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## Supplementary Material for

### **Strategies for containing Ebola in West Africa**

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## S1 Model

We developed a continuous-time stochastic compartment model that takes into account Ebola transmission within and between the community, hospitals, and funerals (Fig S1). We track the number of people susceptible ( $S$ ), latently infected but not yet infectious ( $E$ ), infectious ( $J$  &  $I$ ), deceased victims who may transmit during funerals ( $F$ ), deceased victims who are hygienically buried ( $D$ ), and individuals recovered with immunity ( $R$ ). The  $J$  compartments ensure that the duration of infection for hospitalized Ebola cases is the same whether they were infected in the community or hospital. To distinguish different levels of exposure to Ebola, we stratified by the general community (subscript  $G$ ), hospital patients ( $H$ ) and healthcare workers ( $W$ ), people attending funerals ( $F$ ) and people in quarantine or isolation ( $Q$ ).

Figure S1 shows the per-capita transition hazards for each transition; see Table S1 for parameter definitions and values. The forces of infection are

$$\begin{aligned}
 \lambda_G &= \phi_G \beta_I \frac{I_G}{N_G}, \\
 \lambda_F &= \omega \beta_I \frac{F_T}{F_T + \frac{N_T}{e\gamma_F}}, \\
 \lambda_H &= \beta_I \frac{J_H + J_W + (1 - i_H)(I_H + I_W)}{N_H + N_W}, \text{ and} \\
 \lambda_W &= \phi_W \beta_W \frac{J_H + J_W + (1 - i_H)(I_H + I_W)}{N_H + N_W},
 \end{aligned} \tag{S1}$$

where  $\omega$  was derived from the odds ratio of funeral transmission ( $\hat{\omega}$ ) and the prevalence data from a previous Ebola outbreak in Kikwit (11).

The transition rate to funerals is

$$f_{GF} = \left[ \frac{N_T}{e} + (1 - p_G)(1 - \theta)\gamma_{DG}I_G + (1 - p_H)(1 - i_H)\gamma_{DH}(I_H + I_W) \right] \frac{M_F}{N_G - S_F}, \quad (\text{S2})$$

which accounts for non-Ebola funerals ( $N_T/e$ ) and Ebola funerals. The transition rate to hospitals is

$$f_{GH} = (M_H + 1)h + M_H\theta\gamma_H \frac{I_G}{N_G}, \quad (\text{S3})$$

which accounts for non-Ebola patients ( $h$ ), people visiting their hospitalized relatives ( $M_H$ ) with and without Ebola. The transition rate to an isolation unit is

$$\kappa = \begin{cases} C \left[ 1 - \left( 1 - \frac{\beta_I}{C} \right)^{1/\gamma_H} \right] \phi_C \gamma_H \frac{I_G + J_H + J_W}{E_G} & \text{if } E_G > 0, \\ 0 & \text{otherwise.} \end{cases} \quad (\text{S4})$$

The number of Ebola funerals is  $F_T = F_G + F_H + F_W$ , The size of the general population is  $N_G = S_G + S_F + E_G + I_G + R_G$ . The number of people in hospitals is  $N_H = S_H + E_H + J_H + I_H + R_H$ . The number of healthcare workers is  $N_W = S_W + E_W + J_W + I_W + R_W$ . The number of isolated people is  $N_Q = E_Q + J_Q + I_Q + R_Q$ . The total number of people is  $N_T = N_G + N_H + N_W + N_Q$ . All other per-capita transition rates are constant with respect to the number of people in each compartment. The

following are composites of the basic model parameters:

$$\begin{aligned}
\gamma_{FG} &= (1 - \theta)(1 - p_G)\gamma_{DG}, \\
\bar{\gamma}_{RG} &= (1 - \theta)\gamma_{RG}, \\
\bar{\gamma}_{DG} &= (1 - \theta)p_G\gamma_{DG}, \\
\gamma_{FH} &= (1 - i_H)(1 - p_H)\gamma_{DH}, \\
\bar{\gamma}_{DH} &= [i_H + (1 - i_H)p_H]\gamma_{DH}.
\end{aligned}
\tag{S5}$$

Thus, susceptible people ( $S$ ) who become infected move into latent compartments ( $E$ ). After the incubation period, latently infected people ( $E$ ) move to either the infectious compartments ( $I_G, J_H, J_W$ ) if they are symptomatic (fraction  $\epsilon$ ) or the removed compartments ( $R$ ) if asymptomatic ( $1 - \epsilon$ ). We assume that all infections are symptomatic ( $\epsilon = 1$ ), then vary this parameter in the sensitivity analysis. Infectious people in the general community can be hospitalized, moving into  $I_H$ . People hospitalized when they become symptomatic ( $J_H, J_W$ ) move into the second infectious class ( $I_H, I_W$ ) at the same rate to ensure that people who became infected in the general community and are later hospitalized have the same total duration of infection as those who became infected at the hospital. Infectious people either recover with immunity ( $R$ ), or die. Prior to intervention, we assume that they are buried in a funeral ceremony ( $F$ ) when they die, from which funeral attendees may be infected. After the funeral, the deceased Ebola victim is buried ( $D$ ) and no longer contributes to transmission.

