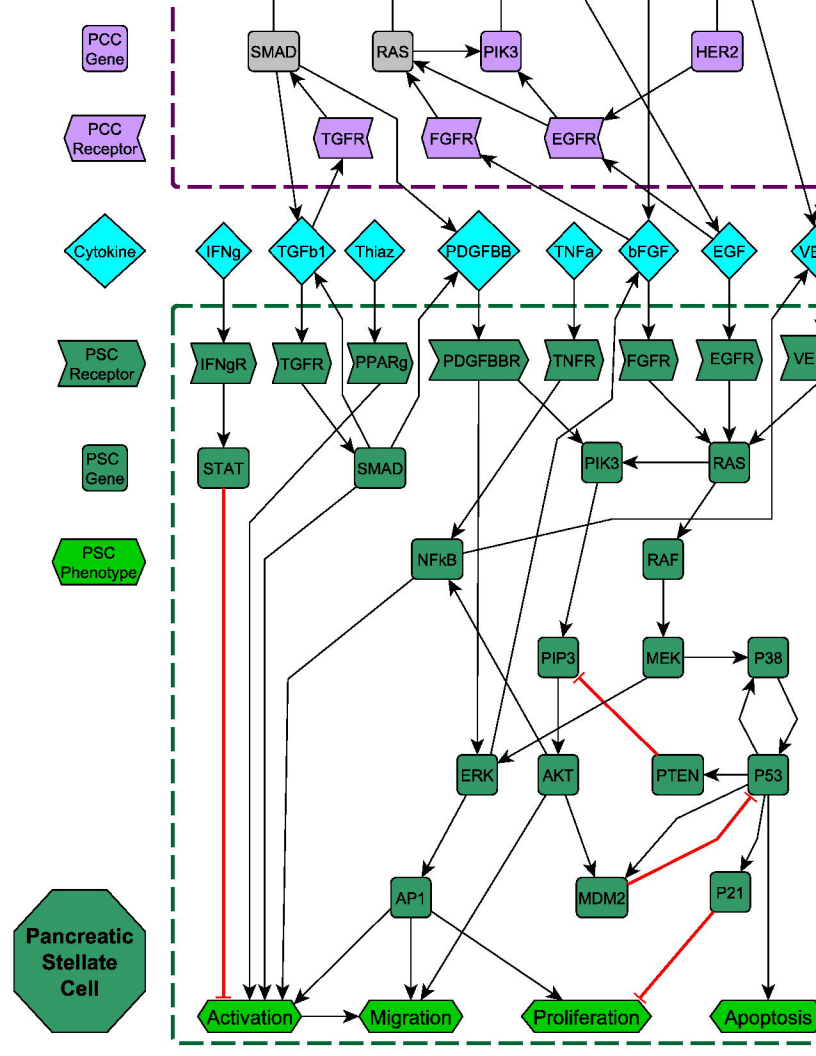
# Modules for T-S Mutation [11,19,24]

