

Table S1a (The hemostatic system entities interaction KCs), **Table S1b** (The complement system entities interaction KCs), **Table S1c** (The hemostatic system and complement system cross-talk KCs), **Table S1d** (The hemostatic and complement systems entities and SARS-CoV-2 structure proteins interaction KCs), and **Table S1e** (Drug-Target interaction KCs).

Table S1a. The hemostatic system entities interaction KCs

Meaning	Rate constant	Value adopted	Source
Association rate for TF and F7	$k_{\text{TF}^+\text{F7}}^+$	$3.2 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$	[1], [2]
Disassociation rate for TF:F7	$k_{\text{TF}^-\text{F7}}^-$	$3.1 \times 10^{-3} \text{ s}^{-1}$	
Association rate for TF and F7a	$k_{\text{TF}^+\text{F7a}}^+$	$2.3 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	[1], [2]
Disassociation rate for TF:F7a	$k_{\text{TF}^-\text{F7a}}^-$	$3.1 \times 10^{-3} \text{ s}^{-1}$	
Activation of F7 by TF:F7a	$k_{\text{catF7}}^{\text{TF}^+\text{F7a}}$	$4.4 \times 10^5 \text{ M}^{-1}\text{s}^{-1}$	[2]
Activation of F7 by F10a	$k_{\text{catF7}}^{\text{F10a}}$	$1.3 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	[2]
Activation of F7 by F2a	$k_{\text{catF7}}^{\text{F2a}}$	$2.3 \times 10^4 \text{ M}^{-1}\text{s}^{-1}$	[2]
Association rate for F7a and F9	k_{F7aF9}^+	$1.0 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$	[2]
Disassociation rate for F7a:F9	k_{F7aF9}^-	0.9 s^{-1}	
Production of F9a by F7a	$k_{\text{catF9a}}^{\text{F7a}}$	$3.6 \times 10^{-5} \text{ s}^{-1}$	[2]
Association rate for F7a and F10	k_{F7aF10}^+	$1.0 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$	[2]
Disassociation rate for F7a:F10	k_{F7aF10}^-	210.0 s^{-1}	
Production of F10a by F7a	$k_{\text{catF10a}}^{\text{F7a}}$	$1.6 \times 10^{-6} \text{ s}^{-1}$	[2]
Association rate for TF:F7a and F9	$k_{\text{TF}^+\text{F7aF9}}^+$	$1.0 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	[2]
Disassociation rate for TF:F7a:F9	$k_{\text{TF}^-\text{F7aF9}}^-$	2.4 s^{-1}	
Activation of F9 by TF:F7a	$k_{\text{catF9}}^{\text{TF}^+\text{F7a}}$	1.8 s^{-1}	[2]

Association rate for TF:F7a and F10	$k_{\text{TF}^+\text{F7aF10}}^+$	$2.5 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	[2]
Disassociation rate for TF:F7a:F10	$k_{\text{TF}^-\text{F7aF10}}^-$	1.05 s^{-1}	
Activation of F10 by TF:F7a	$k_{\text{catF10}}^{\text{TF}^+\text{F7a}}$	6.0 s^{-1}	[2]
Association rate for TF:F7a and F10a	$k_{\text{TF}^+\text{F7aF10a}}^+$	$2.2 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	[2]
Disassociation rate for TF:F7a:F10a	$k_{\text{TF}^-\text{F7aF10a}}^-$	19.0 s^{-1}	
Association rate for F10a and TFPI	k_{F10aTFPI}^+	$9.0 \times 10^5 \text{ M}^{-1}\text{s}^{-1}$	[2]
Disassociation rate for F10a:TFPI	k_{F10aTFPI}^-	$3.6 \times 10^{-4} \text{ s}^{-1}$	
Binding of TF:F7a:F10a and TFPI	$k_{\text{TF}^+\text{F7aF10aTFPI}}^+$	$3.2 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$	[2]
Disassociation of TF:F7a:F10a	$k_{\text{TF}^-\text{F7aF10aTFPI}}^-$	$1.1 \times 10^{-4} \text{ s}^{-1}$	
Binding of F10a:TFPI and TF:F7a	$k_{\text{F10aTFPI}^+\text{TF}^+\text{F7a}}^+$	$5.0 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	[2]
Association rate for F11a and F9	k_{F11aF9}^+	$1.0 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$	[2], [3]
Disassociation rate for F11a:F9	k_{F11aF9}^-	41.0 s^{-1}	
Generation of F9a by F11a	$k_{\text{catF9a}}^{\text{F11a}}$	7.7 s^{-1}	F11a can generate F9a [2], [3].
Inhibition of F11a by AT3	k_{F11aAT3}^+	$3.2 \times 10^2 \text{ M}^{-1}\text{s}^{-1}$	Destruction of F11a by inhibitor AT3 [2], [3].
Binding of F12a and F11	k_{F12aF11}^+	$1.0 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$	[2], [3].
Disassociation of F12a:F11	k_{F12aF11}^-	200.0 s^{-1}	
Production of F11a by F12a	$k_{\text{catF11a}}^{\text{F12a}}$	$5.7 \times 10^{-3} \text{ s}^{-1}$	F12a can produce F11a [3].
Binding of F12 and F12a	k_{F12F12a}^+	$1.0 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$	[2], [3]
Disassociation of F12:F12a	k_{F12F12a}^-	750.0 s^{-1}	

Autocatalysis of F12a amplification by F12:F12a	$k_{\text{catF12a}}^{\text{F12F12a}}$	$3.3 \times 10^{-2} \text{ s}^{-1}$	The product of F12 and F12a autocatalysis of F12a and increase its expression level [2], [3].
Association rate for P-KAL and F12a	k_{PKALF12a}^{+}	$1.0 \times 10^8 \text{ M}^{-1} \text{ s}^{-1}$	Binding of P-KAL with F12 and disassociation of their product P-KAL:F12 [2], [3].
Disassociation rate for P-KAL:F12a	k_{PKALF12a}^{-}	$3.6 \times 10^3 \text{ s}^{-1}$	
Association rate for KAL and F12	k_{KALF12}^{+}	$1.0 \times 10^8 \text{ M}^{-1} \text{ s}^{-1}$	Binding of KAL with F12 and disassociation of their product KAL:F12 [2], [3].
Disassociation rate for KAL:F12	k_{KALF12}^{-}	45.3 s^{-1}	
Production of F12a by KAL	$k_{\text{catF12a}}^{\text{KAL}}$	5.7 s^{-1}	[2], [3]
Association rate for F9a and F10	k_{F9aF10}^{+}	$1.0 \times 10^8 \text{ M}^{-1} \text{ s}^{-1}$	[2], [3]
Disassociation rate for F9a:F10	k_{F9aF10}^{-}	0.64 s^{-1}	
Production of F10a by F9a	$k_{\text{catF10a}}^{\text{F9a}}$	$7.0 \times 10^{-4} \text{ s}^{-1}$	[2], [3]
Association rate for F9a and F8a	k_{F9aF8a}^{+}	$1.0 \times 10^7 \text{ M}^{-1} \text{ s}^{-1}$	[2], [3]
Disassociation rate for F9a:F8a	k_{F9aF8a}^{-}	$5.0 \times 10^{-3} \text{ s}^{-1}$	
Binding of F9a and AT3	k_{F9aAT3}^{+}	$4.9 \times 10^2 \text{ M}^{-1} \text{ s}^{-1}$	Inhibition of F9a by AT3 [2], [3].
Association rate for F9a:F8a and F10	$k_{\text{F9aF8aF10}}^{+}$	$1.0 \times 10^8 \text{ M}^{-1} \text{ s}^{-1}$	[2], [3]
Disassociation rate for F9a:F8a:F10	$k_{\text{F9aF8aF10}}^{-}$	$1.0 \times 10^{-3} \text{ s}^{-1}$	
Activation of F10 by F9a:F8a	$k_{\text{catF10}}^{\text{F9aF8a}}$	8.2 s^{-1}	[2], [3]
Association rate for F10a and F8	k_{F10aF8}^{+}	$1.0 \times 10^8 \text{ M}^{-1} \text{ s}^{-1}$	[2], [3]
Disassociation rate for F10a:F8	k_{F10aF8}^{-}	2.1 s^{-1}	
Production of F8a by F10a	$k_{\text{catF8a}}^{\text{F10a}}$	$2.3 \times 10^{-2} \text{ s}^{-1}$	F10a produce F8a [2], [3].

Binding of F10a and F5a	$k_{F10aF5a}^+$	$4.0 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$	[2], [3]
Disassociation of F10a:F5a	$k_{F10aF5a}^-$	0.2 s^{-1}	
Inhibition of F0a by AT3	$k_{F10aAT3}^+$	$1.5 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	Binding of F10a and AT3 [2], [3].
Binding of F10a:F5a and F2	$k_{F10aF5aF2}^+$	$1.0 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$	[2], [3]
Disassociation of F10a:F5a:F2	$k_{F10aF5aF2}^-$	103.0 s^{-1}	
Activation of F2 by F10a	k_{catF2}^{F10a}	$7.5 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	Binding of F10a and F2 [2], [3].
Inhibition of F2a by A2M	k_{F2aA2M}^+	$2.5 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	Association rate of A2M and F2a [4].
Association rate for TF:F7a and AT3	$k_{TF7aAT3}^+$	$2.3 \times 10^2 \text{ M}^{-1}\text{s}^{-1}$	Inhibition of TF:F7a by AT3 [2].
Binding of F11 and F2a	k_{F11F2a}^+	$1.0 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$	[2], [3]
Disassociation of F11:F2a	k_{F11F2a}^-	5.0 s^{-1}	
Generation of F11a	k_{catF2a}^{F11}	$1.3 \times 10^{-4} \text{ s}^{-1}$	[2], [3]
Inhibition of F2a by AT3	k_{F2aAT3}^+	$7.1 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	Binding of AT3 and F2a [2], [3].
Activation of F5 by F2a	k_{catF5}^{F2a}	$2.0 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	[2], [3]
Binding of F2a and F1	k_{F2aF1}^+	$1.17 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	[5], [6]
Disassociation of F2a:F1	k_{F2aF1}^-	84.0 s^{-1}	
Production of F1a	k_{catF1a}^{F2a}	84 s^{-1}	[5], [6]
Activation of F8 by F2a	k_{catF8}^{F2a}	$2.0 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	F2a binds with F8 [2], [3].
Inhibition of F12a by AT3	$k_{F12aAT3}^+$	$21.6 \text{ M}^{-1}\text{s}^{-1}$	AT3 is associated with F12a and inhibits it [2], [3].
Production of KAL by F12a	k_{catKAL}^{F12a}	40 s^{-1}	F12a can generates KAL) [2], [3].