Susceptible people from the general community ( $S_G$ ) can be hospitalized ( $S_H$ ) for

	<b>Definition</b>	<b>Value</b>	<b>Ref</b>
$N_T(0)$	Population of Liberia	4.09 M	(32)
$S_W(0)$	Healthcare workers	$0.00028N_T(0)$	(33) <sup>*,†</sup>
$e$	Life expectancy	62 y	(34)
$1/\alpha$	Incubation period	9.5 d	(9) <sup>†</sup>
$1/\gamma_{DG}$	Duration from symptom onset to death	7.9 d	(9) <sup>†</sup>
$1/\gamma_{RG}$	Duration from symptom onset to recovery	9 d	(35) <sup>†</sup>
$1/\gamma_H$	Duration between symptom onset and hospitalization	4.9 d	(9) <sup>†</sup>
$1/\gamma_{DH}$	Duration from hospitalization to death	$1/\gamma_{DG} - 1/\gamma_H$	
$1/\gamma_{RH}$	Duration from hospitalization to recovery	$1/\gamma_{RG} - 1/\gamma_H$	
$1/\gamma_F$	Duration from death to burial	2 d	(20)
$1/f_{FG}$	Duration of funeral ceremonies	$1/\gamma_F$	
$h$	Per-capita hospitalization rate for non-Ebola reasons	$62131/N_T(0) \text{ y}^{-1}$	(36) <sup>†</sup>
$1/f_{HG}$	Duration of non-Ebola hospital stay	7 d	(36) <sup>†</sup>
$M_F$	Number of close contacts at funeral	4	(10) <sup>†</sup>
$M_H$	Hospital visitors	1	†
$\hat{\omega}$	Odds of funeral transmission with respect to general community	1.2	(11) <sup>†</sup>
$\epsilon$	Fractions of infections symptomatic	100%	†
$I_G(0)$	Number of cases on 8 June 2014	23 (95% CI : 22 – 24)	‡
$\theta$	Proportion of infectious cases hospitalized	52% (95% CI : 49 – 55%)	‡ †
$\beta_I$	Transmission parameter for the general community and hospital patients	$0.074$ (95% CI : $0.072 - 0.076$ ) $\text{d}^{-1}$	‡
$\beta_W$	Transmission parameter for healthcare workers	$0.27$ (95% CI : $0.25 - 0.30$ ) $\text{d}^{-1}$	‡
$\phi_G$	Reduction in community transmissibility	0–100%	§
$\phi_W$	Reduction in transmissibility to healthcare workers	0–100%	§
$i_H$	Proportion of infectious hospital patients and healthcare workers isolated	0–100%	§
$p_H$	Proportion of Ebola deaths in the hospital and healthcare workers that are hygienically buried	0–100%	§
$p_G$	Proportion of Ebola deaths in the general community that are hygienically buried	0–100%	§
$\phi_C$	Proportion of contacts isolated	0–100%	§
$C$	Number of contacts traced	6	(12)

\*: Includes physicians, nurses and midwives.

†: Varied in sensitivity analysis.

‡: Estimated by calibration to data.

§: Intervention parameter.

Table S1: Model parameters.

reasons other than Ebola (e.g. child birth). They may leave the hospital and return to the general community ( $S_G$ ) without contracting Ebola in the hospital. However, if they contract Ebola in the hospital, they remain in the hospital ( $J_H$ ). A proportion  $\theta$  of people who acquire infection in the general community ( $I_F$ ) are then hospitalized ( $I_H$ ). Similarly, an individual from the community ( $S_G$ ) can attend a funeral ( $S_F$ ) and then return to the general community as susceptible ( $S_G$ ) or latently infected ( $E_G$ ).

Precautions to reduce transmission to healthcare workers include the provision of personal protective equipment, thorough disinfection of facilities with chlorine, safe disposal of non-reusable supplies and infectious waste, and overall reduced contact with Ebola patients (37). We modeled this approach by reducing the force of infection to healthcare workers and term this strategy ‘reduction of transmission to healthcare workers’.

As of August 8, 2014, the government of Liberia launched a large scale quarantine operation, named ‘White Shield’, which included *cordon sanitaire* checkpoints to restrict the movement of people from Ebola-affected zones, as well as a national curfew to reduce community exposure (38). We have incorporated these measures aimed at limiting community contacts into our model by reducing the force of infection in the community, and term this strategy ‘community transmission reduction’.

Case isolation has been achieved by limiting the treatment clinics exclusively to Ebola patients, thereby eliminating transmission to patients hospitalized for other reasons. Within these clinics, isolation precautions are taken to reduce transmission. We modeled isolation by moving the fraction of successfully isolated patients out of

the transmission cycle and term this strategy ‘case isolation’.

In addition to the clinics, the Liberian Ministry of Health and Social Welfare (MoHSW) has implemented contact tracing, which consists of interviewing patients for possible contacts and subsequent quarantining. From these contact tracing procedures, the MoHSW has determined the average number of close contacts of Ebola cases to be 6. In the model, these contacts are quarantined at home for 21 days (21), the maximum duration of the incubation period, with food and water delivered daily, and their health monitored for early symptoms. At the first sign of symptoms, the individual is transported to a clinic for treatment and case isolation. We term this strategy ‘contact tracing and quarantine’. Should any quarantined Ebola patient die, we assume that they are hygienically buried.

Hygienic burial practices minimize transmission through decontamination of the body, including spraying the cadaver with a chlorine solution and placing the cadaver into a plastic body bag, which is then further disinfected (37). Burial teams who handle disinfection and transport of the bodies to grave sites are clothed in personal protective equipment (37). In our model, we distinguished between victims traditionally buried (F) and those hygienically buried (D), and quantify the extent of successful implementation of hygienic burial as the proportion of deceased victims entering F versus D. We term this strategy, ‘hygienic burial’.

We captured the behavioral change in the community as a result of Ebola outbreak by halting both the hospitalization of patients for reasons other than Ebola and the visitation of hospitalized patients, before initiating our interventions.

We quantify these interventions as follows:

**Reducing general-community transmission** Community transmission is reduced by the factor  $1 - \phi_G$ .

**Reducing transmission to healthcare workers** Transmission to healthcare workers is reduced by the factor  $1 - \phi_W$ .

