

# A Dynamic Model for Posttraumatic Stress Disorder Among U.S. Troops in Operation Iraqi Freedom

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We develop a dynamic model in which Operation Iraqi Freedom (OIF) servicemembers incur a random amount of combat stress during each month of deployment, develop posttraumatic stress disorder (PTSD) if their cumulative stress exceeds a servicemember-specific threshold, and then develop symptoms of PTSD after an additional time lag. Using Department of Defense deployment data and Mental Health Advisory Team PTSD survey data to calibrate the model, we predict that—because of the long time lags and the fact that some surveyed servicemembers experience additional combat after being surveyed—the fraction of Army soldiers and Marines who eventually suffer from PTSD will be approximately twice as large as in the raw survey data. We cannot put a confidence interval around this estimate, but there is considerable uncertainty (perhaps  $\pm 30\%$ ). The estimated PTSD rate translates into  $\approx 300,000$  PTSD cases among all Army soldiers and Marines in OIF, with  $\approx 20,000$  new cases each year the war is prolonged. The heterogeneity of threshold levels among servicemembers suggests that although multiple deployments raise an individual's risk of PTSD, in aggregate, multiple deployments lower the total number of PTSD cases by  $\approx 30\%$  relative to a hypothetical case in which the war was fought with many more servicemembers (i.e., a draft) deploying only once. The time lag dynamics suggest that, in aggregate, reserve servicemembers show symptoms  $\approx 1$ – $2$  years before active servicemembers and predict that  $>75\%$  of OIF servicemembers who self-reported symptoms during their second deployment were exposed to the PTSD-generating stress during their first deployment.

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## 1. Introduction

Posttraumatic stress disorder (PTSD) is an often persistent (Kessler et al. 1995) and sometimes debilitating (Zatzick et al. 1997) condition that is common among veterans of past (Centers for Disease Control 1988, Schlenger et al. 1992) and current (Hoge et al. 2004) wars and is strongly associated with the amount of combat exposure (Hoge et al. 2004, Office of the Surgeon Multinational Force (OSMF) 2006b). The tempo of the deployment cycles in Operation Iraqi Freedom (OIF) is higher than for any war since World War II, with many troops on multiple deployments (OSMF 2006b) and some Army soldiers experiencing 15-month deployments (Tyson and White 2007). To assure ample mental health resources to care for returning troops, it is important for the Department of Veterans Affairs (VA) to forecast the timing and number of new PTSD cases over the coming

years, which is complicated by the fact that many cases have delayed onset (Wolfe et al. 1999).

We introduce a dynamic mathematical model, which is described in §2, that uses OIF data to predict the incidence of symptomatic PTSD cases for OIF troops over the next several years and to gain an understanding of the relationship between deployment tempo, combat stress, and PTSD prevalence. The model contains a deployment model, which uses Department of Defense (DOD) data to construct a monthly deployment schedule for individual servicemembers in OIF and a PTSD model, which determines whether each servicemember develops PTSD. The PTSD model is a variant of the strength-stress models used in the reliability literature (Johnson 1988). Each servicemember has a random strength and accumulates stress according to a stochastic process: stress increases during deployments according

to a nonhomogeneous compound Poisson process whose mean is proportional to the average number of monthly casualties in OIF, and the stress decreases during periods between deployments. If a service-member's maximum stress level does not exceed his strength, then he does not develop PTSD. Otherwise, he develops PTSD during the first month in which his stress exceeds his strength and then develops symptoms after a random time lag that depends on whether he is still in the military or has returned to civilian life. The parameters for the PTSD model are estimated from data from several PTSD surveys carried out by the Army's Mental Health Advisory Team (MHAT).

With more data, this model could be used by the DOD to evaluate different deployment scenarios. However, with current data, we envision this model being most useful as a tool the VA could use to help estimate the demand for PTSD treatment by service-members returning from OIF in the coming years. In §3, we use this model to estimate the total number of PTSD cases under several different withdrawal scenarios and also perform various sensitivity analyses. These results are discussed in §4. We are unaware of any other modeling efforts related to PTSD.

## 2. Model Overview

The model has two parts: a deployment model and a PTSD model. We summarize the deployment model in §2.1, but the detailed formulation is in §1 of the online appendix (provided in the e-companion).<sup>1</sup> The PTSD model is described in detail in §2.2. The parameter values for the PTSD model are reported in §2.3, and the parameter estimation procedure appears in §2 of the online appendix.

### 2.1. Deployment Model

The U.S. military attempts to adhere to a unit rotation policy in OIF, where ideally an entire unit is moved into a theater, stays in place for a specified duration, and is then replaced by another unit when it returns home for a brief rest and further training before its next deployment (Congressional Budget Office (CBO) 2005). The lengths of the deployment and rest periods differ for active Army, reserve Army (e.g., National Guard and Army Reserve), and Marine personnel. We do not include Navy or Air Force personnel because they see much less combat than the Army and Marines (Statistical Information Analysis Division (SIAD) 2008a) and because we do not have any PTSD data for them. There are relatively few reserve Marines, and they are combined with the active Marines in our model.

For major combat troops and their attached combat support troops in the active Army, National Guard, and Marines, deployment schedules are constructed (§1.1 of the online appendix) from published data at the brigade level (for active Army and National Guard) or the more detailed battalion level (for Marines), where there are typically three–four battalions per brigade. Data are insufficient to model different occupational categories of service-members in more detail, and the survey data are conflicting, with some suggesting small differences in PTSD rates among different occupational categories in the Army (Smith et al. 2008) and some suggesting significant differences (OSMF 2005); our model captures the variation in risk among different categories at an aggregate level by incorporating a very bursty (compound Poisson) stochastic process for combat exposure. Because detailed deployment histories of unattached support troops (e.g., Army Reserve) are unavailable, we estimate their deployment schedules by assuming that they follow a cyclic deployment and rest schedule (§1.2 of the online appendix). The initial deployment dates for the unattached troops are chosen so that total monthly deployments (SIAD 2008b, GlobalSecurity.org 2008) and certain deployment characteristics (e.g., the fraction of servicemembers on their first deployment) at several snapshots in time (OSMF 2006a, b) are accurately predicted by the model (§1.2.3 of the online appendix). These deployment schedules, as well as other data for our model, are through September 2008.