Activation of KAL by P-KAL	k_{KALPKAL}^+	$2.7 \times 10^4 \text{ M}^{-1}\text{s}^{-1}$	KAL auto-activation [2], [3].
Generation of Lys-Pg by Glu-Pg	$k_{\text{catLys-Pg}}^{\text{Glu-Pg}}$	0.1 s^{-1}	Pg variants are Lys-Pg and Glu-Pg [7]. Lys-Pg can be generated by Pn hydrolysis of Glu-Pg [8]. Conversion of Glu- to Lys-Lys-Pg [9]–[11]. In literature the generation rate of Lys-Pg not available. Assume a small value for it.
Activation of Glu-Pg by tPA	$k_{\text{cattPA}}^{\text{F1a}}$ $k_{\text{mtPA}}^{\text{F1a}}$	$4.1 \times 10^{-7} \text{ s}^{-1}$ 0.073 M	tPA activates Glu-Pg in the presence of F1a [8].
Activation of Lys-Pg by tPA	$k_{\text{cattPA}}^{\text{F1a}}$ $k_{\text{mtPA}}^{\text{F1a}}$	$2.0 \times 10^{-8} \text{ s}^{-1}$ 0.064 M	tPA activates Lys-Pg in the presence of F1a [8].
Association rate for Glu-Pg and FDPs Disassociation rate for Glu-Pg:FDPs	$k_{\text{Glu-PgFDPs}}^+$ $k_{\text{Glu-PgFDPs}}^-$	$5.71 \times 10^5 \text{ M}^{-1}\text{s}^{-1}$ 0.1 s^{-1}	Native Plasminogen (Glu-Pg) binds to FDPs with affinity ($k_d = 175 \times 10^{-9} \text{ M}$) [12]. The Association rate (k^+) can be computed from the formula $k_d = \frac{k^-}{k^+}$. Assume a small value for $k^- = 0.1 \text{ s}^{-1}$.
Association rate for Lys-Pg and FDPs Disassociation rate for Lys-Pg:FDPs	$k_{\text{Lys-PgFDPs}}^+$ $k_{\text{Lys-PgFDPs}}^-$	$1.11 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$ 0.1 s^{-1}	Lys-Pg binds to FDPs with affinity ($k_d = 90 \times 10^{-9} \text{ M}$) [12]. The Association rate (k^+) can be computed from the formula $k_d = \frac{k^-}{k^+}$. Assume a small value for $k^- = 0.1 \text{ s}^{-1}$.
Inhibition of Glu-Pg:FDPs by TAF1a	$k_{\text{Glu-PgFDPsTAF1a}}^+$	$1.7133 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	Glu-Pg is degraded by TAF1a in the presence of FDPs. Treatment of the FDPs with TAF1a removes Glu-Pg binding sites. When FDPs are treated with TAF1a the k_d increases to $1 \times 10^{-6} \text{ M}$, $k_{\text{cat}} = 2.35 \text{ s}^{-1}$, $k_m = 0.143 \mu \text{ M}$ [12]. The Association rate (k^+) can be computed from the formula $k^+ = \frac{k^- + k_{\text{cat}}}{k_m}$. Assume a small value for $k^- = 0.1 \text{ s}^{-1}$.
Inhibition of Lys-Pg:FDPs by TAF1a	$k_{\text{Lys-PgFDPsTAF1a}}^+$	$1.051 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	Lys-Pg is degraded by TAF1a in the presence of FDPs. Treatment of the FDPs with TAF1a removes Glu-Pg binding sites. When FDPs are treated with TAF1a the k_d increases to $1.55 \times 10^{-6} \text{ M}$, $k_{\text{cat}} = 0.888 \text{ s}^{-1}$, $k_m = 0.094 \mu \text{ M}$ [12]. The Association rate (k^+) can be computed from the formula $k^+ = \frac{k^- + k_{\text{cat}}}{k_m}$. Assume a small value for $k^- = 0.1 \text{ s}^{-1}$.

Association rate for F1a and Lys-Pg	$k_{F1aLys-Pg}^+$	$1.0 \times 10^{-7} M^{-1}s^{-1}$	Lys-Pg binds to F1a [7], [8]. The binding rate constant for Pg to F1 and the unbinding rate constant for Pg from F1 [13].
Disassociation rate for F1a:Lys-Pg	$k_{F1aLys-Pg}^-$	$3.8 s^{-1}$	
Activation of F1 by Lys-Pg	k_{catF1}^{Lys-Pg}	$1.0 \times 10^{-7} M^{-1}s^{-1}$	Lys-Pg can binds to F1a, the binding rate measured $1.0 \times 10^{-7} M^{-1}s^{-1}$ [7], [8]. Assumption for activation rate of F1 to F1a by Lys-Pg.
Inhibition of tPA by PAI-1	$k_{tPA PAI-1}^+$	$2 \times 10^7 M^{-1}s^{-1}$	PAI-1 is an efficient inhibitor of tPA [14].
Production of tPA by F1a:Lys-Pg	$k_{cattPA}^{F1aLys-Pg}$	$0.1 s^{-1}$	Assumption for the rate of tPA production by F1a: Lys-Pg, as in propagation phase of fibrinolysis, F1a can generates tPA in the presence of Lys-Pg [15].
Generation of Lys-Pg by Pn	$k_{catLys-Pg}^{Pn}$	$0.1 s^{-1}$	Assumed a small value for the rate of Lys-Pg production by Pn. As in propagation phase of fibrinolysis, Lys-Pg can be produced by Pn [15].
Association rate for P-KAL and HK	k_{PKALHK}^+	$8.3 \times 10^7 M^{-1}s^{-1}$	P-KAL binds to HK with high specificity and affinity ($k_d = 1.2 \times 10^{-8}M$) [16]. The Association rate (k^+) can be computed from the formula $k_d = \frac{k^-}{k^+}$. Assumed a small value for $k^- = 1s^{-1}$.
Disassociation rate for P-KAL:HK	k_{PKALHK}^-	$1s^{-1}$	
Activation of HK by KAL	k_{catHK}^{KAL}	$8.3 \times 10^7 M^{-1}s^{-1}$	Estimated based on association rate of P-KAL and HK.
Generation of BK by P-KAL:HK	k_{catBK}^{PKALHK}	$0.1 s^{-1}$	The product of P-KAL and HK can release BK [17]. The generation rate is unknown, estimated a small value for it.
Association rate for BK and B2R	k_{BKB2R}^+	$3.4 \times 10^8 M^{-1}s^{-1}$	BK binds to B2R with a high affinity ($k_d = 2.90 \times 10^{-9}$) [18]. The disassociation rate (k^{-1}) is unknown, assumed a small value for $k^{-1} = 1 s^{-1}$. Using the formula $k_d = \frac{k^-}{k^+}$, determined k^+ .
Disassociation rate for BK:B2R	k_{BKB2R}^-	$1 s^{-1}$	
Association rate for HK and F11	k_{HKF11}^+	$0.40 \times 10^7 M^{-1}s^{-1}$	F11 can binds to HK with an affinity $k_d = (2 - 3) \times 10^{-7}M$ [19], [20]. Considered $k_d = 2.5 \times 10^{-7}M$. The disassociation rate (k^{-1}) is unknown, assumed $k^{-1} = 1 s^{-1}$. Using the formula $k_d = \frac{k^-}{k^+}$, determined k^+ .
Disassociation rate for HK:F11	k_{HKF11}^-	$1 s^{-1}$	
Production of F11a by HK:F11	$k_{catF11a}^{HKF11}$	$0.1 s^{-1}$	The association of HK and F11 can generate F11a. Assumed, a small value for the production of F11a by product of HK and F11.

Production of tPA by BK:B2R	$k_{\text{catPA}}^{\text{BKB2R}}$	0.1 s^{-1}	The product of BK:B2R stimulates tPA [20]. Assumed, a small value for the production of tPA by BK: B2R.
Activation of the F1 by F2a	$k_{\text{catF1}}^{\text{F2a}}$ $k_{\text{mF1}}^{\text{F2a}}$	59 s^{-1} $3.16 \times 10^{-6} \text{ M}$	Enzyme reactions between F2a and F1 [1].
Cleavage of TAFI by Pn	$k_{\text{catTAFI}}^{\text{Pn}}$ $k_{\text{mTAFI}}^{\text{Pn}}$	$4.0 \times 10^{-4} \text{ s}^{-1}$ $5.5 \times 10^{-8} \text{ M}$	Pn cleaves TAFI at Arg92, generating TAFIa. Pn-mediated TAFI activation [9].
Cleavage of TAFI by F2a	$k_{\text{catTAFI}}^{\text{F2a}}$ $k_{\text{mTAFI}}^{\text{F2a}}$	0.17 s^{-1} $8.3 \times 10^{-7} \text{ M}$	F2a can cleaves TAFI and generates active TAFIa [21].
Inhibition of Glu-Pg by TAFIa	$k_{\text{Glu-PgTAFIa}}^{+}$	$1.69 \times 10^7 \text{ M}^{-1} \text{ s}^{-1}$	TAFIa inhibits the activation of Glu-Pg and the conversion of Glu- to Lys-Pg [9]–[11]. $k_{\text{cat}} = 2.30 \text{ s}^{-1}$, $k_m = 0.142 \mu\text{M}$ [22], [23]. The disassociation rate (k^{-}) is unknown, assumed a small value for $k^{-} = 0.1 \text{ s}^{-1}$. Using the formula $k^{+} = \frac{k^{-} + k_{\text{cat}}}{k_m}$, determined the association rate (k^{+}).
Inhibition of Lys-Pg by TAFIa	$k_{\text{Lys-PgTAFIa}}^{+}$	$1.051 \times 10^7 \text{ M}^{-1} \text{ s}^{-1}$	Assumption for association rate of Lys-Pg and TAFIa based on inhibition of Lys-Pg:FDPs by TAFIa.
Inhibition of C3a by TAFIa	k_{C3aTAFIa}^{+}	$2.36 \times 10^5 \text{ M}^{-1} \text{ s}^{-1}$	TAFIa can inhibit C3a. $k_{\text{cat}} = 8.4 \text{ s}^{-1}$, $k_m = 35.9 \mu\text{M}$ [22], [23]. Assume a small value for $k^{-} = 0.1 \text{ s}^{-1}$. Using the formula $k^{+} = \frac{k^{-} + k_{\text{cat}}}{k_m}$, determined k^{+} .
Inhibition of C5a by TAFIa	k_{C5aTAFIa}^{+}	$1.35 \times 10^5 \text{ M}^{-1} \text{ s}^{-1}$	TAFIa can inhibit C5a. $k_{\text{cat}} = 29.5 \text{ s}^{-1}$, $k_m = 219.0 \mu\text{M}$ [22], [23]. Assume a small value for $k^{-} = 0.1 \text{ s}^{-1}$. Using the formula $k^{+} = \frac{k^{-} + k_{\text{cat}}}{k_m}$, determined k^{+} .
Inhibition of BK by TAFIa	k_{BKTAFIa}^{+}	$2.8 \times 10^5 \text{ M}^{-1} \text{ s}^{-1}$	TAFIa can inactivate BK. $k_{\text{cat}} = 19.7 \text{ s}^{-1}$, $k_m = 70.6 \mu\text{M}$ [22], [23]. Assume a small value for $k^{-} = 0.1 \text{ s}^{-1}$. Using the formula $k^{+} = \frac{k^{-} + k_{\text{cat}}}{k_m}$, determined k^{+} .
Activation of F12 by Pn	$k_{\text{catF12}}^{\text{Pn}}$	$1.0 \times 10^3 \text{ M}^{-1} \text{ s}^{-1}$	Pn can cleave and activate F12 [24]. Estimated a small value for activation of F12 by Pn.