**Contact tracing** For new cases  $(I_G, J_H, J_W)$ ,  $C$  contacts are listed to be traced and a proportion  $\phi_C$  are isolated if they become infected (subscript  $Q$  compartments). Transmission from infectious people in isolation is assumed to be prevented, but imperfect prevention of transmission is incorporated by varying the proportion of cases successfully isolated. People who die from Ebola in isolation are assumed to be buried hygienically.

**Isolation of hospitalized cases** A proportion  $i_H$  of hospitalized symptomatic cases  $(I_H, I_W)$  are isolated, preventing transmission.

**Hygienic burial of hospital deaths** A proportion  $p_H$  of deaths of hospitalized cases  $(I_H, I_W)$  are buried without a traditional funeral.

**Hygienic burial of non-hospital deaths** A proportion  $p_G$  of deaths of non-hospitalized cases  $(I_G)$  are buried without a traditional funeral.

The model incorporated stochasticity such that the number of individuals that move from one epidemiological or location class to another was sampled from a Poisson distribution, the rate of which was determined by the current number of people in each class over time. This stochasticity allowed the model to capture uncertainty in the number of new cases that arises in part from stochastic fluctuations and



comprises multiple spatial chains of cases that accrue to comprise the full epidemic trajectory.

The stochastic model was simulation by Gillespie's tau-leap method (39), with a time step of one day. The initial conditions for each simulation (at  $t = 0$ , 8 June 2014) were  $I_G(0)$ ,  $S_W(0)$  and  $N_T(0)$  as in Table S1,  $S_H(0) = 5S_W(0)$ ,  $S_F(0) = 0$ ,  $S_G(0) = N_T(0) - S_H(0) - S_W(0)$ , and all other compartments equal to 0. Any model interventions were assumed to begin at  $t = 104$  d (20 September 2014).

## S2 The calculation of $R_0$

The next-generation method (40, 41, Section S3) yields

$$R_0 = \frac{1}{2} \left[ R_0^{GG} + R_0^{HH} + \sqrt{(R_0^{GG} - R_0^{HH})^2 + 4R_0^{GH}R_0^{HG}} \right]. \quad (\text{S6})$$

The number of new cases in  $G$  (i.e. into  $E_G$ ) produced by a current new case in  $G$  (i.e. from  $E_G$ ) is

$$R_0^{GG} = \tau_G \delta_G \frac{S_G}{N_T} + \tau_F (\mu_G + \eta_G \mu_H) \delta_F \frac{S_F}{N_T}. \quad (\text{S7})$$

which includes direct transmission from infectious people,

$$R_0^{GGI} = \tau_G \delta_G \frac{S_G}{N_T}, \quad (\text{S8})$$

and transmission from deceased victims, whether they die in  $I_G$  ( $\mu_G$ ) or in  $I_H$  ( $\eta_G \mu_H$ ),

$$R_0^{GGF} = \tau_F (\mu_G + \eta_G \mu_H) \delta_F \frac{S_F}{N_T}. \quad (\text{S9})$$

The number of new cases in  $H$  and  $W$  (i.e.  $E_H$  and  $E_W$ ) produced by a current new case in  $H$  or  $W$  ( $E_H$  or  $E_W$ ) is

$$R_0^{HH} = (\delta_{HJ} + \delta_{HI}) \left( \tau_H \frac{S_H}{N_T} + \tau_W \frac{S_W}{N_T} \right), \quad (\text{S10})$$

which occurs by direct transmission, either to a hospital patient

$$R_0^{HHH} = \tau_H (\delta_{HJ} + \delta_{HI}) \frac{S_H}{N_T} \quad (\text{S11})$$

or to a healthcare worker

$$R_0^{HHW} = \tau_W (\delta_{HJ} + \delta_{HI}) \frac{S_W}{N_T}. \quad (\text{S12})$$

The number of new cases in  $G$  from a current new case in  $H$  or  $W$  is

$$R_0^{GH} = \tau_F \mu_H \delta_F \frac{S_F}{N_T}, \quad (\text{S13})$$

which occurs by funeral transmission. The number of new cases in  $H$  and  $W$  from a current new case in  $G$  is

$$R_0^{HG} = \eta_G \delta_{HI} \left( \tau_H \frac{S_H}{N_T} + \tau_W \frac{S_W}{N_T} \right), \quad (\text{S14})$$

which requires that the person in  $G$  be hospitalized ( $\eta_G$ ), and occurs by direct transmission, either to a hospital patient

$$R_0^{HGH} = \tau_H \eta_G \delta_{HI} \frac{S_H}{N_T} \quad (\text{S15})$$

or to a healthcare worker

$$R_0^{HGW} = \tau_W \eta_G \delta_{HI} \frac{S_W}{N_T}. \quad (\text{S16})$$

The effective transmission rate of infectious people in the general population to susceptible people in the general population is

$$\tau_G = \epsilon \beta_I \frac{N_T}{N_G}. \quad (\text{S17})$$

The effective transmission rate from infectious people in the hospital ( $I_H$  or  $I_W$ ) to susceptible hospital patients is

$$\tau_H = \epsilon \beta_I \frac{N_T}{N_H + N_W}. \quad (\text{S18})$$

The effective transmission rate from infectious people in the hospital ( $I_H$  or  $I_W$ ) to susceptible healthcare workers is

$$\tau_W = \epsilon \beta_W \frac{N_T}{N_H + N_W}. \quad (\text{S19})$$

The effective transmission rate of funerals to susceptible people at funerals is

$$\tau_F = \epsilon\omega\beta_I e\gamma_F. \quad (\text{S20})$$

The duration that the body of a deceased victim spends infectious in the general community is