We assume that every servicemember who starts a deployment stays until the end of the deployment. A fraction of troops—based on annual continuation rate data for the various military branches (CBO 2006)—in a particular deployment leave the military before the next planned deployment, and separated servicemembers are replaced by new servicemembers to maintain constant troop strength in each unit.

### 2.2. PTSD Model

The PTSD model can be viewed as a variant of the strength-stress models used in the reliability of manufactured items (Johnson 1988), in which both the strength and the stress of a servicemember are random (and, in our case, where the stress varies over time according to a stochastic process). The model has four main components. The first characterizes the precise sequence of months that each servicemember deploys, the second models a servicemember's exposure to and recovery from stress, the third defines the relationship between stress and developing PTSD, and the final part describes the delay between when a servicemember develops PTSD and when symptoms manifest themselves.

<sup>1</sup> An electronic companion to this paper is available as part of the online version that can be found at <http://mansci.journal.informs.org/>.

**2.2.1. Deployment History.** In our model  $j = 1, 2, 3$  denotes active Army, reserve Army (which includes Army Reserve and National Guard), and (active plus reserve) Marines, respectively. The  $k$ th servicemember of type  $j$  has an indicator process  $\{C_{kj}(t), t = 1, 2, \dots\}$  that characterizes his or her deployment history, where  $C_{kj}(t) = 1$  if servicemember  $k$  was deployed during month  $t$  and  $C_{kj}(t) = 0$  otherwise. There are many potential deployment history vectors because different servicemembers are first deployed at different times, separate from the military at different times, and may be attached or unattached. There are cohorts of troops with the same deployment schedules and hence the same  $C_{kj}(t)$  vector. For example, in our model the 1st Brigade, 1st Armored Division consists of 5,000 servicemembers and deploys twice to OIF (see §1.1 and Table 1 in the online appendix). This brigade has three associated cohorts: 1,879 troops who deploy only during the first tour of duty, 3,121 troops who deploy for both tours of duty, and 1,879 troops who deploy for only the second tour. Although we define these cohorts deterministically for computational tractability, in reality the number of servicemembers in each cohort will be related to a multinomial random variable with a very small (e.g.,  $\approx 0.01$ ) coefficient of variation. All troops within the same cohort have the same  $C_{kj}(t)$  vector, and the  $C_{kj}(t)$  vector associated with one cohort is different from the  $C_{kj}(t)$  vector associated with another cohort. In the following subsections, we calculate whether (and when) servicemember  $k$  of type  $j$  develops PTSD as a function of  $\{C_{kj}(t), t = 1, 2, \dots\}$ .

**2.2.2. Stress Exposure and Recovery.** Let  $D_{kj}(t)$  be the random cumulative stress of the  $k$ th servicemember of type  $j$  at the end of month  $t$ . We assume that the initial stress just before the first month of deployment is an independent and identically distributed (i.i.d.) exponential random variable with mean  $\alpha^{-1}$ , regardless of troop type. This reflects the heterogeneity of servicemembers' precombat experiences. We let  $\{E_{kj}(t), t = 1, 2, \dots\}$  be independent (but not identically distributed) random variables that represent the random stress that the  $k$ th servicemember of type  $j$  incurs during month  $t$  if he or she is deployed during that month. To maintain computational tractability when we analyze the sum of these random variables, and to allow flexibility in the amount of inter-servicemember variability in combat exposure, we model  $E_{kj}(t)$  as a compound Poisson random variable with mean  $\lambda_j(t)$  and constant batch size  $b$  (independent of  $j$  and  $t$ ); hence, the mean of the underlying Poisson random variable is  $\lambda_j(t)/b$  and the variance of  $E_{kj}(t)$  is  $b\lambda_j(t)$ . The constant batch size does not vary by military branch or month, which

allows the probability mass function of the cumulative stress to be easily derived while allowing flexibility in the coefficient of variation of the monthly stress among deployed troops.

Traumatic experience is causally related to PTSD (Fontana and Rosenheck 1998), and we assume that the average monthly stress,  $\lambda_j(t)$ , is related to the total number of OIF casualties per servicemember. The correlation between the number of fatalities and the number wounded in each month is 0.75 for the Army and 0.85 for the Marines (§2 of the online appendix), and there have been 6.99 times as many wounded as fatalities for Army and 8.51 times as many wounded as fatalities for Marines (SIAD 2008a). We model stress so that the fatalities and wounded are equally represented (although our quantitative results change little if we equate monthly stress to either fatalities or wounded). That is, the mean amount of stress for Army soldiers in a particular month is set equal to  $(6.99 \times \text{fatalities} + \text{wounded})$  for that month divided by the total deployment for that month; for Marines the mean amount of stress in a particular month is  $(8.51 \times \text{fatalities} + \text{wounded})$  for that month divided by the total deployment for that month. The average monthly stress  $\lambda_j(t)$  is illustrated in Figure 6 in the online appendix.

To model the partial recuperation of servicemembers when they are not deployed, we assume there is a geometric decay at monthly rate  $\theta$  during months when  $C_{kj}(t) = 0$ , where  $\theta \in [0, 1]$ ; i.e., the cumulative stress level decreases from  $D_{kj}(t)$  to  $\theta D_{kj}(t)$  after a month of no deployment. If a servicemember first deploys in month  $t$ , he does not undergo recuperation during months  $0, \dots, t - 1$ . Let  $\tau_{kj}(t)$  be the month during which the current deployment started if  $C_{kj}(t) = 1$ , and let  $\tau_{kj}(t)$  be the month during which the current break started if  $C_{kj}(t) = 0$ . Then for  $t = 1, 2, \dots$ , the stress dynamics are given by

$$D_{kj}(t) = D_{kj}(\tau_{kj}(t) - 1) + \sum_{s=\tau_{kj}(t)}^t E_{kj}(s) \quad \text{if } C_{kj}(t) = 1, \quad (1)$$

$$D_{kj}(t) = D_{kj}(\tau_{kj}(t) - 1)\theta^{t-\tau_{kj}(t)+1} \quad \text{if } C_{kj}(t) = 0. \quad (2)$$

In §2.3, we calibrate our model with data from the MHAT studies (OSMF 2006a, b). The MHAT surveys have been administered annually since late 2003 (OSMF 2003, 2005, 2006a, b, 2008). Their purpose is to assess the behavioral health of servicemembers deployed to OIF (OSMF 2006a). A sample of servicemembers deployed to OIF are given a survey with questions on environmental factors, individual and unit characteristics, and behavioral health status (OSMF 2006a). One of the key findings of these reports is that there is a positive correlation between the

probability of developing PTSD and combat intensity, number of deployments, and length of deployments (OSMF 2006a, b). In our model the probability of developing PTSD is an increasing function of the stress level  $D_{kj}(t)$  (see Equation (5)). Therefore, because  $\lambda_j(t)$  should be a reasonable representation of the combat intensity and the stress level increases with the length and number of deployments (see Equations (1) and (2)), our formulation is consistent with the findings in the MHAT studies.