Cleavage of F1 by Pn	k_{catF1}^{Pn} k_{mF1}^{Pn}	$7.1 s^{-1}$ $6.5 \times 10^{-6} M$	Pn can activates and cleaves F1 [25].
Cleavage of F1a by Pn	k_{catF1a}^{Pn} k_{mF1a}^{Pn}	$6.4 s^{-1}$ $0.14 \times 10^{-6} M$	Degradation of F1a by Pn which cleaved F1a [26].
Cleavage of Glu-Pg by Pn	$k_{catGlu-Pg}^{Pn}$ $k_{mGlu-Pg}^{Pn}$	$0.1 s^{-1}$ $1.0 \times 10^{-4} M$	Plasmin also enhances fibrinolysis by converting native Pg (Glu-Pg) to Lys-Pg. Pn cleaves Glu-Pg at lysine 77/78 result in Lys-Pg generation [12]. Assumption for the cleavage of Glu-Pg by Pn.
Cleavage of Glu-Pg by KAL	$k_{catGlu-Pg}^{KAL}$ $k_{mGlu-Pg}^{KAL}$	$1.6 \times 10^{-4} s^{-1}$ $5.6 \times 10^{-7} M$	[27]
Cleavage of Glu-Pg by tPA	$k_{catGlu-Pg}^{tPA}$ $k_{mGlu-Pg}^{tPA}$	$4.1 \times 10^{-7} s^{-1}$ 0.073 M	[8]

Table S1b. The complement entities interaction KCs

Meaning	Rate constant	Value adopted	Source
Binding rate for C1r and C1s	k_{C1rC1s}^+	$0.61 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$	Association and disassociation rates of C1r and C1s are measured with respect to different modules such as x-ray structure of the N-terminal CUB-epidermal growth factor (EGF) marked by different residues. Among the high affinity sites at C1rCUB ₁ -Y56A, binding rates and disassociation rates measured as $k^+ = 0.61 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$ and $k^- = 3.44 \times 10^{-3} \text{ s}^{-1}$ [28].
Disassociation rate for C1r:C1s	k_{C1rC1s}^-	$3.44 \times 10^{-3} \text{ s}^{-1}$	
Binding rate for C1q and C1r:C1s	k_{C1qrs}^+	$0.82 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$	[28]–[31]
Disassociation rate for C1q :C1r:C1s	k_{C1qrs}^-	$1.2 \times 10^{-3} \text{ s}^{-1}$	
Binding rate for C1s and C4	k_{C1sC4}^+	$7.9 \times 10^5 \text{ M}^{-1}\text{s}^{-1}$	[32]
Disassociation rate for C1s:C4	k_{C1sC4}^-	$4.8 \times 10^{-1} \text{ s}^{-1}$	
Binding rate for MASP2 and C4	$k_{MASP2C4}^+$	$7.9 \times 10^5 \text{ M}^{-1}\text{s}^{-1}$	Estimation based on MASP2 functionally homologous of C1q [32], [33].
Disassociation rate for MASP2:C4	$k_{MASP2C4}^-$	$4.8 \times 10^{-1} \text{ s}^{-1}$	
Binding rate for C3W and FB	k_{C3WB}^+	$1.1 \times 10^4 \text{ M}^{-1}\text{s}^{-1}$	[30], [31], [34]
Disassociation rate for C3W:FB	k_{C3WB}^-	$1.4 \times 10^{-3} \text{ s}^{-1}$	
Binding rate for C3W and Factor H (FH)	k_{C3WH}^+	$1.1 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$	Estimation structurally/functionally homologous protein C3b and FH [30], [31], [34].
Disassociation rate for C3W:FH	k_{C3WH}^-	$6.0 \times 10^{-2} \text{ s}^{-1}$	
Binding rate for fC3b and FB	k_{fC3bFB}^+	$21.3 \times 10^4 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31].
Disassociation rate for fC3b:FB	k_{fC3bFB}^-	$15.5 \times 10^{-2} \text{ s}^{-1}$	

Binding rate for fC3b:C4b:P and FB	$k_{\text{fC3bC4bPFB}}^+$	$21.3 \times 10^4 \text{ M}^{-1} \text{ s}^{-1}$	[29]–[31], [35].
Disassociation rate for fC3b:C4b:P:FB	$k_{\text{fC3bC4bPFB}}^-$	$15.5 \times 10^{-2} \text{ s}^{-1}$	
Binding rate for IgG:fC3b and FB	$k_{\text{IgGfC3bFB}}^+$	$21.3 \times 10^4 \text{ M}^{-1} \text{ s}^{-1}$	Estimation functionally homologous protein fC3b and FB.
Disassociation rate for IgG:fC3b:FB	$k_{\text{IgGfC3bFB}}^-$	$15.5 \times 10^{-2} \text{ s}^{-1}$	
Binding rate for C3b and P	k_{C3bP}^+	$1.5 \times 10^5 \text{ M}^{-1} \text{ s}^{-1}$	[29]–[31], [36]
Disassociation rate for C3b:P	k_{C3bP}^-	$15.5 \times 10^{-5} \text{ s}^{-1}$	
Binding rate for fC3b:C4b and P	k_{fC3bC4bP}^+	$1.5 \times 10^5 \text{ M}^{-1} \text{ s}^{-1}$	[29]–[31]
Disassociation rate for fC3b:C4b:P	k_{fC3bC4bP}^-	$15.5 \times 10^{-5} \text{ s}^{-1}$	
Binding rate for C3b and FH	k_{C3bFH}^+	$1.1 \times 10^6 \text{ M}^{-1} \text{ s}^{-1}$	[29]–[31], [37]
Disassociation rate for C3b:FH	k_{C3bFH}^-	$5.9 \times 10^{-2} \text{ s}^{-1}$	
Binding rate for fC3b:Bb and FH	k_{fC3bBbFH}^+	$1.1 \times 10^6 \text{ M}^{-1} \text{ s}^{-1}$	Estimation functionally homologous protein C3b and FH.
Disassociation rate for fC3b:Bb:FH	k_{fC3bBbFH}^-	$5.9 \times 10^{-2} \text{ s}^{-1}$	
Binding rate for C3b:Bb:C3b and FH	$k_{\text{C3bBbC3bFH}}^+$	$1.1 \times 10^6 \text{ M}^{-1} \text{ s}^{-1}$	Estimation functionally homologous protein C3b and FH.
Disassociation rate for C3b:Bb:C3b:FH	$k_{\text{C3bBbC3bFH}}^-$	$5.9 \times 10^{-2} \text{ s}^{-1}$	
Binding rate for C4b and C4BP	k_{C4bC4BP}^+	$2.0 \times 10^5 \text{ M}^{-1} \text{ s}^{-1}$	[29]–[31], [38]–[40].
Disassociation rate for C4b:C4BP	k_{C4bC4BP}^-	$1.6 \times 10^{-2} \text{ s}^{-1}$	
Binding rate for C4b:C2a and C4BP	$k_{\text{C4bC2aC4BP}}^+$	$2.0 \times 10^5 \text{ M}^{-1} \text{ s}^{-1}$	Estimation functionally homologous protein C4b and C4BP.
Disassociation rate for C4b:C2a:C4BP	$k_{\text{C4bC2aC4BP}}^-$	$1.6 \times 10^{-2} \text{ s}^{-1}$	