$$\delta_G = \frac{1}{\theta\gamma_H + (1 - \theta)(\gamma_{DG} + \gamma_{RG})}. \quad (\text{S21})$$

the duration that a hospital patient or hospital worker spends in the first infectious ( $J$ ) classes is

$$\delta_{HJ} = \frac{1}{\gamma_H}. \quad (\text{S22})$$

The duration that a person spends in the second hospital infectious ( $I$ ) classes is

$$\delta_{HI} = \frac{1}{\gamma_{DH} + \gamma_{RH}}. \quad (\text{S23})$$

The duration that an infected body is infectious is

$$\delta_F = \frac{1}{\gamma_F}. \quad (\text{S24})$$

The case mortality in  $I_G$  is

$$\mu_G = \frac{(1 - \theta)\gamma_{DG}}{\theta\gamma_H + (1 - \theta)(\gamma_{DG} + \gamma_{RG})}. \quad (\text{S25})$$

The case mortality in  $I_H$  &  $I_W$  is

$$\mu_H = \frac{\gamma_{DH}}{\gamma_{DH} + \gamma_{RH}}. \quad (\text{S26})$$

The case hospitalization in  $I_G$  is

$$\eta_G = \frac{\theta\gamma_H}{\theta\gamma_H + (1 - \theta)(\gamma_{DG} + \gamma_{RG})}. \quad (\text{S27})$$

### S3 Next-generation matrix

At the disease-free equilibrium, the flow in and out of  $S_G$ ,  $S_F$ , and  $S_H$  are balanced:

$$\begin{aligned} f_{GF}S_G &= f_{FG}S_F, \text{ and} \\ f_{GH}S_G &= f_{HG}S_H, \end{aligned} \quad (\text{S28})$$

where

$$\begin{aligned} f_{GF} &= \frac{M_F N_T}{e S_G}, \\ f_{GH} &= (M_H + 1)h, \text{ and} \\ N_T &= S_G + S_F + S_H + S_W. \end{aligned} \quad (\text{S29})$$

Therefore

$$\begin{aligned}
S_G &= \frac{\left(1 - \frac{M_F}{ef_{FG}}\right) - \frac{S_W}{N_T}}{1 + \frac{(M_H+1)h}{f_{HG}}} N_T, \\
S_F &= \frac{M_F}{ef_{FG}} N_T, \text{ and} \\
S_H &= \frac{(M_H + 1)h}{f_{HG}} \frac{\left(1 - \frac{M_F}{ef_{FG}}\right) - \frac{S_W}{N_T}}{1 + \frac{(M_H+1)h}{f_{HG}}} N_T.
\end{aligned} \tag{S30}$$

Following van den Driessche and Watmough (41), with no interventions ( $\phi_G = \phi_W = i_H = p_H = p_G = \phi_C = 0$ ), the infected compartments are

$$\mathbf{x} = \left(E_G, I_G, F_G, E_H, J_H, I_H, F_H, E_W, J_W, I_W, F_W\right)^T, \tag{S31}$$

and the disease-free equilibrium has

$$\mathbf{x}_0 = \left(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0\right)^T. \tag{S32}$$

New infections occur at the rates

$$\mathcal{F} = \left[\lambda_G S_G + \lambda_F S_F, 0, 0, \lambda_H S_H, 0, 0, 0, \lambda_W S_W, 0, 0, 0\right]^T, \tag{S33}$$

and state transitions occur at rates:

$$\mathcal{V} = \begin{bmatrix} \alpha E_G \\ -\epsilon\alpha E_G + [\theta\gamma_H + (1-\theta)(\gamma_{DG} + \gamma_{RG})] I_G \\ -(1-\theta)\gamma_{DG} I_G + \gamma_F F_G \\ \alpha E_H \\ -\epsilon\alpha E_H + \gamma_H J_H \\ -\gamma_H J_H - \theta\gamma_H I_G + (\gamma_{DH} + \gamma_{RH}) I_H \\ -\gamma_{DH} I_H + \gamma_F F_H \\ \alpha E_W \\ -\epsilon\alpha E_W + \gamma_H J_W \\ -\gamma_H J_W + (\gamma_{DH} + \gamma_{RH}) I_W \\ -\gamma_{DH} I_W + \gamma_F F_W \end{bmatrix}. \quad (\text{S34})$$

The derivatives of these at the disease-free equilibrium are

$$\mathbf{F} = \begin{bmatrix} \mathbf{F}_{GG} & \mathbf{F}_{GH} & \mathbf{F}_{GW} \\ \mathbf{0} & \mathbf{F}_{HH} & \mathbf{F}_{HW} \\ \mathbf{0} & \mathbf{F}_{WH} & \mathbf{F}_{WW} \end{bmatrix}, \text{ and } \mathbf{V} = \begin{bmatrix} \mathbf{V}_{GG} & \mathbf{0} & \mathbf{0} \\ \mathbf{V}_{HG} & \mathbf{V}_{HH} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{V}_{WW} \end{bmatrix}, \quad (\text{S35})$$

with the new infection submatrices

$$\begin{aligned}
\mathbf{F}_{\mathbf{G}\mathbf{G}} &= \begin{bmatrix} 0 & \beta_I \frac{S_G}{N_G} & \omega \beta_I e \gamma_F \frac{S_F}{N_G} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \\
\mathbf{F}_{\mathbf{G}\mathbf{H}} = \mathbf{F}_{\mathbf{G}\mathbf{W}} &= \begin{bmatrix} 0 & 0 & 0 & \omega \beta_I e \gamma_F \frac{S_F}{N_G} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \\
\mathbf{F}_{\mathbf{H}\mathbf{H}} = \mathbf{F}_{\mathbf{H}\mathbf{W}} &= \begin{bmatrix} 0 & \beta_I \frac{S_H}{N_H+N_W} & \beta_I \frac{S_H}{N_H+N_W} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \text{ and} \\
\mathbf{F}_{\mathbf{W}\mathbf{H}} = \mathbf{F}_{\mathbf{W}\mathbf{W}} &= \begin{bmatrix} 0 & \beta_W \frac{S_W}{N_H+N_W} & \beta_W \frac{S_W}{N_H+N_W} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},
\end{aligned} \tag{S36}$$