**2.2.3. Stress Threshold.** Each servicemember has a different random threshold for stress, which represents his or her strength in the stress-strength model. We denote this stress threshold by  $\bar{D}_{kj}$  and assume a servicemember gets PTSD if  $\max_t D_{kj}(t) \geq \bar{D}_{kj}$ . In particular, if his cumulative stress exceeds  $\bar{D}_{kj}$  at some point, then he develops PTSD, even if subsequent rest periods bring the cumulative stress level below  $\bar{D}_{kj}$ . Hence, our model is a variant of strength-stress models (Johnson 1988), in which both the strength ( $\bar{D}_{kj}$ ) and stress ( $\{D_{kj}(t), t = 1, 2, \dots\}$ ) are random. Servicemember  $k$  of type  $j$  develops PTSD in month  $\bar{t}_{kj}$ , which satisfies

$$\bar{t}_{kj} = \min\{t \mid D_{kj}(t) \geq \bar{D}_{kj}\}. \quad (3)$$

The number of servicemembers of type  $j$  who have developed PTSD by month  $t$ , which we denote by  $Y_j(t)$ , is

$$Y_j(t) = \sum_k I_{\{\bar{t}_{kj} \leq t\}}, \quad (4)$$

where  $I_{\{x\}}$  is the indicator function of the event  $x$ .

We assume that  $\bar{D}_{kj}$  has an exponential distribution with mean  $\gamma^{-1}$ . The thresholds have the same distribution regardless of the troop type, which is consistent with empirical studies (OSMF 2005, 2006a; Milliken et al. 2007) comparing active Army and reserve servicemembers. However, PTSD rates can vary significantly across different unit types (e.g., transportation versus Medical; see annex A of OSMF 2005), which suggests that the threshold can depend on unit type. A more detailed analysis would further divide troops into specific unit types (e.g., transportation, combat, military police, engineering, medical, etc.), with each unit type having its own distribution for the threshold value and the stress process. Unfortunately, such refined data are not available to perform this detailed analysis.

The motivation for choosing an exponential distribution for  $\bar{D}_{kj}$  is based on dose-response functions in the infectious disease literature. The distribution of the stress threshold has a one-to-one correspondence to a dose-response function, where the response is the likelihood of PTSD and the dose is the maximum cumulative stress. There are two standard dose-response models in the infectious disease

literature: the Poisson model, which is used for some respirable diseases (Wells 1955), and a sigmoid (e.g., probit or logit) model, where the response is a sigmoid function of the logarithm of the dose (Finney 1971). Though PTSD is not an infectious disease, there are general similarities between the development of PTSD and the progression of infectious diseases, and we use the Poisson model in the base case (which yields the exponential distribution for  $\bar{D}_{kj}$ ) and perform a sensitivity analysis using the probit model.

The Poisson model is consistent with a “one-hit” model, where the size of the dose is Poisson with mean  $\gamma D$  and a single unit of dose that “hits” the target (e.g., enters the lungs, or in this case is sufficiently stressful) is sufficient to cause infection. For the Poisson model, the relationship between the response (i.e., the likelihood of PTSD) and the dose (i.e., cumulative stress) is given by

$$1 - e^{-\gamma D}, \quad (5)$$

where  $D$  is the cumulative stress and  $\gamma$  gives a measure of how much stress is required to cause PTSD. Consequently, the probability of developing PTSD is a concave function of the maximum cumulative stress level experienced by a servicemember (see also Figure 2 in §3.2), and the first traumatic event a soldier is exposed to during a deployment will have the largest marginal impact on his risk of developing PTSD.

To determine the distribution that corresponds to Equation (5), we assume there is an i.i.d.  $U[0, 1]$  random variable  $u_{kj}$  associated with each servicemember. Inverting Equation (5) yields each servicemember’s random stress threshold  $\bar{D}_{kj}$ , given by

$$\bar{D}_{kj} = -\frac{1}{\gamma} \ln(1 - u_{kj}). \quad (6)$$

This value is an exponential random variable with mean  $\gamma^{-1}$ .

The probability that a servicemember of type  $j$  develops PTSD is given by  $P(\max_t D_{kj}(t) > \bar{D}_{kj})$ . The cumulative stress  $D_{kj}(t)$  is nondecreasing in time if there is no recuperation ( $\theta = 1$ ), and in this case we can make a back-of-the-envelope estimate of this probability as a function of the total number of months deployed,  $m$ . We assume that  $t_m$  is the final month that this servicemember deploys, and to facilitate this estimation we ignore the initial stress. This, combined with the assumption of no recuperation, implies that  $D_{kj}(t_m)$  is a compound Poisson random variable with batch size  $b$  and mean  $\sum_{t: C_{kj}(t)=1} \lambda_j(t)$ . Recalling that the threshold  $\bar{D}_{kj}$  has an exponential distribution with mean  $\gamma^{-1}$ , we have that the

probability of developing PTSD is given by

$$P\left(\max_t D_{kj}(t) > \bar{D}_{kj}\right) = P(D_{kj}(t_m) > \bar{D}_{kj}), \quad (7a)$$

$$= \mathbf{E}[P(D_{kj}(t_m) > \bar{D}_{kj} | D_{kj}(t_m))], \quad (7b)$$

$$= \mathbf{E}[1 - e^{-\gamma D_{kj}(t_m)}], \quad (7c)$$

$$= 1 - \exp\left(-\sum_{t: C_{kj}(t)=1} \frac{\lambda_j(t)}{b}(1 - e^{-\gamma b})\right). \quad (7d)$$