Binding rate for C3b and CR1	k_{C3bCR1}^+	$4.4 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31], [37]
Disassociation rate for C3b:CR1	k_{C3bCR1}^-	$5.7 \times 10^{-2} \text{ s}^{-1}$	
Binding rate for C3b:Bb and CR1	$k_{C3bBbCR1}^+$	$4.4 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$	Estimation functionally homologous protein C3b and CR1.
Disassociation rate for C3b:Bb:CR1	$k_{C3bBbCR1}^-$	$5.7 \times 10^{-2} \text{ s}^{-1}$	
Binding rate for fC3b:Bb:C3b and CR1	$k_{fC3bBbC3bCR1}^+$	$9.8 \times 10^4 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31], [37]
Disassociation rate for fC3b:Bb:C3b:CR1	$k_{fC3bBbC3bCR1}^-$	$2.1 \times 10^{-3} \text{ s}^{-1}$	
Binding rate for fC3b:Bb and DAF	$k_{fC3bBbDAF}^+$	$1.4 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31], [41]
Disassociation rate for fC3b:Bb:DAF	$k_{fC3bBbDAF}^-$	$1.2 \times 10^{-3} \text{ s}^{-1}$	
Binding rate for C4b:C2a and DAF	$k_{C4bC2aDAF}^+$	$1.4 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	Estimation functionally homologous protein C3 convertase (fC3b:Bb) and DAF.
Disassociation rate for C4b:C2a:DAF	$k_{C4bC2aDAF}^-$	$1.2 \times 10^{-3} \text{ s}^{-1}$	
Binding rate for C4b:C2a:C3b and DAF	$k_{C4bC2aC3bDAF}^+$	$1.4 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	Estimation functionally homologous protein C3 convertase (C4b:C2a) and DAF.
Disassociation rate for C4b:C2a:C3b:DAF	$k_{C4bC2aC3bDAF}^-$	$1.2 \times 10^{-3} \text{ s}^{-1}$	
Binding rate for fC3b:Bb:C3b and DAF	$k_{fC3bBbC3bDAF}^+$	$1.4 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	Estimation functionally homologous protein C3 convertase (fC3b:Bb) and DAF.
Disassociation rate for fC3b:Bb:C3b:DAF	$k_{fC3bBbC3bDAF}^-$	$1.2 \times 10^{-3} \text{ s}^{-1}$	
Binding rate for C4b:C2a and CR1	$k_{C4bC2aCR1}^+$	$3.8 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31].
Disassociation rate for C4b:C2a:CR1	$k_{C4bC2aCR1}^-$	$4.2 \times 10^{-2} \text{ s}^{-1}$	
Binding rate for C3W:Bb and FH	$k_{C3WBbFH}^+$	$1.1 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$	Estimation functionally homologous protein C3b and FH.
Disassociation rate for C3W:Bb:FH	$k_{C3WBbFH}^-$	$5.9 \times 10^{-2} \text{ s}^{-1}$	
Binding rate for C5b and C6	k_{C5bC6}^+	$6.0 \times 10^4 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31], [38], [42]
Disassociation rate for C5b:C6	k_{C5bC6}^-	$9.0 \times 10^{-8} \text{ s}^{-1}$	

Binding rate for C5b:C6 and C7	$k_{C5bC6C7}^+$	$7.3 \times 10^5 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31], [38], [42]
Disassociation rate for C5b:C6:C7	$k_{C5bC6C7}^-$	$1.5 \times 10^{-7} \text{ s}^{-1}$	
Binding rate for C5b:C6:C7 and C8	$k_{C5bC6C7C8}^+$	$1.1 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31], [38], [42]
Disassociation rate for C5b:C6:C7:C8	$k_{C5bC6C7C8}^-$	$9.8 \times 10^{-7} \text{ s}^{-1}$	
Binding rate for C5b:C6:C7:C8 and C9	$k_{C5bC6C7C8C9}^+$	$2.8 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31], [38], [42]
Disassociation rate for C5b:C6:C7:C8:C9	$k_{C5bC6C7C8C9}^-$	$2.8 \times 10^{-6} \text{ s}^{-1}$	
Association of fC3b to H ₂ O	k_{fC3b}^+	$4.2 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31]
Binding rate for fC3b and IgG	$k_{IgGfC3b}^+$	$4.2 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31]
Activation rate of C1	k_{C1}^+	$2.1 \times 10^{-5} \text{ s}^{-1}$	[29]–[31]
Binding rate for C1 and inhibitor C1INH	$k_{C1C1INH}^+$	$4.3 \times 10^5 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31], [43]
Binding rate of C1INH and MASP1	$k_{C1INHMASP1}^+$	$6.3 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	[14], [29]–[31], [44]
Binding rate of C1INH and MASP2	$k_{C1INHMASP2}^+$	$2.2 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	[29]–[32]
Disassociation of initial C3-convertase C3W:Bb	k_{C3WBb}^-	$9.0 \times 10^{-3} \text{ s}^{-1}$	Estimation is based on the increases in decay rate of fC3b:Bb. The enzyme C3W:Bb is less active and less stable compared to C3bBb [29]–[31], [43].
Disassociation of C3-convertase fC3b:Bb	k_{fC3bBb}^-	$7.70 \times 10^{-3} \text{ s}^{-1}$	
Binding rate for C4b and C2	k_{C4bC2}^+	$1.6 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31], [34].
Disassociation rate for C4b:C2	k_{C4bC2}^-	$4.2 \times 10^{-3} \text{ s}^{-1}$	
Binding rate for fC3b:C4b and C2	$k_{fC3bC4bC2}^+$	$1.6 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31].
Disassociation rate for fC3b:C4b:C2	$k_{fC3bC4bC2}^-$	$4.2 \times 10^{-3} \text{ s}^{-1}$	

Binding rate for C4b and C2a	k_{C4bC2a}^+	$4.4 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	In Quasi steady-state approximation (QSSA), rapid equilibrium leads to $k_m \approx k_d$, QSSA leads to $k_m \approx \frac{k_{cat}}{k^+}$ [45]. $\frac{k_{cat}}{k_m} = 4.4 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$ [46] , $k^- = 5.8 \times 10^{-3} \text{ s}^{-1}$ [38].
Disassociation rate for C4b:C2a	k_{C4bC2a}^-	$5.8 \times 10^{-3} \text{ s}^{-1}$	
Binding rate for C4b:C2a and C3b	$k_{C4bC2aC3b}^+$	$1.5 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	In Quasi steady-state approximation (QSSA), rapid equilibrium leads to $k_m \approx k_d$, QSSA leads to $k_m \approx \frac{k_{cat}}{k^+}$ [45]. $\frac{k_{cat}}{k_m} = 1.5 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$ [46] , $k^- = 5.0 \times 10^{-3} \text{ s}^{-1}$ [30].
Disassociation of C5-convertase C4b:C2a:C3b	$k_{C4bC2aC3b}^-$	$5.0 \times 10^{-3} \text{ s}^{-1}$	
Binding rate for fC3b and C3b:FB	$k_{fC3bC3bFB}^+$	$21.3 \times 10^4 \text{ M}^{-1}\text{s}^{-1}$	Estimation functionally homologous protein fC3b and FB.
Disassociation of fC3b:C3b:FB	$k_{fC3bC3bFB}^-$	$15.5 \times 10^{-2} \text{ s}^{-1}$	
Binding rate for fC3b and C3b:FB	$k_{fC3bC4bFB}^+$	$21.3 \times 10^4 \text{ M}^{-1}\text{s}^{-1}$	Estimation functionally homologous protein fC3b and FB.
Disassociation of fC3b:C3b:FB	$k_{fC3bC4bFB}^-$	$15.5 \times 10^{-2} \text{ s}^{-1}$	
Decay of C3-convertase (fC3b:Bb) by down-regulator Factor H (FH)	$k_{dfC3bBbFH}^-$	$1.7 \times 10^{-2} \text{ s}^{-1}$	[47]
Binding rate for fC3b and C4b	$k_{fC3bC4b}^+$	$4.2 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$	[29]–[31]
Decay of initial C3-convertase (C3W:Bb) by inhibitor FH	$k_{dC3WBbFH}^-$	$1.7 \times 10^{-2} \text{ s}^{-1}$	It is assumed based on the decay of functionally homologous protein fC3b:Bb down-regulated by FH.
Decay of C3-convertase (fC3b:Bb) by inhibitor DAF	$k_{dfC3bBbDAF}^-$	$1.7 \times 10^{-2} \text{ s}^{-1}$	It is assumed based on the decay of functionally homologous protein fC3b:Bb down-regulated by FH.
Decay of C3-convertase (fC3b:Bb) by inhibitor CR1	$k_{dfC3bBbCR1}^-$	$1.7 \times 10^{-2} \text{ s}^{-1}$	It is assumed based on the decay of functionally homologous protein fC3b:Bb down-regulated by FH.
Disassociation of C3-convertase (fC3b:Bb) and properdin (P) complex (fC3b:Bb:P)	$k_{fC3bBbP}^-$	$7.7 \times 10^{-4} \text{ s}^{-1}$	[29]–[31], [48]

Disassociation of C5-convertase (C3b:Bb:C3b) and properdin (P) complex (C3b:Bb:C3b:P)	$k_{C3bBbC3bP}^-$	$5.7 \times 10^{-4} s^{-1}$	[30], [31], [48]
Decay of C5-convertase (C3b:Bb:C3b) by inhibitor FH	$k_{dC3bBbC3bFH}^-$	$1.7 \times 10^{-2} s^{-1}$	It is assumed based on the decay of functionally homologous protein fC3b:Bb down-regulated by FH.
Decay of C5-convertase (fC3b:Bb:C3b) by inhibitor CR1	$k_{dC3bBbC3bCR1}^-$	$1.7 \times 10^{-2} s^{-1}$	It is assumed based on the decay of functionally homologous protein fC3b:Bb down-regulated by FH.
Decay of C5-convertase (C3b:Bb:C3b) by inhibitor DAF on host cell	$k_{dC3bBbC3bDAF}^-$	$1.7 \times 10^{-2} s^{-1}$	It is assumed based on the decay of functionally homologous protein fC3b:Bb down-regulated by FH.
Decay of C5-convertase (C4b:C2a:C3b) by inhibitor CR1 on host cell	$k_{dC4bC2aC3bCR1}^-$	$1.7 \times 10^{-2} s^{-1}$	It is assumed based on the decay of functionally homologous protein fC3b:Bb down-regulated by FH.
Decay of C5-convertase (C4b:C2a:C3b) by inhibitor DAF on host cell	$k_{dC4bC2aC3bDAF}^-$	$1.7 \times 10^{-2} s^{-1}$	It is assumed based on the decay of functionally homologous protein fC3b:Bb down-regulated by FH.
Decay of C3-convertase (C4b:C2a) by down-regulator C4BP on host cell	$k_{dC4bC2aC4BP}^-$	$1.7 \times 10^{-2} s^{-1}$	It is assumed based on the decay of functionally homologous protein fC3b:Bb down-regulated by FH.
Decay of C3-convertase (C4b:C2a) by down-regulator CR1 on host cell	$k_{dC4bC2aCR1}^-$	$1.7 \times 10^{-2} s^{-1}$	It is assumed based on the decay of functionally homologous protein fC3b:Bb down-regulated by FH.
Decay of C3-convertase (C4b:C2a) by down-regulator CR1 on host cell	$k_{dC4bC2aDAF}^-$	$1.7 \times 10^{-2} s^{-1}$	It is assumed based on the decay of functionally homologous protein fC3b:Bb down-regulated by FH.
Decay of C5-convertase (C4b:C2a:C3b) by down-regulator CR1 on host cell	$k_{dC4bC2aC3bCR1}^-$	$1.7 \times 10^{-2} s^{-1}$	It is assumed based on the decay of functionally homologous protein C4b:C2a down-regulated by CR1.