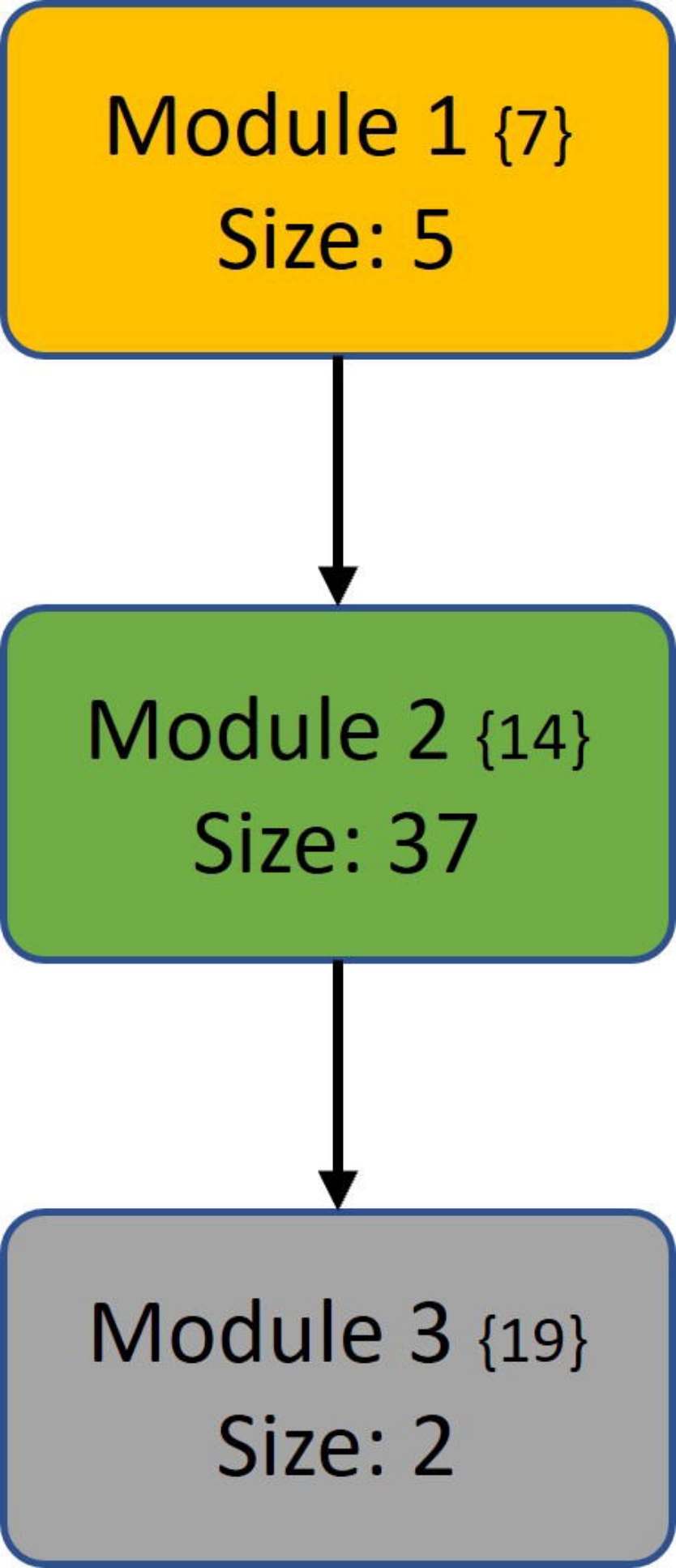



Module 1 {17}
Size: 3
NFkB
{31}
T {1}
S {3}
Module 3 {44}
Size: 16
Module 2 {34}
Size: 2
K {4}
P↓
B↑

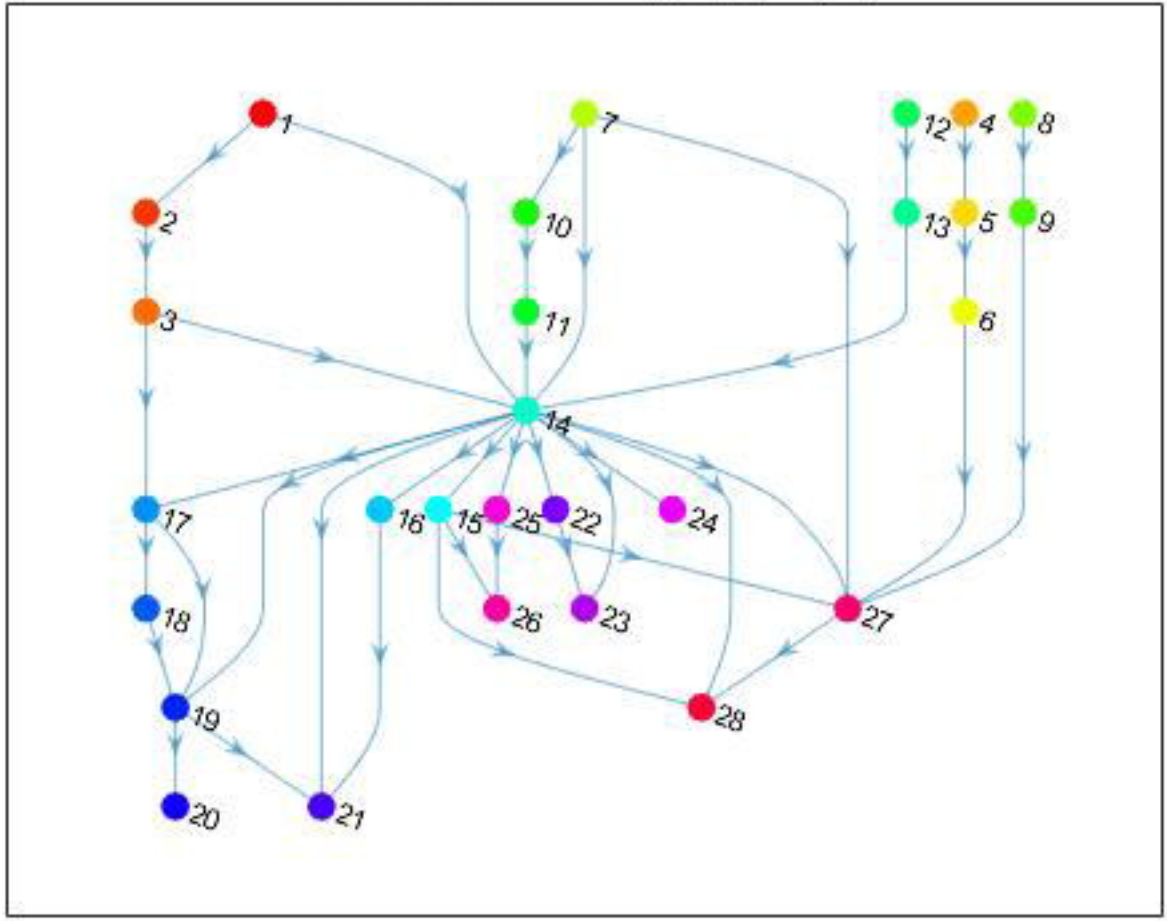
# Modules for T-S-K Mutation [17,34,44]