The step from (7c) to (7d) makes use of the moment generating function of a Poisson random variable. If we assume  $\lambda_j(t) = \lambda_j$  is constant, then the expression in Equation (7d) simplifies to

$$P\left(\max_t D_{kj}(t) > \bar{D}_{kj}\right) = 1 - e^{-\kappa_j m}, \quad (8)$$

where  $m$  is the total number of months a servicemember deploys, and  $\kappa_j = (\lambda_j/b)(1 - e^{-\gamma b})$ . Substituting average values for  $\lambda_j$  and the base-case values of  $b$  and  $\gamma$ , all of which are estimated in §2 of the online appendix, we find that  $\kappa_1 = 0.028$  for the Army and  $\kappa_3 = 0.040$  for the Marines.

Before turning to the time lag before symptom onset, we note that another conceivable distribution for  $\bar{D}_{kj}$  would be the distribution corresponding to the probit dose-response model. Many dose-response curves have a sigmoid (e.g., probit or logit) behavior between the response and the logarithm of the dose (Finney 1971). The probit version states that the probability that a servicemember with cumulative stress  $D$  develops PTSD is  $\Phi(\beta \ln(D/ID_{50}))$ , where  $\Phi(\cdot)$  is the cumulative distribution function (cdf) of the standard normal distribution,  $ID_{50}$  is the cumulative stress that causes PTSD in half the population, and the probit slope  $\beta$  determines the population heterogeneity. For the probit model, Equation (6) is replaced by  $\bar{D}_{kj} = ID_{50} \exp(\Phi^{-1}(u_{kj})/\beta)$ . When we need to distinguish between the exponential distribution corresponding to the Poisson dose-response function and the threshold distribution corresponding to the probit dose-response function, we will refer to the dose-response function (i.e., the Poisson model or the probit model).

**2.2.4. Time Lag Dynamics.** A servicemember with PTSD experiences a lognormal time lag between the first time his cumulative stress level exceeds  $\bar{D}$  and the time at which he first develops symptoms. We choose a lognormal random variable because the time lag is qualitatively similar to the latent periods of infectious diseases, which often fit lognormal distributions well (Limpert et al. 2001). In this analysis we assume that someone “develops” symptoms not when he first physically exhibits symptoms, but when

he first reports or admits to symptoms. Hence, there are two components to the time lag: the lag between the traumatic event and the physical manifestation of symptoms, and the delay between the onset of symptoms and the reporting of symptoms. Because the studies we use to calibrate our model collect data from self-reported surveys (Wolfe et al. 1999, Milliken et al. 2007), we cannot disentangle these two factors. It is important that both factors are embedded in the time lag, because both contribute to the underreporting of PTSD. In addition, the great majority of servicemembers and veterans would not receive treatment until they self-reported symptoms, which is consistent with our model’s goal of helping to estimate the demand for mental health resources.

Recent data suggest that the time lag depends strongly on whether a servicemember is physically in the military (in our model,  $j = 1$  or  $j = 3$  and the servicemember has not discontinued service, or  $j = 2$  and  $C_{k2}(t) = 1$ ) or has returned to civilian life ( $j = 1$  or  $j = 3$  and the servicemember has discontinued service, or  $j = 2$  and  $C_{k2}(t) = 0$ ) (Milliken et al. 2007). The difference may be caused by organizational barriers to mental health care (Hoge et al. 2004, Figure 11 in OSMF 2006b), health-care benefits, the perceived stigma (Figure 10 in OSMF 2006b) associated with mental health problems (note that the survey results in Milliken et al. 2007 become part of each servicemember’s personal record), the military support system, the possibility of delayed discharge after symptoms are revealed, the expectation while in the military that mental health will improve on return to civilian life, and the stress involved with readjustment to civilian life (Milliken et al. 2007, Tanielian et al. 2008). To account for this dependence, we model both a military time lag and a civilian time lag. We assume that a servicemember’s time lag is given by the random variable  $T_1$  when he is physically in the military (even if he is not deployed), but switches to the random variable  $T_2$  when he returns to civilian life. We assume that this switch occurs in a memoryless manner, so that the time lag while in civilian life is independent of how long the servicemember was symptomless while physically in the military. However, if a Reserve Army servicemember ( $j = 2$ ) serves multiple deployments, we assume that history is maintained across consecutive periods while the servicemember is physically in the military and consecutive periods while he or she is in civilian life; i.e.,  $T_1$  and  $T_2$  apply to the cumulative amount of time physically in the military and in civilian life, respectively. For  $i = 1, 2$ , we let  $f_i(t)$  and  $F_i(t)$  denote the pdf and cdf of  $T_i$ , which is lognormal with median  $e^{\mu_i}$  and dispersion factor  $e^{s_i}$ .

We let  $S_j(t)$  denote the cumulative number of servicemembers of type  $j$  who have developed PTSD

symptoms by time  $t$ . Defining  $X_{kj}$  as the amount of time between when servicemember  $k$  of type  $j$  develops PTSD and the onset of symptoms, we have

$$S_j(t) = \sum_k I_{\{\bar{t}_{kj} + X_{kj} \leq t\}}. \quad (9)$$

Although the  $X_{kj}$  are independent, they are not identically distributed. The  $X_{kj}$  depend on several factors, including whether a servicemember is active or reserve, the specific deployment schedule, and when the servicemember separates from the military. Thus, the  $X_{kj}$  cannot be written easily as a function of  $T_1$  and  $T_2$ .

We do not model the amount of time the PTSD persists, which depends on a variety of factors, including the severity of symptoms and the amount of mental health care received. Although symptoms of PTSD can abate without treatment in a minority of cases, PTSD is known to be a persistent condition if left untreated, in which symptoms come and go over long periods of time (Kessler et al. 1995). Because we are interested in estimating the number of servicemembers who may require mental health-care treatment at some point in their lives, once someone develops symptomatic PTSD in our model, he or she does not recover on his or her own.