Disassociation of C5-convertase C4b:C2a:C3b	$k_{C4bC2aC3b}^-$	$5.0 \times 10^{-3} s^{-1}$	[29]–[31], [46]
Binding rate for CR1 and fC3b:C4b	$k_{fC3bC4bCR1}^+$	$9.8 \times 10^4 M^{-1}s^{-1}$	[29]–[31], [49]
Disassociation rate for fC3b:C4b:CR1	$k_{fC3bC4bCR1}^-$	$2.1 \times 10^{-3} s^{-1}$	
Binding rate for fC3b:C4b and FH	$k_{fC3bC4bFH}^+$	$1.1 \times 10^6 M^{-1}s^{-1}$	Estimation functionally homologous protein fC3b and FH.
Disassociation rate for fC3b:C4b:FH	$k_{fC3bC4bFH}^-$	$5.9 \times 10^{-2} s^{-1}$	
Binding rate for CR1 and fC3b:C4b:C2a	$k_{fC3bC4bC2aCR1}^+$	$9.8 \times 10^4 M^{-1}s^{-1}$	Estimation functionally homologous protein fC3b:C4b and CR1.
Disassociation rate for fC3b:C4b:C2a:CR1	$k_{fC3bC4bC2aCR1}^-$	$2.1 \times 10^{-3} s^{-1}$	
Binding rate for fC3b:Bb and P	$k_{fC3bBbP}^+$	$1.5 \times 10^5 M^{-1}s^{-1}$	Estimation functionally homologous protein fC3b and P.
Disassociation rate for fC3b:Bb:P	$k_{fC3bBbP}^-$	$15.5 \times 10^{-5} s^{-1}$	
Binding rate for fC3b:Bb:C3b and P	$k_{fC3bBbC3bP}^+$	$1.5 \times 10^5 M^{-1}s^{-1}$	Estimation functionally homologous protein fC3b and P.
Disassociation rate for fC3b:Bb:C3b:P	$k_{fC3bBbC3bP}^-$	$15.5 \times 10^{-5} s^{-1}$	
Binding rate for fC3b:FB and P	$k_{fC3bFBP}^+$	$1.5 \times 10^5 M^{-1}s^{-1}$	Estimation functionally homologous protein fC3b and P.
Disassociation rate for fC3b:FB:P	$k_{fC3bFBP}^-$	$15.5 \times 10^{-5} s^{-1}$	
Binding rate for IgG:fC3b:FB and P	$k_{fC3bFBP}^+$	$1.5 \times 10^5 M^{-1}s^{-1}$	Estimation functionally homologous protein fC3b and P.
Disassociation rate for IgG:fC3b:FB:P	$k_{fC3bFBP}^-$	$15.5 \times 10^{-5} s^{-1}$	
Synthesis rate of FB	k_{sFB}^+	$0.013 \times 10^{-9} Ms^{-1}$	[50]
Degradation rate of FB	k_{dFB}^-	$5.5 \times 10^{-6} s^{-1}$	[50]
Synthesis rate of FH	k_{sFH}^+	$0.0117 \times 10^{-9} Ms^{-1}$	[50]
Degradation rate of FH	k_{dFH}^-	$3.7 \times 10^{-6} s^{-1}$	[50]

Synthesis rate of properdin (P)	k_{sP}^+	$1.17 \times 10^{-12} \text{ Ms}^{-1}$	[50]
Degradation rate of properdin (P)	k_{dP}^-	$2.7 \times 10^{-7} \text{ s}^{-1}$	[50]
Synthesis rate of C3	k_{sC3}^+	$0.053 \times 10^{-9} \text{ Ms}^{-1}$	[50]
Degradation rate of C3	k_{dC3}^-	$6.5 \times 10^{-6} \text{ s}^{-1}$	[50]
Hydrolysis of C3W	k_{C3W}^+	$4.5 \times 10^{-6} \text{ s}^{-1}$	[29]–[31]
Binding rate for MBL and MASP1	$k_{MBLMA SP1}^+$	$2.1 \times 10^5 \text{ M}^{-1} \text{ s}^{-1}$	[31], [51]
Disassociation rate for MBL:MASP1	$k_{MBLMA SP1}^-$	$6.8 \times 10^{-4} \text{ s}^{-1}$	
Binding rate for MBL and MASP2	$k_{MBLMA SP2}^+$	$2.3 \times 10^5 \text{ M}^{-1} \text{ s}^{-1}$	[31], [51]
Disassociation rate for MBL:MASP2	$k_{MBLMA SP2}^-$	$5.5 \times 10^{-4} \text{ s}^{-1}$	
Cleavage of C3 by initial C3-convertase (C3W:Bb)	k_{catC3}^{C3WBb} k_{mC3}^{C3WBb}	0.78 s^{-1} $11.6 \times 10^{-6} \text{ M}$	The C3b:Bb have a half-life ($t_{1/2}$) of $90 \pm 2 \text{ s}$ and $\frac{k_{cat}}{k_m}$ of $31.1 \times 10^4 \pm 0.8 \times 10^4 \text{ M}^{-1} \text{ s}^{-1}$. The C3b:Bb structurally/functionally homologous C3W:Bb [31] is found slightly less stable than C3b:Bb. It is known that C3W:Bb have $t_{1/2}$ of $77 \pm 3 \text{ s}$ and presented only half the activity of C3b:Bb ($\frac{k_{cat}}{k_m}$ is $16.3 \times 10^4 \pm 16.3 \times 10^4 \text{ M}^{-1} \text{ s}^{-1}$) [43]. Assumption k_m of C3W:Bb found from increasing k_m of C3b:Bb [31].
Cleavage of C3 by C3-convertase (C3b:Bb)	k_{catC3}^{C3bBb} k_{mC3}^{C3bBb}	1.78 s^{-1} $5.86 \times 10^{-6} \text{ M}$	[29]–[31], [43]
Cleavage of C3 by C3-convertase (C4b:C2a)	k_{catC3}^{C4b2a} k_{mC3}^{C4b2a}	3.17 s^{-1} $1.8 \times 10^{-6} \text{ M}$	[29]–[31], [38]
Cleavage of C5 by C3-convertase (C3b:Bb)	k_{catC5}^{C3bBb} k_{mC5}^{C3bBb}	$1.1 \times 10^{-2} \text{ s}^{-1}$ $24.0 \times 10^{-6} \text{ M}$	[29]–[31], [38]

Cleavage of C5 by C3-convertase (C4b:C2a)	k_{catC5}^{C4b2a} k_{mC5}^{C4b2a}	$2.2 \times 10^{-2} s^{-1}$ $8.9 \times 10^{-6} M$	[29]–[31], [38]
Cleavage of C5 by C5-convertase (C4b:C2a:C3b)	$k_{catC5}^{C4b2a3b}$ $k_{mC5}^{C4b2a3b}$	$2.0 \times 10^{-2} s^{-1}$ $5.1 \times 10^{-9} M$	[38]
Cleavage of C5 by C5-convertase (C3b:Bb:C3b)	$k_{catC5}^{C3bBbC3b}$ $k_{mC5}^{C3bBbC3b}$	$3.0 \times 10^{-3} s^{-1}$ $48.0 \times 10^{-9} M$	[29]–[31], [38]
Activation of the product/complex C3W:FB by Factor D (FD)	$k_{catC3WFB}^{FD}$ k_{mC3WFB}^{FD}	$5.0 s^{-1}$ $2.5 \times 10^{-6} M$	[29]–[31]
Down-regulation/inhibition of the complex fC3b:FB by negative regulator Factor I (FI)	$k_{catfC3bFB}^{FI}$ $k_{mfC3bFB}^{FI}$	$1.32 s^{-1}$ $2.52 \times 10^{-7} M$	It is assumed based on the FI inhibition of functionally homologous protein C3b:FB.
Down-regulation of the complex C3b:FB by inhibitor FI	$k_{catC3bFB}^{FI}$ k_{mC3bFB}^{FI}	$1.32 s^{-1}$ $2.52 \times 10^{-7} M$	[29]–[31]
Down-regulation of the complex C3W:FB by FI	$k_{catC3WFB}^{FI}$ k_{mC3WFB}^{FI}	$1.32 s^{-1}$ $2.52 \times 10^{-7} M$	It is assumed based on the FI inhibition of functionally homologous protein C3b:FB.
Down-regulation of the complex fC3b:C4b:FB by FI	$k_{catfC3bC4bFB}^{FI}$ $k_{mfC3bC4bFB}^{FI}$	$1.32 s^{-1}$ $2.52 \times 10^{-7} M$	It is assumed based on the FI inhibition of functionally homologous protein C3b:FB.
Up-regulation of the complex fC3b:FB by FD	$k_{catfC3bFB}^{FD}$ $k_{mfC3bFB}^{FD}$	$1.32 s^{-1}$ $2.52 \times 10^{-7} M$	Estimation structurally/functionally homologous protein C3W and Factor B [29]–[31], [34].

Inhibition of the complex fC3b:CR1 by FI	$k_{\text{catfC3bCR1}}^{\text{FI}}$ $k_{\text{mfC3bCR1}}^{\text{FI}}$	1.32 s^{-1} $2.52 \times 10^{-7} \text{ M}$	It is assumed based on the FI inhibition of functionally homologous protein C3b:FH.
Inhibition of the complex C3b:CR1 by FI	$k_{\text{catC3bCR1}}^{\text{FI}}$ $k_{\text{mC3bCR1}}^{\text{FI}}$	1.32 s^{-1} $2.52 \times 10^{-7} \text{ M}$	It is assumed based on the FI inhibition of functionally homologous protein C3b:FH.
Inhibition of the complex fC3b:C4b:CR1 by FI	$k_{\text{catfC3bC4bCR1}}^{\text{FI}}$ $k_{\text{mfC3bC4bCR1}}^{\text{FI}}$	1.32 s^{-1} $2.52 \times 10^{-7} \text{ M}$	It is assumed based on the FI inhibition of functionally homologous protein C3b:FH.
Activation of the complex fC3b:FB:P by FD	$k_{\text{catfC3bFBP}}^{\text{FD}}$ $k_{\text{mfC3bFBP}}^{\text{FD}}$	5.0 s^{-1} $2.5 \times 10^{-6} \text{ M}$	It is assumed based on the FD upregulation of functionally homologous protein C3W:FB.
Activation of the complex IgG:fC3b:FB by FD	$k_{\text{catIgGfC3bFB}}^{\text{FD}}$ $k_{\text{mIgGfC3bFB}}^{\text{FD}}$	5.0 s^{-1} $2.5 \times 10^{-6} \text{ M}$	It is assumed based on the FD upregulation of functionally homologous protein C3W/FB.
Activation of the complex IgG:fC3b:FB:P by FD	$k_{\text{catIgGfC3bFBP}}^{\text{FD}}$ $k_{\text{mIgGfC3bFBP}}^{\text{FD}}$	5.0 s^{-1} $2.5 \times 10^{-6} \text{ M}$	It is assumed based on the FD upregulation of functionally homologous protein C3W:FB.
Activation of the complex fC3b:C4b:FB by FD	$k_{\text{catfC3bC4bFB}}^{\text{FD}}$ $k_{\text{mfC3bC4bFB}}^{\text{FD}}$	5.0 s^{-1} $2.5 \times 10^{-6} \text{ M}$	It is assumed based on the FD upregulation of functionally homologous protein C3W:FB .
Activation of the complex C3b:FB by FD	$k_{\text{catC3bFB}}^{\text{FD}}$ $k_{\text{mC3bFB}}^{\text{FD}}$	5.0 s^{-1} $2.5 \times 10^{-6} \text{ M}$	It is assumed based on the FD upregulation of functionally homologous protein fC3b:FB [29]–[31].
Activation of the complex fC3b:C4b:P:FB by FD	$k_{\text{catfC3bC4bPFB}}^{\text{FD}}$ $k_{\text{mfC3bC4bPFB}}^{\text{FD}}$	5.0 s^{-1} $2.5 \times 10^{-6} \text{ M}$	It is assumed based on the FD upregulation of functionally homologous protein C3W:FB [29]–[31].