and the state transition submatrices

$$\begin{aligned}
\mathbf{V}_{\mathbf{GG}} &= \begin{bmatrix} \alpha & 0 & 0 \\ -\alpha\epsilon & \theta\gamma_H + (1-\theta)(\gamma_{DG} + \gamma_{RG}) & 0 \\ 0 & -(1-\theta)\gamma_{DG} & \gamma_F \end{bmatrix}, \\
\mathbf{V}_{\mathbf{HG}} &= \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & -\theta\gamma_H & 0 \\ 0 & 0 & 0 \end{bmatrix}, \text{ and} \\
\mathbf{V}_{\mathbf{HH}} = \mathbf{V}_{\mathbf{WW}} &= \begin{bmatrix} \alpha & 0 & 0 & 0 \\ -\alpha\epsilon & \gamma_H & 0 & 0 \\ 0 & -\gamma_H & \gamma_{DH} + \gamma_{RH} & 0 \\ 0 & 0 & -\gamma_{DH} & \gamma_F \end{bmatrix}.
\end{aligned} \tag{S37}$$

The next-generation matrix is

$$\mathbf{G} = \mathbf{FV}^{-1} = \begin{bmatrix} \mathbf{G}_{\mathbf{GG}} & \mathbf{G}_{\mathbf{GH}} & \mathbf{G}_{\mathbf{GW}} \\ \mathbf{G}_{\mathbf{HG}} & \mathbf{G}_{\mathbf{HH}} & \mathbf{G}_{\mathbf{HW}} \\ \mathbf{G}_{\mathbf{WG}} & \mathbf{G}_{\mathbf{WH}} & \mathbf{G}_{\mathbf{WW}} \end{bmatrix}, \tag{S38}$$

with the submatrices

$$\begin{aligned}
\mathbf{G}_{GG} &= \mathbf{F}_{GG} \mathbf{V}_{GG}^{-1} - \mathbf{F}_{GH} \mathbf{V}_{HH}^{-1} \mathbf{V}_{HG} \mathbf{V}_{GG}^{-1}, \\
\mathbf{G}_{GH} &= \mathbf{G}_{GW} = \mathbf{F}_{GH} \mathbf{V}_{HH}^{-1}, \\
\mathbf{G}_{HG} &= -\mathbf{F}_{HH} \mathbf{V}_{HH}^{-1} \mathbf{V}_{HG} \mathbf{V}_{GG}^{-1}, \\
\mathbf{G}_{HH} &= \mathbf{G}_{HW} = \mathbf{F}_{HH} \mathbf{V}_{HH}^{-1}, \\
\mathbf{G}_{WG} &= -\mathbf{F}_{WH} \mathbf{V}_{HH}^{-1} \mathbf{V}_{HG} \mathbf{V}_{GG}^{-1}, \text{ and} \\
\mathbf{G}_{WH} &= \mathbf{G}_{WW} = \mathbf{F}_{WH} \mathbf{V}_{HH}^{-1}.
\end{aligned} \tag{S39}$$

$R_0$  is the largest eigenvalue of  $\mathbf{G}$ . Only the first rows of submatrices of  $\mathbf{F}$  and  $\mathbf{G}$  are non-zero. Thus, rows 2, 3, 5, 6, 7, 9, 10, and 11 of  $\mathbf{G}$  are all zero, the eigenvalues of  $\mathbf{G}$  are 0 with multiplicity 6 and the eigenvalues of the matrix

$$\mathbf{G}_1 = \begin{bmatrix} g_{11}^{GG} & g_{11}^{GH} & g_{11}^{GW} \\ g_{11}^{HG} & g_{11}^{HH} & g_{11}^{HW} \\ g_{11}^{WG} & g_{11}^{WH} & g_{11}^{WW} \end{bmatrix}, \tag{S40}$$

where  $g_{11}^{XY}$  is the 1,1th entry of the submatrix  $\mathbf{G}_{XY}$ . The last two columns of  $\mathbf{G}_1$  are equal since  $\mathbf{G}_{GH} = \mathbf{G}_{GW}$ ,  $\mathbf{G}_{HH} = \mathbf{G}_{HW}$ , and  $\mathbf{G}_{WH} = \mathbf{G}_{WW}$ , so the eigenvalues of  $\mathbf{G}_1$  are 0 and the eigenvalues of

$$\mathbf{G}_2 = \begin{bmatrix} g_{11}^{GG} & g_{11}^{GH} \\ g_{11}^{HG} + g_{11}^{WG} & g_{11}^{HH} + g_{11}^{WH} \end{bmatrix}. \tag{S41}$$

$\mathbf{G}_2$  (and thus  $\mathbf{G}$ ) has largest eigenvalue

$$R_0 = \frac{1}{2} \left[ R_0^{GG} + R_0^{HH} + \sqrt{(R_0^{GG} - R_0^{HH})^2 + 4R_0^{GH}R_0^{HG}} \right], \quad (\text{S42})$$

with

$$\begin{aligned} R_0^{GG} &= g_{11}^{GG}, \\ R_0^{HH} &= g_{11}^{HH} + g_{11}^{WH}, \\ R_0^{GH} &= g_{11}^{GH}, \text{ and} \\ R_0^{HG} &= g_{11}^{HG} + g_{11}^{WG}. \end{aligned} \quad (\text{S43})$$

Computing the 1, 1 entries of the submatrices of  $\mathbf{G}$  from equations (S39) yields S6–S27.