### 2.3. PTSD Parameter Estimates

The predicted troop levels track the official DOD troop numbers reasonably well: The average relative monthly deviation is <10%, with larger deviations occurring during the first 7 months of OIF (§1.3 in the online appendix). The parameter estimates for the PTSD model are presented in §2 of the online appendix. The four parameters of the lognormal time lags are derived from sparse longitudinal data (Wolfe et al. 1999, Milliken et al. 2007), and their estimates are  $\mu_1 = 2.47$ ,  $s_1 = 2.73$ ,  $\mu_2 = 1.40$ , and  $s_2 = 0.57$ . The military time lag until symptoms (median 11.78 months, mean 40.87 years) is longer and much more heavily tailed than the civilian time lag (median 4.05 months, mean 4.77 months), implying that some career military servicemembers in our model will never exhibit PTSD symptoms.

The four PTSD parameters (mean initial stress, batch size, mean threshold value, recuperation rate) are estimated using a least squares approach based on 17 PTSD rates from MHAT surveys for various groups of servicemembers at various points in time (OSMF 2006a, b). See §2 in the online appendix for more details on the estimation procedure. The mean initial stress ( $\alpha^{-1} = 0.0068$ ) is comparable to the average monthly stress from combat (0.0051 for Army, 0.0090 for Marines) in our model, suggesting that the stress endured during a month of exposure to combat could be greater than the stress previously

accumulated in a servicemember's lifetime, including the anticipation of deployment. The mean threshold value ( $\gamma^{-1} = 0.130$ ) is approximately equal to the average stress accumulated during two deployments. The batch size in the compound Poisson process ( $b = 0.0631$ ) is approximately equal to the average stress in one deployment. That is, the stress process is extremely bursty, with rare (e.g., approximately one-third of servicemembers are not exposed to any combat-related stress during a deployment) large jumps that represent particularly stressful events. In addition, servicemembers who do screen for PTSD in our model are going to vary by the ratio of their maximum stress divided by their threshold. Although we are focused on the fraction of servicemembers who get PTSD, to the extent that the severity of PTSD (and hence the type and intensity of treatment required) is related to the maximum stress-to-threshold ratio, our model predicts that there will be a range of severity of symptoms. Finally, there is full recuperation ( $\theta = 0$ ), which implies that there is no accumulation of stress across deployments in our base-case model. However, deploying multiple times does increase the probability of developing PTSD because of the greater exposure to trauma. Such a small value of  $\theta$  also implies that after he or she returns from a deployment, the stress level of the servicemember will drop below the precombat level. This is not implausible because of the stress (caused by uncertainty and inexperience) leading up to the first deployment.

The model's predicted PTSD rates are compared with the 17 reported MHAT PTSD rates in Table 1. The values in Table 1 are of the form  $P_j(\text{MHAT-}k)$ , which is the probability that a type  $j$  servicemember has symptomatic PTSD during the  $k$ th MHAT study. For example, " $P_{1+2}(\text{MHAT-IV})$ , 1st deployment" is the probability that active and reserve Army servicemembers on their first deployment had symptomatic PTSD during MHAT-IV (which was administered in October 2006). The 18th value in Table 1 is the fraction of troops exposed to no combat. This quantity is not an MHAT PTSD rate but is the fraction of Army soldiers finishing a deployment between 2004 and 2006 who were not exposed to any traumatic combat experiences. See §2 in the online appendix for more details on the values in Table 1 and how they were estimated.

The optimal parameter values achieve an average relative deviation of 15% for the 18 values in Table 1. Our model significantly underestimates the data point from the first MHAT study,  $P_{1+2}(\text{MHAT-I})$ . Indeed, the fact that this rate is greater than the PTSD rates in MHAT-II ( $P_{1+2}(\text{MHAT-II})$ ) and MHAT-III ( $P_{1+2}(\text{MHAT-III})$ ) is somewhat puzzling and is not commented on by the authors of MHAT-II or MHAT-III (OSMF 2005, 2006a). One possible explanation is that early in OIF the servicemembers may have been

**Table 1** Reported PTSD Prevalence Rates and Estimated PTSD Prevalence Rates for the Base-Case Model  
( $\alpha = 146.7$ ,  $\gamma = 7.72$ ,  $b = 0.0626$ , and  $\theta = 0$ )

PTSD prevalence rate	Reported	Estimated	References
$P_{1+2}$ (MHAT-I)	0.158	0.072	OSMF (2006a, p. 20)
$P_{1+2}$ (MHAT-II)	0.113	0.109	OSMF (2006a, p. 20)
$P_{1+2}$ (MHAT-III)	0.136	0.133	OSMF (2006a, p. 20)
$P_1$ (MHAT-III), 1st deployment	0.125	0.127	OSMF (2006a, p. 21)
$P_1$ (MHAT-III), >1st deployment	0.184	0.178	OSMF (2006a, p. 21)
$P_{1+2}$ (MHAT-IV)	0.170	0.148	OSMF (2006b, Figure 6)
$P_3$ (MHAT-IV)	0.140	0.166	OSMF (2006b, Figure 6)
$P_{1+2}$ (MHAT-IV), low exposure to trauma	0.080	0.026	OSMF (2006b, Figure 7a)
$P_{1+2}$ (MHAT-IV), medium exposure to trauma	0.140	0.139	OSMF (2006b, Figure 7a)
$P_{1+2}$ (MHAT-IV), high exposure to trauma	0.280	0.281	OSMF (2006b, Figure 7a)
$P_3$ (MHAT-IV), low exposure to trauma	0.060	0.050	OSMF (2006b, Figure 7b)
$P_3$ (MHAT-IV), medium exposure to trauma	0.110	0.159	OSMF (2006b, Figure 7b)
$P_3$ (MHAT-IV), high exposure to trauma	0.280	0.291	OSMF (2006b, Figure 7b)
$P_{1+2}$ (MHAT-IV), 1st deployment	0.150	0.110	OSMF (2006b, Figure 8)
$P_{1+2}$ (MHAT-IV), >1st deployment	0.240	0.236	OSMF (2006b, Figure 8)
$P_{1+2}$ (MHAT-IV), $\leq 6$ months on cur. depl.	0.120	0.118	OSMF (2006b, Figure 9)
$P_{1+2}$ (MHAT-IV), >6 months on cur. depl.	0.190	0.174	OSMF (2006b, Figure 9)
Fraction of troops exposed to no combat	0.320	0.304	Milliken et al. (2007)
Root mean square error		0.030	

Note.  $P_j$ (MHAT- $k$ ) represents the probability of a type  $j$  servicemember having symptomatic PTSD during the administration of the  $k$ th MHAT survey, where  $j = 1 + 2$  represents active and Army Reserve soldiers.

overly optimistic that OIF would progress similarly to the Gulf War with little exposure to combat and trauma. When that did not happen, these early deployers were more susceptible to PTSD, whereas servicemembers deploying later had more realistic expectations and perhaps more training to prepare for the nature of the war. This hypothesis suggests that the stress threshold  $\bar{D}_{kj}$  may have a time-varying distribution.