Cleavage rate of MASP fragment (MASP1) on C2 substrate	k_{catC2}^{MASP1} k_{mC2}^{MASP1}	0.1 s^{-1} $4.8 \times 10^{-6} \text{ M}$	[31], [52]
Cleavage rate of MASP fragment (MASP2) on C2 substrate	k_{catC2}^{MASP2} k_{mC2}^{MASP2}	5.6 s^{-1} $5.2 \times 10^{-6} \text{ M}$	[31], [53]
Cleavage rate of MASP fragment (MASP1) on C4 substrate	k_{catC4}^{MASP1} k_{mC4}^{MASP1}	$2.0 \times 10^{-3} \text{ s}^{-1}$ $5.2 \times 10^{-6} \text{ M}$	[31], [53]
Cleavage rate of MASP fragment (MASP2) on C4 substrate	k_{catC4}^{MASP2} k_{mC4}^{MASP2}	1.9 s^{-1} $8.5 \times 10^{-8} \text{ M}$	[31], [53]
Cleavage rate of C2 by C1	k_{catC2}^{C1} k_{mC2}^{C1}	5.1 s^{-1} $6.1 \times 10^{-6} \text{ M}$	[31], [53]
Cleavage rate of C4 by C1	k_{catC4}^{C1} k_{mC4}^{C1}	5.4 s^{-1} $6.10 \times 10^{-6} \text{ M}$	[31], [53]
Cleavage rate of fC3b:C3b:FB by FD	$k_{catfC3bC3bFB}^{FD}$ $k_{mfC3bC3bFB}^{FD}$	5.0 s^{-1} $2.5 \times 10^{-6} \text{ M}$	[29]–[31]
Binding rate for C3a and C3aR1 Disassociation rate for C3a:C3aR1	$k_{C3aC3aR1}^{+}$ $k_{C3aC3aR1}^{-}$	$2.6 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$ 0.1 s^{-1}	Disassociation constant (k_d) is $3.85 \times 10^{-9} \text{ M}$ [54]. Assume $k^{-} = 0.1 \text{ s}^{-1}$, Estimation for k^{+} by formula $k_d = \frac{k^{-}}{k^{+}}$.
Binding rate for C5a and C5aR1 Disassociation rate for C5a:C5aR1	$k_{C5aC5aR1}^{+}$ $k_{C5aC5aR1}^{-}$	$2.2 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$ 0.1 s^{-1}	Disassociation constant (k_d) for the product C5a:C5aR1 is $\approx 4.5 \times 10^{-9} \text{ M}$ and free energy of association (ΔG_{bind}) is $-13.6 \pm 4.1 \text{ kcalmol}^{-1}$ [55]. Assumed $k^{-} = 0.1 \text{ s}^{-1}$, estimated for k^{+} by formula $k_d = \frac{k^{-}}{k^{+}} = e^{\frac{\Delta G_{bind}}{RT}}$.

Inhibition of C1s by C1INH	$k_{C1INH C1s}^+$	$9.5 \times 10^4 \text{ M}^{-1} \text{ s}^{-1}$	C1 inhibitor binds to C1s [56].
Degradation rate of FD	k_{dFD}^-	$2.2 \times 10^{-4} \text{ s}^{-1}$	[57], [58]
Synthesis rate of FD	k_{sFD}^+	$1.3 \times 10^{-11} \text{ Ms}^{-1}$	[57]
Synthesis rate of C5	k_{sC5}^+	$2.2 \times 10^{-12} \text{ Ms}^{-1}$	[57]
Degradation rate of C5	k_{dC5}^-	$5.6 \times 10^{-6} \text{ s}^{-1}$	[57]
Synthesis rate of C6	k_{sC6}^+	$1.8 \times 10^{-10} \text{ Ms}^{-1}$	[57]
Degradation rate of C6	k_{dC6}^-	$2.9 \times 10^{-4} \text{ s}^{-1}$	[57]
Synthesis rate of C7	k_{sC7}^+	$1.9 \times 10^{-12} \text{ Ms}^{-1}$	[57]
Degradation rate of C7	k_{dC7}^-	$3.2 \times 10^{-6} \text{ s}^{-1}$	[57]
Synthesis rate of C8	k_{sC8}^+	$1.4 \times 10^{-12} \text{ Ms}^{-1}$	[57]
Degradation rate of C8	k_{dC8}^-	$4.01 \times 10^{-6} \text{ s}^{-1}$	[57]
Synthesis rate of C9	k_{sC9}^+	$3.4 \times 10^{-12} \text{ Ms}^{-1}$	[57]
Degradation rate of C9	k_{dC9}^-	$4.01 \times 10^{-6} \text{ s}^{-1}$	[57]
Degradation rate of Ba	k_{dBa}^-	$7.7 \times 10^{-5} \text{ s}^{-1}$	[57]
Degradation rate of Bb	k_{dBb}^-	$7.7 \times 10^{-5} \text{ s}^{-1}$	[57]
Degradation rate of C3W	k_{dC3W}^-	$4.01 \times 10^{-6} \text{ s}^{-1}$	[57]
Degradation rate of C3WFB	k_{dC3WFB}^-	$4.01 \times 10^{-6} \text{ s}^{-1}$	[57]
Degradation rate of C3WBb	k_{dC3WBb}^-	$4.01 \times 10^{-6} \text{ s}^{-1}$	[57]
Degradation rate of fC3b	k_{dfC3b}^-	$4.01 \times 10^{-6} \text{ s}^{-1}$	[57]

Degradation rate of fC3bFB	$k_{dfC3bFB}^-$	$4.01 \times 10^{-6} s^{-1}$	[57]
Degradation rate of fC3bBb	$k_{dfC3bBb}^-$	$4.01 \times 10^{-6} s^{-1}$	[57]
Degradation rate of C5b	k_{dC5b}^-	$5.0 \times 10^{-3} s^{-1}$	[57], [59]
Degradation rate of C5a	k_{dC5a}^-	$1.15 \times 10^{-2} s^{-1}$	[57], [60]
Degradation rate of C3b	k_{dC3b}^-	$4.01 \times 10^{-6} s^{-1}$	[57]
Degradation rate of C3a	k_{dC3a}^-	$1.15 \times 10^{-2} s^{-1}$	[57]
Binding rate for C1q and gC1qR Disassociation rate for C1q:gC1qR	$k_{C1qgC1qR}^+$ $k_{C1qC1qR}^-$	$4.0 \times 10^2 M^{-1}s^{-1}$ $0.1 s^{-1}$	C1q binds to gC1q-receptor (gC1qR) with an affinity ($k_d = 250 \times 10^{-6}$) [61][50]. The disassociation rate (k^{-1}) is unknown, assumed a small value for $k^{-1} = 0.1 s^{-1}$. Using the formula $k_d = \frac{k^-}{k^+}$, determined k^+ .
Inhibition of MAC by CD59	$k_{MACCD59}^+$	$1.0 \times 10^6 M^{-1}s^{-1}$	The terminal complement complex (TCC), C5b-9 (MAC) bind to CD59 glycoprotein, also known as MAC-inhibitory protein (MAC-IP) [29].

Table S1c. The hemostatic system and complement system cross-talk KCs

Meaning	Rate constant	Value adopted	Source
Inhibition of F12a by C1INH	$k_{C1INHf12a}^+$	$3.6 \times 10^3 M^{-1}s^{-1}$	C1INH suppresses F12a [62], [63].
Inhibition of KAL by C1INH	$k_{C1INHkAL}^+$	$1.7 \times 10^4 M^{-1}s^{-1}$	C1INH suppresses KAL [62], [63]. Association rate of C1INH and KLK [64].
Inhibition of F2a by FH	k_{FHF2a}^+	$3.367 \times 10^7 M^{-1}s^{-1}$	For FH and F2a the disassociation constant (k_d) is measured 29.9nM. Binding affinity of FH and F2a range (3.1 - 200 nM) [65]. Estimation for association rate (k_{FHF2a}^+) based on k_d .