Without case isolation ( $C = 0$ ), a similar calculation shows that the intervention reproduction number is

$$R = \frac{1}{2} \left[ R^{GG} + R^{HH} + \sqrt{(R^{GG} - R^{HH})^2 + 4R^{GH}R^{HG}} \right], \quad (\text{S44})$$

with

$$\begin{aligned}
R^{GG} &= R^{GGI} + R^{GGF}, \\
R^{GGI} &= \phi_G \tau_G \delta_G \frac{S_G}{N_T}, \\
R^{GGF} &= \tau_F [(1 - p_G) \mu_G + (1 - i_H)(1 - p_H) \eta_G \mu_H] \delta_F \frac{S_F}{N_T}, \\
R^{HH} &= R^{HHH} + R^{HHW}, \\
R^{HHH} &= \tau_H [\delta_{HJ} + (1 - i_H) \delta_{HI}] \frac{S_H}{N_T}, \\
R^{HHW} &= \phi_W \tau_W [\delta_{HJ} + (1 - i_H) \delta_{HI}] \frac{S_W}{N_T}, \\
R^{GH} &= (1 - i_H)(1 - p_H) \tau_F \mu_H \delta_F \frac{S_F}{N_T}, \\
R^{HG} &= R^{HGH} + R^{HGW}, \\
R^{HGH} &= (1 - i_H) \tau_H \eta_G \delta_{HI} \frac{S_H}{N_T}, \text{ and} \\
R^{HGW} &= (1 - i_H) \phi_W \tau_W \eta_G \delta_{HI} \frac{S_W}{N_T},
\end{aligned} \tag{S45}$$

where the  $\tau$ 's,  $\delta$ 's,  $\mu$ 's, and  $\eta_G$  are as in S17–S27.

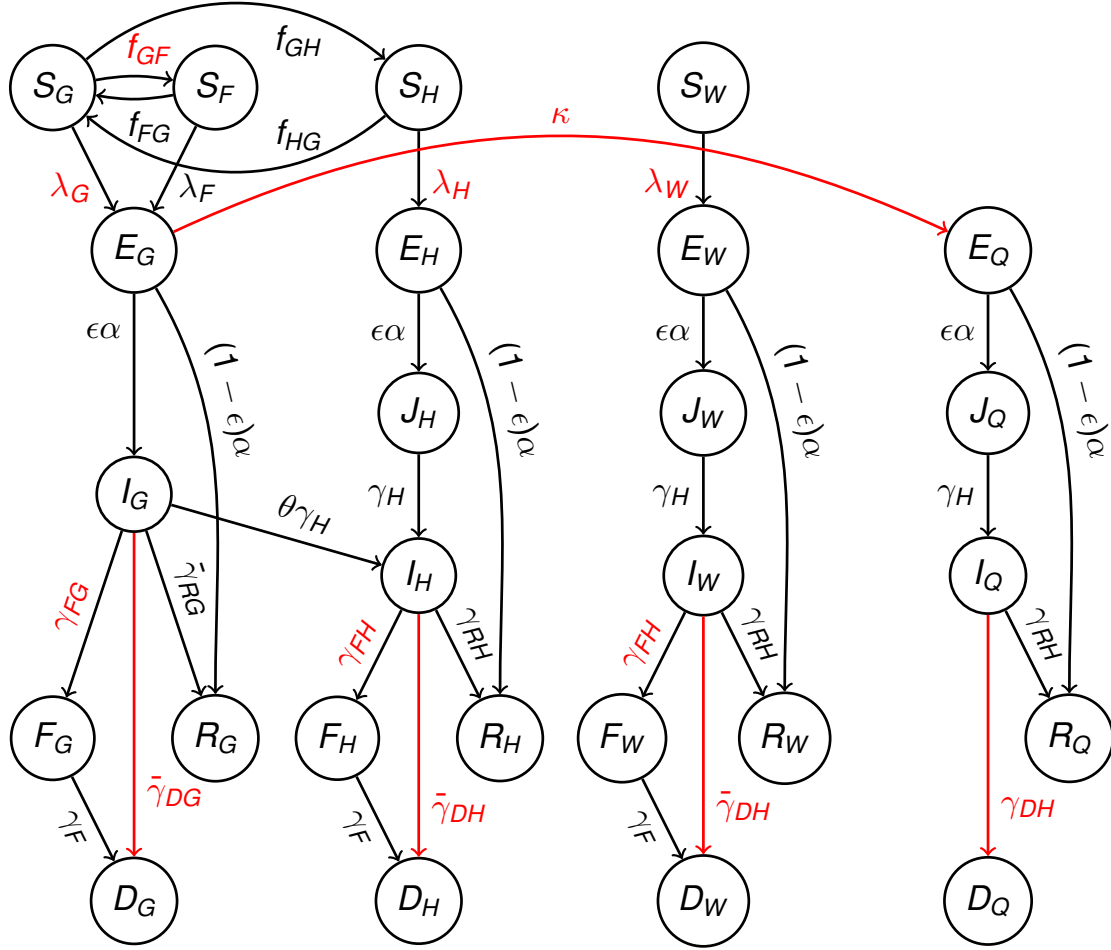


Figure S1: Model description. Our continuous-time stochastic model of Ebola transmission between and within the community, hospitals, and funerals (SOM). We tracked the number of people who are susceptible (S); latently infected (E); infectious (J & I); deceased Ebola victims buried in traditional West African funeral customs (F); buried Ebola victims (D); and recovered with immunity (R). We also distinguished between individuals who have different levels of exposure to Ebola based on whether they are part of the general community (subscript G), hospitalized for a reason other than Ebola (H), healthcare workers (W), attending a funeral ceremony (F) or quarantined (Q). This diagram shows the per-capita transition rates between compartments, with red labels indicating rates that can be impacted by interventions and red arrows indicating rates that are entirely due to interventions. The J compartments ensure that the duration of infection for hospitalized Ebola victims is the same whether they were infected in the community or the hospital. The forces of infection are functions of the number of infectious people, as are the transition rates  $f_{GF}$  to funerals,  $f_{GH}$  to hospitalization and to quarantine (30). Table S1 specifies parameter definitions and values.