Our model also underestimates the PTSD rate for Army servicemembers exposed to the least amount of trauma ( $P_{1+2}$ (MHAT-IV), low exposure to trauma). These servicemembers may not have had as much training to prepare them for—nor the proper expectations regarding whether they will be involved in—traumatic incidents. When these servicemembers are exposed to combat or improvised explosive devices (IEDs), these events may have a more significant effect on them. This suggests that stress thresholds may be correlated with stress levels. If data existed at a more refined level (e.g., by occupation), we could incorporate this aspect into our model.

The model overestimates the PTSD rate for Marines exposed to an average amount of trauma ( $P_3$ (MHAT-IV), medium exposure to trauma). Of the four Marine PTSD rates, our model overestimates three of them. It is possible that Marines are better able to handle the stress (via self-selection and more intense screening and training) or perhaps less inclined to admit PTSD symptoms, than their Army counterparts. This implies that the threshold distribution may depend on the branch of the military or that the Marines may have a different time lag than the

Army. The Marine data point that the model underestimates is the PTSD rate for Marines exposed to the least amount of trauma ( $P_3$ (MHAT-IV), low exposure to trauma). If there is both an inadequate training and expectations factor that causes the model to underestimate the PTSD rate for servicemembers exposed to low amounts of trauma (as described in the previous paragraph) and a Marine factor that causes our model to overestimate the PTSD rate, then both of these competing factors will contribute to the estimate of the value “ $P_3$ (MHAT-IV), low exposure to trauma.”

The only other data point that does not fit well in the model is the PTSD rate for Army servicemembers on their first deployment during MHAT-IV ( $P_{1+2}$ (MHAT-IV), 1st deployment). This is more than three years into OIF, so naive expectations should not be a factor. Because this data point only includes first deployers, inexperience may play a role, but the model fits the data point “ $P_1$ (MHAT-III), 1st deployment” well (although the MHAT-III value does not include reserve servicemembers).

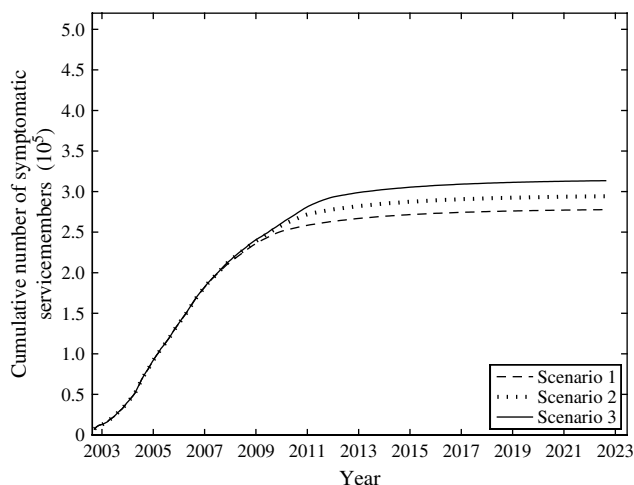
### 3. Results

We provide the base-case results in §3.1, followed by three model modifications in §3.2 and four sensitivity analyses in §3.3.

#### 3.1. Base-Case Results

We compute PTSD rates under three possible future withdrawal scenarios. In all three scenarios, the total troop level drops to 140,000 in July 2008 (Burns 2008) (as previously announced by President Bush in

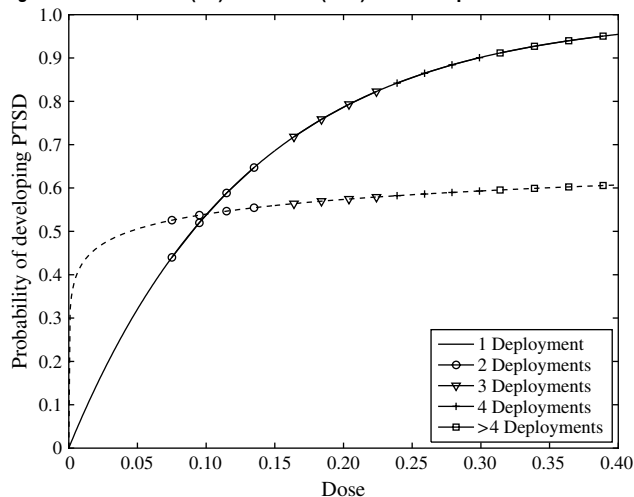
**Figure 1** Cumulative Number of Symptomatic Servicemembers for the Three Withdrawal Scenarios, as Predicted by the Model



2007), the troop level stays at 140,000 until withdrawal begins, and it takes 13 months to withdraw (modeled as a linear drop from 140,000 to 0) (CBO 2007). In the three scenarios, the withdrawal starts in February 2009, February 2010, and February 2011, respectively. We assume the stress process  $\lambda_i(t)$  (calculated separately for Army and Marine servicemembers) in each month starting with October 2008 is equal to the average value of  $\lambda_i(t)$  over October 2007–September 2008; because stress is measured as casualties per deployed servicemember, this assumption implies that the casualties are also dropping linearly to 0 during the 13-month withdrawal process. To perform this analysis, we need to specify the troop deployment schedules after September 2008, which we do through a combination of predicting combat units' future deployments using published data and estimating unattached support units' future deployments by assuming that they follow a cyclic deployment and rest schedule (§1.4 of the online appendix).