Inhibition of F1a by FH	k_{FHF1a}^+	$2.62 \times 10^7 \text{ M}^{-1}\text{s}^{-1}$	FH and F1a disassociation constant (k_d) is measured 38.2nM. Binding affinity of FH and F2a range (3.1 - 200 nM) [65]. Estimation for association rate (k_{FHF1a}^+) based on k_d .
Cleavage of C5 by Pn	$k_{\text{catC5}}^{\text{Pn}}$ $k_{\text{mC5}}^{\text{Pn}}$	0.056 s^{-1} 2.4×10^{-6}	Pn can cleaves C5 generating C5a with a catalytic efficiency ($\frac{k_{\text{cat}}}{k_m}$) of $2.3 \times 10^4 \text{ M}^{-1}\text{s}^{-1}$ [66], [67].
Cleavage of C5 by F2a	$k_{\text{catC5}}^{\text{F2a}}$ $k_{\text{mC5}}^{\text{F2a}}$	0.56 s^{-1} 2.4×10^{-6}	F2a is less effective than Pn in cleaving C5 to generates C5a [66]. We could estimate catalytic efficiency ($\frac{k_{\text{cat}}}{k_m}$) of $2.3 \pm 0.6 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$ based on Pn catalytic efficiency.
Cleavage of C3 by Pn	$k_{\text{catC3}}^{\text{Pn}}$ $k_{\text{mC3}}^{\text{Pn}}$	0.056 s^{-1} 2.4×10^{-6}	Pn can cleaves C3. The cleavage of C3 by Pn is assumed based on functionally homologous C5.
Cleavage of C3 by F2a	$k_{\text{catC3}}^{\text{F2a}}$ $k_{\text{mC3}}^{\text{F2a}}$	0.56 s^{-1} 2.4×10^{-6}	The cleavage of C3 by F2a is assumed based on functionally homologous C5.
Cleavage of C3 by F10a	$k_{\text{catC3}}^{\text{F10a}}$ $k_{\text{mC3}}^{\text{F10a}}$	0.056 s^{-1} 2.4×10^{-5}	F10a is less effective than Pn in cleaving C3 to generates C3a [66]. We could estimate catalytic efficiency ($\frac{k_{\text{cat}}}{k_m}$) of $2.3 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$ based on Pn catalytic efficiency.
Cleavage of C5 by F10a	$k_{\text{catC5}}^{\text{F10a}}$ $k_{\text{mC5}}^{\text{F10a}}$	0.056 s^{-1} 2.4×10^{-5}	F10a is less effective than Pn in cleaving C5 to generates C5a, respectively [66]. We could estimate catalytic efficiency ($\frac{k_{\text{cat}}}{k_m}$) of $2.3 \pm 0.6 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$ based on Pn catalytic efficiency.
Production of TF by C5a	$k_{\text{catTF}}^{\text{C5a}}$	$0.01 \times 10^{-3} \text{ s}^{-1}$	C5a can increase the activity and expression of TF [68]. C5a stimulate the expression of TF on neutrophils via C5aR [62], [69]. Estimated a small value.
Production of TF by C3a	$k_{\text{catTF}}^{\text{C3a}}$	$0.01 \times 10^{-3} \text{ s}^{-1}$	C3a increases the expression of TF [68]. Assumption based on C3a functional homologous C5a.
Inhibition of F11a by CIINH	$k_{\text{C1INH F11a}}^+$	$1.8 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	CIINH binds with F11a and inhibit it [2], [3].
Inhibition of F2a by CIINH	$k_{\text{C1INH F2a}}^+$	$1.0 \times 10^2 \text{ M}^{-1}\text{s}^{-1}$	C1-INH can inhibit F2a. Assumption.

Inhibition of Plasmin by C1INH	$k_{C1INHPlasmin}^+$	$5.5 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	Kinetic analysis for inhibition experiment with Pn, generated an association rate for plasma C1INH [70].
Generation of F1 by IL6	k_{catF1}^{IL6}	$0.01 \times 10^{-6} \text{ s}^{-1}$	IL6 induces the expression of F1. IL-6 correlated positively with F1 concentration [71]. Assume a small random value for generation of F1 by IL6.
Activation rate of F2 by MASP1	k_{catF2}^{MASP1}	$141.0 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	MASP1 activates and cleaves F2 to F2a [63], [72], [73]. Activation of F2 wildtype by MASP1 computed via kinetic analysis [74].
Activation rate of F2 by MASP2	k_{catF2}^{MASP2}	$141.0 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	MASP2 activates and cleaves F2 into F2a [63], [73]. Activation rate of F2 by MASP2 estimated based on functionally homologous MASP1.
Activation rate of F1 by MASP1	k_{catF1}^{MASP1}	$141.0 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	MASP1 activate F1 to F1a [63], [72], [73]. Assumed based on the activation rate of F2 by MASP1.
Activation rate of F1 by MASP2	k_{catF1}^{MASP2}	$141.0 \times 10^3 \text{ M}^{-1}\text{s}^{-1}$	MASP1 activate F1 to F1a [63], [72], [73]. Activation rate of F1 by MASP2 estimated based on functionally homologous MASP1.
Generation of F2 by MAC	k_{catF2}^{MAC}	0.1 s^{-1}	Estimation for MAC (C5b-C9) can generate F2 [62].
Activation of C1r by F12a	k_{catC1r}^{F12a}	$1.0 \times 10^2 \text{ M}^{-1}\text{s}^{-1}$	F12a can activate the C1r [62], [68], [75]. Assumed a small value for the activation of C1r by F12a.
Association rate for gC1qR and F12 Disassociation rate for gC1qR:F12	$k_{gC1qRF12}^+$ $k_{gC1qRF12}^-$	$8.3 \times 10^{13} \text{ M}^{-1}\text{s}^{-1}$ 0.1 s^{-1}	gC1qR binds to F12 with disassociation constant $k_d = 120 \times 10^{-9} \text{ M}$ [76]. Assumed a small value for $k^{-1} = 0.1 \text{ s}^{-1}$. Using formula $k_d = \frac{k^-}{k^+}$, estimated for k^+ .
Production of F12a by gC1qR:F12	$k_{catF12a}^{gC1qRF12}$	0.1 s^{-1}	The complex of gC1qR and F12 can generates F12a [77].
Association rate for gC1qR and HK Disassociation rate for gC1qR:HK	$k_{gC1qRHK}^+$ $k_{gC1qRHK}^-$	$5.3 \times 10^{11} \text{ M}^{-1}\text{s}^{-1}$ 0.1 s^{-1}	gC1q-receptor (gC1qR) binds to HK with disassociation constant $k_d = 1.9 \times 10^{-9} \text{ M}$ [76]. Assumed a small value for $k^{-1} = 0.1 \text{ s}^{-1}$. Using formula $k_d = \frac{k^-}{k^+}$, estimated k^+ .
Production of BK by gC1qR:HK	$k_{catBK}^{gC1qRHK}$	0.1 s^{-1}	The product of gC1qR and HK can release BK [77]. The generation rate is unknown, estimated a small value for it.

Cleavage of FB by KAL	k_{catFB}^{KAL} k_{mFB}^{KAL}	$0.01\ s^{-1}$ $1.0 \times 10^{-12}\ M$	KAL can cleaves FB [68], [77]. Assumed small values for the catalytic rate constant and Michaelis constant. FB co-exist with hydrolyzed C3 (C3W) and fC3b.
Cleavage of C3 by KAL	k_{catC3}^{KAL} k_{mC3}^{KAL}	$0.01\ s^{-1}$ $1.0 \times 10^{-12}\ M$	KAL can cleaves C3 [68], [77]. Assumed small values for the catalytic rate constant and Michaelis constant.
Cleavage of C5 by KAL	k_{catC5}^{KAL} k_{mC5}^{KAL}	$0.01\ s^{-1}$ $1.0 \times 10^{-12}\ M$	Kallikrein can cleaves C5 [68]. Assumed small values for the catalytic rate constant and Michaelis constant.
Degradation rate of IL-6	k_{dIL6}^-	$0.01s^{-1}$	Assumption.
Production of IL6 by BK:B2R	k_{IL6}^{BKB2R}	$0.1\ s^{-1}$	BK via B2R can stimulate the production of IL6 [78] Estimation for the production rate of IL6 by BK and B2R complex.
Production of IL6 by C3a:C3aR	$k_{IL6}^{C3aC3aR1}$	$0.1\ s^{-1}$	C3a via C3aR1 can induce IL6 [79]. Estimation for the production rate of IL6 by C3a and C3aR complex.
Production of IL6 by C5a:C5aR	$k_{IL6}^{C5aC5aR1}$	$0.1\ s^{-1}$	C5a via C5aR1 can induce interleukin 6 (IL6) [79]. Estimation for production rate of IL6 by C5a and C5aR complex.
Activation of Pro-FD by MASP1	$k_{catPro-FD}^{MASP1}$	$3.9 \times 10^3\ M^{-1}s^{-1}$	Activation and cleavage of Pro-FD by MASP1 that can be converted into active FD [80].
Activation of Pro-FD by MASP2	$k_{catPro-FD}^{MASP2}$	$7.2 \times 10^3\ M^{-1}s^{-1}$	Activation and cleavage of Pro-FD by MASP2 that can be converted into active FD [80].
Activation of Pro-FD by F2a	$k_{catPro-FD}^{F2a}$	$4.6 \times 10^2\ M^{-1}s^{-1}$	The activation rates of pro-FD by thrombin (F2a) [80].
Cleavage of TAFI by MASP1	$k_{catTAFI}^{MASP1}$ k_{mTAFI}^{MASP1}	$0.1\ s^{-1}$ $3.9 \times 10^{-8}\ M$	MASP-1 directly activates/cleaves TAFI [72], [77].

Table S1d. The hemostatic and complement systems entities and SARS-CoV-2 structure proteins interaction KCs

Meaning	Rate constant	Value adopted	Source
Association rate for gC1qR and CoV2S	$k_{gC1qRCoV2S}^+$	$1.1 \times 10^{10} M^{-1} s^{-1}$	Binding of S protein of SARS-CoV-2 (termed as CoV2S) on the surface of wild-type gC1qR of classical pathway of the complement system. The binding affinity is $k_d = 91 \times 10^{-12} M$ [81]. The disassociation rate (k^-) is unknown, assume $k^- = 1 s^{-1}$. Using $k_d = \frac{k^-}{k^+}$ determined k^+ .
Association rate for gC1qR and CoV2N	$k_{C1qRCoV2N}^+$	$1.7 \times 10^5 M^{-1} s^{-1}$	Binding of N protein of SARS-CoV-2 (termed as CoV2N) on the surface of wild-type gC1qR of classical pathway of the complement system. The binding affinity is $k_d = 6 \times 10^{-6} M$ [81]. Assume $k^- = 1 s^{-1}$. Using $k_d = \frac{k^-}{k^+}$ determined k^+ .
Association rate for gC1qR and CoV2M	$k_{C1qRCoV2M}^+$	$2.4 \times 10^9 M^{-1} s^{-1}$	Binding of the M protein of SARS-CoV-2 (termed as CoV2M) on the surface of wild-type C1qR of classical pathway of the complement system. The binding affinity is $k_d = 410 \times 10^{-12} M$ [81]. Assume $k^- = 1 s^{-1}$. Using $k_d = \frac{k^-}{k^+}$ determined k^+ .
Association rate for gC1qR and CoV2E	$k_{C1qRCoV2E}^+$	$2.4 \times 10^9 M^{-1} s^{-1}$	Binding of the E protein of SARS-CoV-2 (termed as CoV2E) on the surface of wild-type C1qR classical pathway of the complement system. The binding affinity $k_d = 410 \times 10^{-12} M$ [81]. Assume $k^- = 1 s^{-1}$. Using $k_d = \frac{k^-}{k^+}$ determined k^+ .
Association rate for F12 and CoV2S	$k_{CoV2SF12}^+$	$1.0 \times 10^6 M^{-1} s^{-1}$	S protein of SARS-CoV-2 (termed as CoV2S) binds to inactive coagulation factor F12 of contact pathway that is intrinsic pathway of the coagulation cascade [81]. Assumed for binding rate of F12 and CoV2S.
Association rate for F12 and CoV2N	$k_{CoV2NF12}^+$	$1.0 \times 10^4 M^{-1} s^{-1}$	N protein of SARS-CoV-2 (termed as CoV2N) binds to coagulation factor F12 [81]. Assumption for binding rate of F12 and CoV2N.
Association rate for F12 and CoV2M	$k_{CoV2MF12}^+$	$1.0 \times 10^4 M^{-1} s^{-1}$	M protein of SARS-CoV-2 (termed as CoV2M) binds to coagulation factor F12 [81]. Assumption for binding rate of F12 and CoV2M.