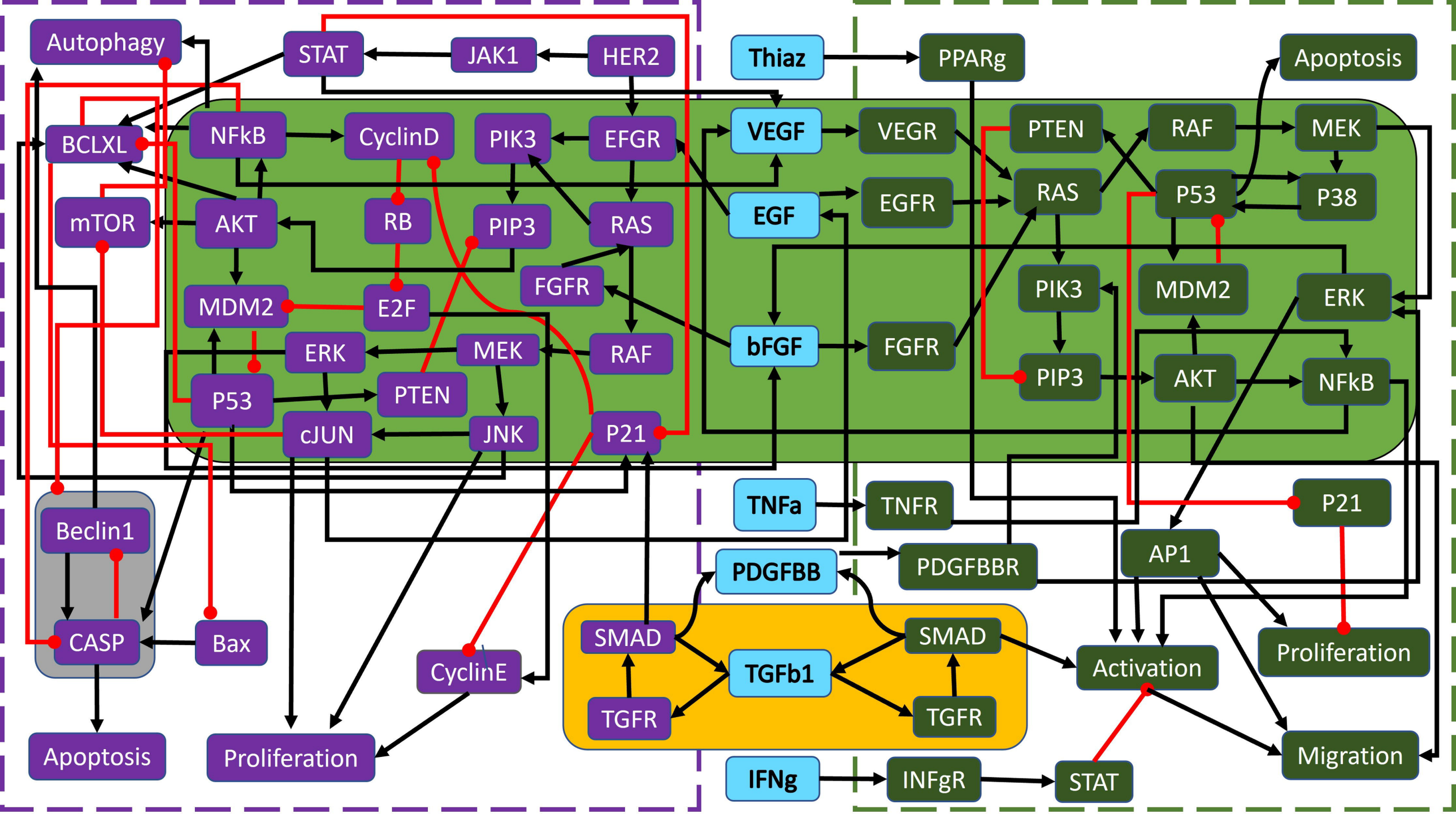


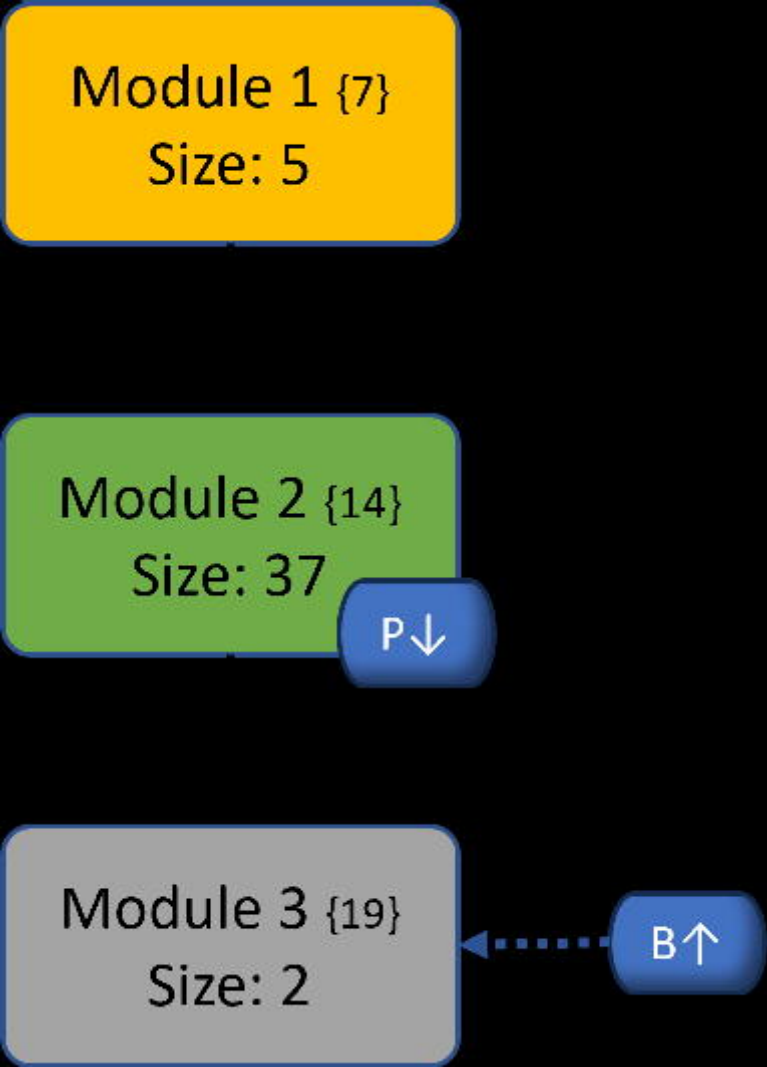

PCC Gene
PCC Receptor
Cytokine
PSC Receptor
PSC Gene
PSC Phenotype
Pancreatic Stellate Cell
SMAD
RAS
PIK3
HER2
TGFR
FGFR
EGFR
IFNg
TGFb1
Thiaz
PDGFBB
TNFa
bFGF
EGF
IFNgR
PPARg
PDGFBBR
TNFR
STAT
NFkB
RAF
PIP3
MEK
P38
ERK
AKT
PTEN
P53
AP1
MDM2
P21
Activation
Migration
Proliferation
Apoptosis


Module 1 {7}
Size: 5
Module 2 {14}
Size: 37
Module 3 {19}
Size: 2

**Modules for Wild-Type [7,14,19]**




Autophagy
STAT
JAK1
HER2
Thiaz
PPARg
Apoptosis
BCLXL
NFkB
CyclinD
PIK3
EFGR
VEGF
VEGR
PTEN
RAF
MEK
mTOR
AKT
RB
PIP3
RAS
EGF
EGFR
RAS
P53
P38
FGFR
PIK3
MDM2
ERK
MDM2
E2F
ERK
MEK
RAF
bFGF
FGFR
PIP3
AKT
NFkB
P53
PTEN
cJUN
JNK
P21
TNFa
TNFR
P21
Beclin1
PDGFBB
PDGFBBR
AP1
SMAD
SMAD
CASP
Bax
CyclinE
TGFb1
Activation
Proliferation
TGFR
TGFR
Apoptosis
Proliferation
Migration
IFNg
INFgR
STAT


Module 1 {7}
Size: 5
Module 2 {14}
Size: 37
P↓
Module 3 {19}
Size: 2
B↑