## S4 Model fitting

We used weighted least squares to fit the model to case data. The objective function,  $f = \sum_{ij} w_{ij}(d_{ij} - m_{ij})^2$  was minimized over all data sets  $i$ , all time points  $j$ , for data points  $d_{ij}$  and model points  $m_{ij}$ . Consistent with the assumption that the all cumulative case counts are Poisson distributed,  $w_{ij} = \frac{1}{d_{ij}+1}$ .

We fit our model to data from the Liberian Ministry of Health and Social Welfare situation reports from 8 June to 7 August, 2014 inclusive, and estimated the transmission parameters  $(\beta_I, \beta_W)$ , hospitalization rate  $(\theta)$ , and the index case at the beginning of the epidemic  $(I_G(0))$ . We parameterized  $\alpha$ ,  $\gamma_H$ , and  $\gamma_{DG}$  from estimates from the current outbreak and parameterized  $\gamma_{RG}$  and  $\omega$  from estimates from the previous outbreaks Table S1, fitting our model concurrently to cumulative counts of 1) non-healthcare worker incidence, 2) fatalities, 3) healthcare worker incidence, and 4) hospital admissions. We used all suspected, probable, and confirmed cases reported in the situation reports.

To fit our model to data, we converted our event-based stochastic model to a discrete-time difference equation model such that any set of parameter values maps to a single epidemic trajectory over time. We evaluated the difference equation model, fitted the output to the data, and calculated the best-fit estimates of the parameters  $\beta_I$ ,  $\beta_W$ ,  $\theta$ , and  $I_G(0)$ , by minimizing the weighted least squares difference between the model output and the data with the Quasi-Newton algorithm. The confidence intervals for the parameters were generated by sampling from a multivariate normal distribution with covariance matrix given by the inverse of the Hessian matrix estimated during the model fitting.

We tested the robustness of our model calibration procedure by using both unbounded non-derivative (Simplex) and unbounded derivative (Quasi-Newton) optimization routines. We used a range of feasible initial conditions to ensure robustness of the optimization. Both optimization routines over all initial conditions converged to the same estimated parameter values. Although these searches are not exhaustive, and thus do not constitute formal proof of identifiability, they strongly suggest that our model is identifiable.

## **S5 Model implementation**

The model was coded and the results plotted using Matlab R2014a (The Mathworks, Natick), with `fminunc` and `fminsearch` used for the derivative and non-derivative based optimization routines, respectively.

## **S6 Impact of under-reporting on model predictions**

If both hospital and community cases are under-reported by up to 40%, the combination of nosocomial intervention that achieve a 90% reduction in transmission to healthcare workers and hygienic burial of 90% of hospital deaths is sufficient to control the epidemic (Fig S2a). Over 40% under-reporting, it becomes necessary to extend interventions into the community with either community hygienic burial (Fig S2b) or case isolation with contact-tracing and quarantining (Fig S2c). When the under-reporting rate in the community approaches 40%, control strategies must employ hygienic burial of both deceased hospitalized cases and deceased community cases to curtail the epidemic.



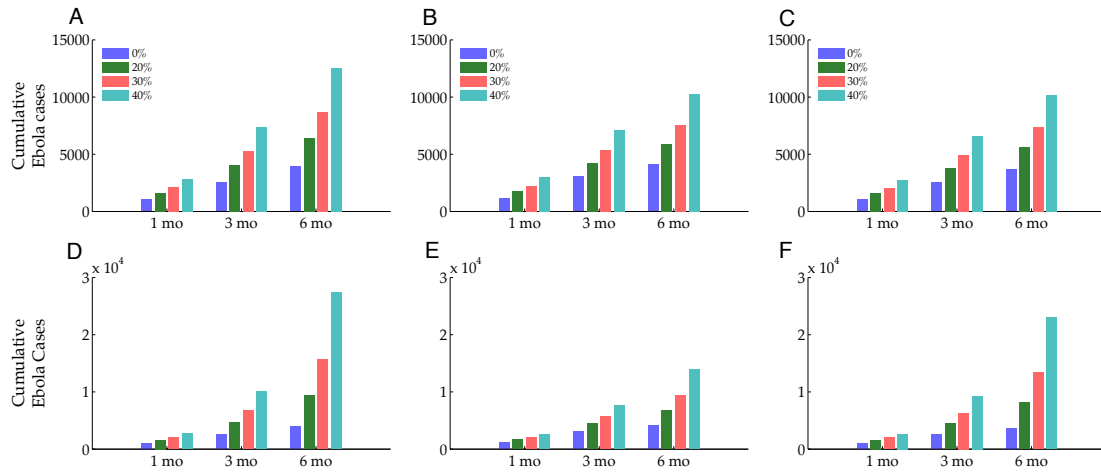


Figure S2: We varied the levels of under-reporting of Ebola cases and deaths in both community and hospital (A,B,C) as well as only in community (D,E,F). We quantified the impact of under-reporting on effectiveness of three intervention strategies: A,D) 90% reduction in transmission to healthcare workers and 90% successful hygienic burial of hospital cases, B,E) Successful hygienic burial of 80% of hospital and 30% of community cases, and C,F) Case isolation of 80% of hospital cases with concurrent contact tracing and isolation of 50% of infected contacts.