Association rate for F12 and CoV2E	k_{CoV2EF12}^+	$1.0 \times 10^4 M^{-1} s^{-1}$	E protein of SARS-CoV-2 (termed as CoV2E) binds to coagulation factor F12 [81]. Assumption for binding rate of F12 and CoV2S.
Association rate for HK and CoV2S	k_{HKCoV2S}^+	$1.0 \times 10^3 M^{-1} s^{-1}$	S protein of SARS-CoV-2 (termed as CoV2S) binds to HK of the system KKS [81]. Assumption for binding rate.
Association rate for HK and CoV2N	k_{HKCoV2N}^+	$1.0 \times 10^3 M^{-1} s^{-1}$	N protein of SARS-CoV-2 (termed as CoV2N) binds to HK of the system KKS [81]. Assumption for binding rate.
Association rate for HK and CoV2M	k_{HKCoV2M}^+	$1.0 \times 10^3 M^{-1} s^{-1}$	M protein of SARS-CoV-2 (termed as CoV2M) binds to HK of the system KKS [81]. Assumption for binding rate.
Association rate for HK and CoV2E	k_{HKCoV2E}^+	$1.0 \times 10^3 M^{-1} s^{-1}$	E protein of SARS-CoV-2 (termed as CoV2E) binds to HK of the system KKS [81]. Assumption for binding rate.
Association rate for MASP2 by CoV2N protein	$k_{\text{MASP2CoV2N}}^+$	$6.9 \times 10^4 M^{-1} s^{-1}$	The N protein of SARS-CoV-2 (termed as CoV2N) can cleaved/ activate MASP2 [82]. $k_{\text{cat}} = 3.64 s^{-1}$ and $k_m = 5.437 \times 10^{-5} M$. Assume a small value for disassociation rate $k^- = 0.1 s^{-1}$. Using the formula $k^+ = \frac{k^- + k_{\text{cat}}}{k_m}$, determined k^+ .
Binding rate for IgG and CoV2S Disassociation rate for the complex IgG/CoV2S	k_{CoV2SIgG}^+ k_{CoV2SIgG}^-	$2.14 \times 10^5 M^{-1} s^{-1}$ $1.70 \times 10^{-2} s^{-1}$	Association rate for IgG via Fab (ID-1F4) bind with spike (S) protein of SARS-CoV-2 [83].
Association rate of CoV2S/IgG and C1q	$k_{\text{CoV2SIgGC1q}}^+$	$2.3 \times 10^5 M^{-1} s^{-1}$	S protein of SARS-CoV-2 binding by antibody IgG mediated C1q activate complement system through classical pathway [44]. An antibody IgG1 subclass antibodies Rituximab (RTX) and Ofatumumab (OFA) bind with C1q. Association rates of C1q and RTX, C1q and OFA are $5.2 \times 10^4 M^{-1} s^{-1}$ and $2.3 \times 10^5 M^{-1} s^{-1}$ [84]. Estimation for IgG based on structurally/ functionally homologous IgG1 subclass.
Cleavage rate of CoV2S by F10a	$k_{\text{catCoV2S}}^{\text{F10a}}$ $k_{\text{mCoV2S}}^{\text{F10a}}$	$1.79 \times 10^{-1} s^{-1}$ $4.035 \times 10^{-5} M$	Cleavage rate of S protein by active coagulation factor F10a [85].
Cleavage rate of CoV2S by F2a	$k_{\text{catCoV2S}}^{\text{F2a}}$ $k_{\text{mCoV2S}}^{\text{F2a}}$	$5.20 \times 10^{-2} s^{-1}$ $16.34 \times 10^{-6} M$	Cleavage rate of S protein by activated coagulation factor F2a [85].

Table S1e. The Drug-Target interaction KCs

Meaning	Rate constant	Value adopted	Source
Association of Heparin to C2	k_{HepC2}^+	$4.13 \times 10^3 M^{-1} s^{-1}$	[86]
Disassociation of Heparin:C2	k_{HepC2}^-	$1.32 \times 10^{-3} s^{-1}$	
Association of Heparin to C3	k_{HepC3}^+	$1.83 \times 10^4 M^{-1} s^{-1}$	[86]
Disassociation of Heparin:C3	k_{HepC3}^-	$5.73 \times 10^{-4} s^{-1}$	
Association of Heparin to C4	k_{HepC4}^+	$9.64 \times 10^4 M^{-1} s^{-1}$	[86]
Disassociation of Heparin:C4	k_{HepC4}^-	$3.46 \times 10^{-3} s^{-1}$	
Association of Heparin to C5	k_{HepC5}^+	$2.43 \times 10^5 M^{-1} s^{-1}$	[86]
Disassociation of Heparin:C5	k_{HepC5}^-	$1.35 \times 10^{-3} s^{-1}$	
Association of Heparin to C6	k_{HepC6}^+	$1.72 \times 10^4 M^{-1} s^{-1}$	[86]
Disassociation of Heparin:C6	k_{HepC6}^-	$5.58 \times 10^{-4} s^{-1}$	
Association of Heparin to C7	k_{HepC7}^+	$6.25 \times 10^4 M^{-1} s^{-1}$	[86]
Disassociation of Heparin:C7	k_{HepC7}^-	$9.60 \times 10^{-4} s^{-1}$	
Association of Heparin to C8	k_{HepC8}^+	$4.17 \times 10^4 M^{-1} s^{-1}$	[86]
Disassociation of Heparin:C8	k_{HepC8}^-	$5.74 \times 10^{-3} s^{-1}$	
Association of Heparin to C9	k_{HepC9}^+	$3.43 \times 10^4 M^{-1} s^{-1}$	[86]
Disassociation of Heparin:C9	k_{HepC9}^-	$4.51 \times 10^{-3} s^{-1}$	

Activation of AT3 by Heparin	k_{HepAT3}^+	$1 \times 10^2 M^{-1} s^{-1}$	Heparin as potentiator of AT3 [87]. Assumption.
Inhibition of C5aR1 by Avdoralimab	k_{AvdC5aR1}^+	$1.0 \times 10^3 M^{-1} s^{-1}$	Avdoralimab (IPH5401) is a therapeutic antibody that specifically binds and blocks C5a receptors (C5aR1). It is safely used in COVID-19 patients [88]. Assumption.
Inhibition of tPA by Tranexamic acid (TXA)	k_{TXAtPA}^+	$1.0 \times 10^2 M^{-1} s^{-1}$	Assumption.

Abbreviations

MBL, Mannose binding lectin; MASP1 and MASP2 stand for MBL associated Mannan-binding lectin serine protease MASP-1 and MASP-2; C2, Complement component 2; C2a, Complement component 2a; C2b, Complement component 2a; C3aR1, C3a anaphylatoxin Receptor 1; C4BP, Complement component 4 binding protein; CR1, Complement receptor 1; DAF, Decay Accelerating Factor; FB, FD, FH and FI stand for Complement factor B, D, H and I, respectively; P, Properdin; IgG, Immunoglobulin G; C1q, C1r, C1s Complement components C1q, C1r, C1s; C1, Activated Complement component 1; Ba, cleavage fragment of FB; Bb, cleavage fragment of FB; MAC, Membrane attack complex (MAC); IL6, Interleukin 6; F1 stand for Coagulation factor 1 (Fibrinogen); F1a, Coagulation factor 1a (Fibrin); F2, Coagulation factor 2 (Pro-Thrombin); F2a, Coagulation factor 2a (Thrombin); F5, Coagulation factor 5 (Labile factor, Proaccelerin); F5a, Activated coagulation factor 5; F7, Coagulation Factor 7 (Proconvertin); F7a, Activated coagulation factor 7; F8, Coagulation factor 8 (Antihemophilic factor A); F8a, Activated coagulation factor 8; F9, Coagulation factor 9 (Antihemophilic factor B); F9a, Activated coagulation factor 9; F10, Coagulation factor 10 (Thrombokinase); F10, Activated coagulation factor 10; F11, Coagulation factor 11 (Plasma thromboplastin antecedent); F11, Activated coagulation factor 11; F12, Coagulation factor 12 (Hageman factor); F12a, Activated coagulation factor 12; KAL, Kallikrein; P-KAL, Pre-Kallikrein; Pn, Plasmin; Pg, Plasminogen; TF, Tissue factor (Tissue thromboplastin); TFPI, Tissue factor pathway inhibitor; TAFI, Thrombin-activatable fibrinolysis inhibitor; TAFIa, Activated Thrombin-activatable fibrinolysis inhibitor; BK, Bradykinin; C3a and C5a stand for complement anaphylatoxins;

HK, High-molecular-weight kininogen; tPA, Tissue-type plasminogen activator; A2M, Association rate of Alpha-2 macroglobulin; AT3, Anti-Thrombin 3 (Serpine); PAI-1, Plasminogen activator inhibitor-1; B2R, B2-receptor.

S stand for Spike protein; N, Nucleocapsid; M, Membrane; E, Envelope;

KKS, Kallikrein-kinin system.

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