Figure 1 shows the predicted cumulative number of symptomatic PTSD cases as a function of time, starting from the beginning of OIF in March 2003, for the three different withdrawal strategies. Current and past cases of PTSD are when the three curves are together, and future cases are when the three curves split apart. By February 2023, our model predicts that 278,000, 294,000, and 313,000 servicemembers will have exhibited symptoms of PTSD under withdrawal scenarios 1, 2, and 3, respectively. This constitutes  $\approx 40\%$  of the active Army and Marines and  $\approx 32\%$  of the Army Reserve who deploy to OIF (Figure 8 in the online appendix). Because of the difference in the military and civilian time lags, symptomatic cases among the active Army soldiers and Marines lag behind the symptomatic cases in the reserve Army by  $\approx 1$ –2 years (§3.1 in the online appendix). As expected,

**Figure 2** Poisson (—) vs. Probit (---) Dose-Response Curves



*Note.* The dose (which represents the maximum cumulative stress experienced by a servicemember) is translated into number of deployments, for the no recuperation scenario, using the average monthly stress ( $\lambda$ ) and deployment lengths.

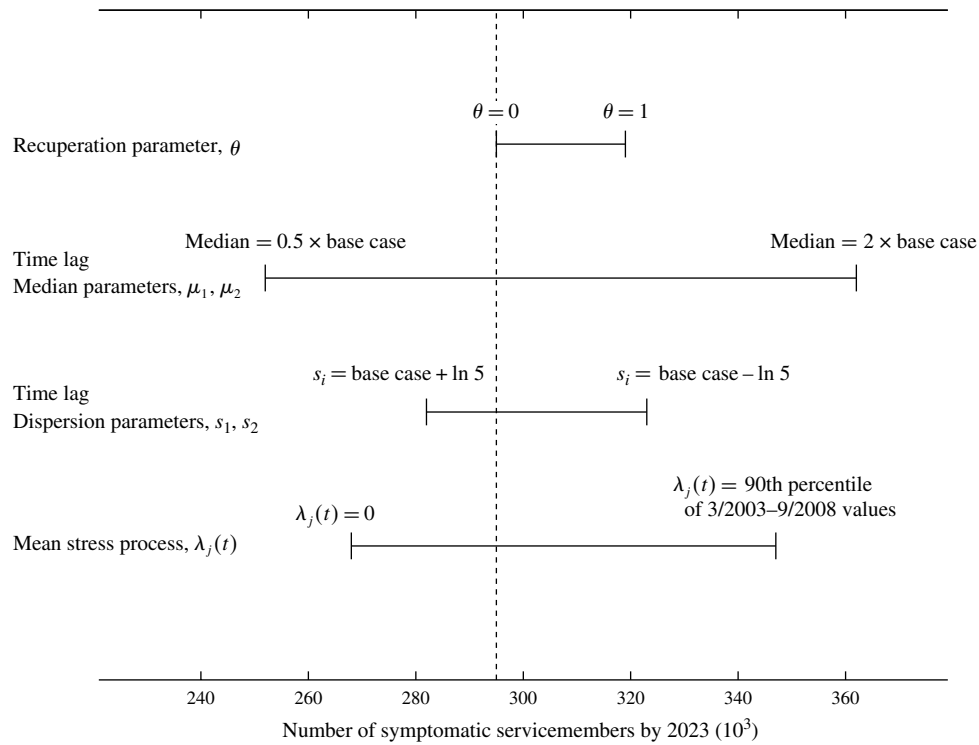
there is considerable heterogeneity among servicemembers in both the number of stressful events experienced (in scenario 2, 34.7% of servicemembers experience no stressful events, 0.2% experience  $\geq 9$  events; Table 19 in the online appendix) and the maximum cumulative stress/strength threshold ratio (in scenario 2, 71% of servicemembers are less than one order of magnitude from the critical ratio of 1, and 6% of servicemembers are more than two orders of magnitude away from 1; Figure 9 in the online appendix).

### 3.2. Model Modifications

We analyze several variations of our model to test how robust the model is (§3.2 in the online appendix). Replacing the Poisson dose-response function by the probit leads to a model with no recuperation ( $\theta = 1$ ) and a stronger cumulative effect from multiple deployments. The probit model has full recuperation, and the Poisson model has no recuperation because the probit dose-response curve is much flatter than the Poisson dose-response curve (Figure 2). The joint estimation of the PTSD parameter values leads to a one-dimensional subspace of solutions that yields nearly identical sum-of-squared deviations, which is only slightly lower than the base-case sum of squares; solutions in this subspace have no recuperation ( $\theta = 1$ ) and a similar batch size, and the mean initial stress level and the  $ID_{50}$  vary in a systematic way. The probit model predicts  $\approx 5\%$  fewer PTSD cases than the Poisson model, for the entire subspace of solutions.

To isolate the effect of multiple deployments, we consider the hypothetical case in which there is an

**Figure 3** Range of the Predicted Number of Servicemembers with Symptomatic PTSD for Each of the Scenarios Described in §3.3



*Notes.* The labels on the left side refer to the parameter being varied in each scenario (all other parameters remain at their base-case value). The base-case prediction is the dashed vertical line. These results are for withdrawal scenario 2, which begins in February 2010.

involuntary military draft, and each servicemember only deploys once. That is, we maintain the same PTSD parameter values and the same brigade/battalion rotation as in the base-case, but each time a unit is deployed, each servicemember is new. We make this modification for past, current, and future deployments. In this extreme scenario, the fraction of servicemembers that are symptomatic with PTSD is reduced to  $\approx 0.30$ , but the total number of symptomatic PTSD cases increases by  $>30\%$  because many more servicemembers are exposed to combat.

The decision to leave the military and return to civilian life may be related to a servicemember’s mental health (Hoge et al. 2006). To investigate an extreme version of this phenomenon, we modify the model so that at the end of each deployment the servicemembers who return to civilian life are those that currently have the highest stress-to-threshold ratio (see §3.2 in the online appendix for details). Using the base-case parameters, the number of symptomatic PTSD cases increases by  $\approx 5\%$  relative to the base case; although servicemembers who redeploy may be more resilient, servicemembers who have developed PTSD are likely to separate from the military and be replaced by new servicemembers who are susceptible to PTSD, thereby causing the increase. However, under this variant of the model, the fit of the MHAT probabilities with the base-case parameters is poor

(Table 18 in the online appendix). After a recalculation of the optimal parameters for this version of the model, the number of symptomatic PTSD cases increases by  $\approx 60\%$ ; the fit is still worse than the base case (see §3.2 in the online appendix), suggesting that the decision to leave the military may depend on a variety of other factors (such as family, finances, camaraderie, morale) and not primarily on a servicemember’s ability to cope with the stress that he has been exposed to.