## **S7 Model Predictions for Montserrado County, Liberia**

Our estimated transmission parameters from fitting our model to Montserrado county data, suggest that there is less community transmission but more per capita transmission to healthcare workers in Montserrado county than in the country as a whole (Fig S3). While our results suggest that the control interventions are more effective in curtailing the outbreak in Montserrado county compared to national level, our conclusions regarding the relative effectiveness of intervention strategies remain consistent with those from the country-level analysis. Specifically, reducing the number of daily cases below one after six months of intervention cannot be achieved by reductions in community and nosocomial transmission alone and the most feasible way to achieve swift control of epidemic is to use a combination of intervention strategies.

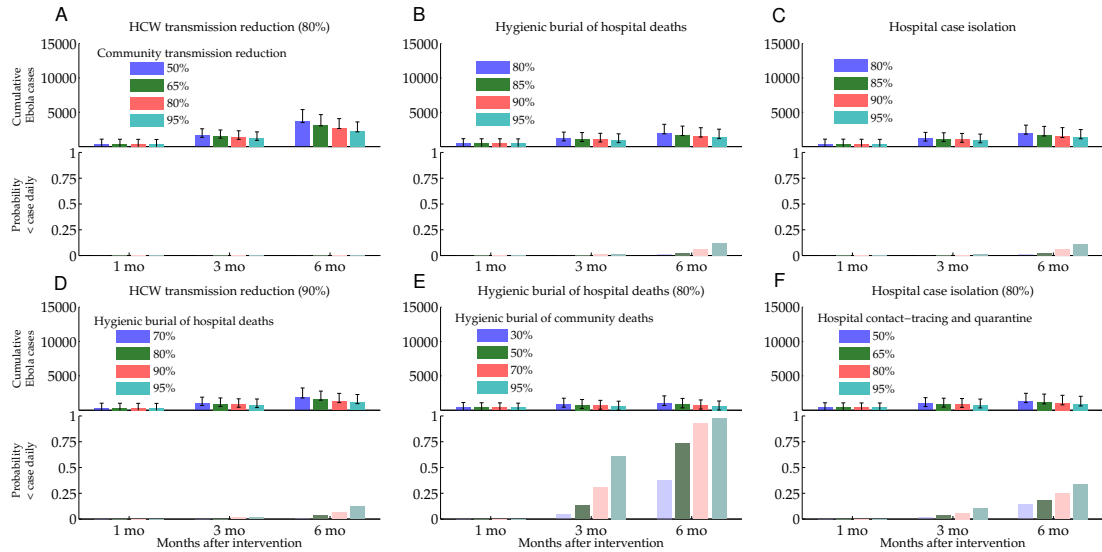


Figure S3: Model predictions of the cumulative number of new cases, as well as the probability of fewer than one case daily after one, three, and six months for Montserrado county for the following six intervention strategies: A.) reduction of transmission to healthcare workers and varying reductions in community transmission, B.) increasing proportions of hygienic burial of hospital cases, C.) increasing proportions of case isolation after hospitalization of cases, D. reduction of transmission to healthcare workers and increasing proportions of hygienic burial of hospital cases, E.) hygienic burial of hospitalized cases with increasing proportions of hygienic burial of community cases, and F.) case isolation of hospital cases with concurrent contact tracing and isolation of infected contacts. Cumulative cases over time were calculated from the deterministic difference equation model. One thousand simulations of the stochastic model were used to generate the cumulative case count error bars (95% prediction interval) and to estimate the probability of less than one new case per day.

## S8 Sensitivity and elasticity calculations

We calculated sensitivities and elasticities of our predictions with respect to the epidemiological parameters of our model (42). Sensitivity of an outcome was quantified as the increase or decrease in outcome unit per increase in parameter unit. For example, a sensitivity of  $s$  for a particular parameter meant that an increase of that parameter by 0.01 would lead to a change in the outcome measure by  $0.01s$ . Elasticity of an outcome quantified the proportional change in the outcome when a parameter is increased, and thus was dimensionless. For example, an elasticity of  $e$  meant that varying a particular parameter by a factor of 1.01 would lead to the outcome measure scaled by a factor of  $1.01e$ .

## S9 Additional sensitivity and elasticity results

As there was insufficient data on funeral transmission from the current epidemic to estimate the risk of contracting Ebola at a funeral, we estimated this odds ratio from a previous Ebola outbreak in Kikwit (11). To reduce a potential bias in overestimating the importance of funeral transmission, we conservatively assumed the lower confidence bound of 1.2 for the odds ratio of contracting Ebola for funeral attendance relative to non-funeral attendance. If the odds ratio of funeral transmission were lower than 1.2, more transmission would occur in the general community and at hospitals, suggesting that a lower value would reduce the effectiveness of funeral-based interventions. For example, the elasticity of  $-0.3$  for a combination strategy that relies heavily on interrupting funeral transmission suggested that if the odds

of contracting Ebola at a funeral were reduced from 1.2 by a factor of 15% to 1.02, the cumulative number of cases under this strategy would increase less than 5%. Therefore, given that our results highlight the importance of combined interventions to contain the outbreak at this point, the lower odds used for contracting Ebola at a funeral would strengthen our message.

We assume that all Ebola cases become symptomatic. However, if, as theorized during other Ebola outbreaks (26), a small proportion of cases were asymptomatic, the efficacy of any intervention strategy would be improved (negative elasticity for  $1 - \epsilon$ ; Fig 4)).

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