**3.3. Sensitivity Analyses**

We perform four sensitivity analyses of the PTSD parameter value estimates (§3.3 in the online appendix). Figure 3 presents the range of the predicted number of symptomatic PTSD cases for each of these scenarios. First we disallow recuperation in the base-case model by setting  $\theta = 1$ . Keeping the other PTSD parameters at their base-case level leads to  $<10\%$  more symptomatic PTSD cases than in the base case, and reoptimizing the values of the remaining PTSD parameters leads to an increase in the number of symptomatic PTSD cases of  $<5\%$ . In both cases, the fit of the PTSD model is not much worse than in the base case (Table 18 in the online appendix).

The next two analyses involve the two time lag parameters. Because we have limited data with which to estimate these parameters, we vary the median

time lags (for both the military and civilian time lags) by a factor of two while maintaining the same dispersion factors. When the median time lag is increased by a factor of two, the mean threshold level ( $\gamma^{-1}$ ) decreases and there is no recuperation ( $\theta = 1$ ), so as to achieve the symptomatic PTSD rates in the MHAT studies (OSMF 2006a, b). In this case,  $\approx 20\%$  more servicemembers develop symptomatic PTSD. Similarly, when the median time lag is decreased by a factor of two from the base-case level, the mean threshold level increases, there is full recuperation ( $\theta = 0$ ), and the number of symptomatic PTSD cases decreases by  $\approx 15\%$ . Because the mean of the military time lag is much greater than the median in the base case, we also varied the dispersion factors. We increased and decreased the dispersion factors (for both the military and civilian time lags) by a factor of five (which is achieved by changing the parameters  $s_i$  by  $\pm \ln 5$ ) while maintaining the same medians. This has a significant impact on the variability of the military time lag distribution. The mean of the military time lag decreases from 40.87 years to 1.84 years when we reduce the dispersion and increases to more than 12,000 years when the dispersion increases. Even with these extreme variations from the base-case distribution, the results for these two scenarios were less than a 10% deviation from the base case (see §3.3 in the online appendix; see also Figure 3).

Finally we analyze three different values for the future mean stress process  $\lambda_j(t)$ . We consider the stress level to be 0, the median value between March 2003 and September 2008 and the 90th percentile value between March 2003 and September 2008. When we decrease the future stress level to 0,  $\approx 10\%$  fewer servicemembers develop symptomatic PTSD, and when we increase the future stress level to the median and 90th percentile value, the number of symptomatic PTSD cases increases by  $\approx 8\%$  and  $\approx 18\%$ , respectively.

## 4. Discussion

### 4.1. Limitations of Study

There are many organizational (e.g., training, leadership; see Figure 1 in OSMF 2006b), demographic (e.g., age, gender, marital status), and environmental (weather, uncertain future deployment) factors (OSMF 2003) that affect the behavioral health status of troops. Our model focuses on the impact of two interrelated factors: combat exposure and deployment schedule. The deployment cycle impacts PTSD prevalence in our model in two ways, by allowing for combat exposure during deployment and partial recuperation in between deployments. However, our analysis does not provide a reliable estimate for the recuperation rate because values at the two

extremes of 0 and 1 are obtained, depending on the choice of the dose-response function and the value of the median time lag until symptom onset. Moreover, our analysis is unable to shed any light on the nature of the dose-response relationship: when switching from a one-parameter Poisson model to a two-parameter probit model, we appear to be overfitting our model to the available data, as revealed by the one-dimensional subspace of solutions under the probit model.

Our analysis highlights the need for additional data (beyond the need to better estimate the time lag, as noted below), which would allow us to analyze more complex variants of the model. For example, the stress threshold distribution may vary over time and by troop type (see §2.3), and different types of servicemembers engage in different kinds of activities during their time between deployments, implying that the recuperation rate  $\theta$  may vary by troop type. Furthermore, the aggregation of casualty and PTSD data for soldiers prevented us from attempting to understand the differences in combat exposure and PTSD for different segments of the Army (e.g., combat versus transportation versus medical), even though our model explicitly captures the heterogeneity in combat exposure via the batch size in the compound Poisson process. On a related point, it is possible that IEDs, which became more common during the summer of 2005 (O'Hanlon and Campbell 2007, p. 31), caused additional stress in support troops (e.g., troops involved in transport logistics). However, IEDs are responsible for 80% of Army casualties in OIF (O'Hanlon and Campbell 2007, p. 31), so our measurement of monthly stress should indirectly capture this. Finally, we have assumed that the time lag is independent of the amount of combat exposure, although data suggest that individuals with more combat exposure were more likely to experience longer time lags (Gray et al. 2004).

### 4.2. Robustness of Results

Nonetheless, from the viewpoint of estimating the cumulative number of servicemembers (more specifically, Army soldiers and Marines) who will develop PTSD from OIF, our results appear to be quite robust: The difference in PTSD rates between allowing full recuperation between deployments and no recuperation is  $< 5\%$  (this small effect is due partially to the fact that our model predicts that more than half of the deployed servicemembers in OIF deploy only once), and the Poisson and probit models generate PTSD rates that differ by  $< 10\%$ . The casualty rate in OIF has been declining since the first half of 2007 (SIAD 2008a; see also Figure 6 in the online appendix), and if this rate continues to decline, then the PTSD rates could drop by as much as 10%–15%. However, if the

stress levels increase to those occurring earlier in OIF, then the PTSD rates could increase by 10%–20%.

Among the parameters in our model, the median time lag has the biggest impact on our results (Figure 3). The data available to estimate the time lag parameters are sparse, and it may be that two studies (Wolfe et al. 1999, Milliken et al. 2007) are too few to generate reliable estimates for the time lag parameters. Nonetheless, our base-case estimate gives a PTSD rate that is approximately twice the values in recent OIF surveys (Hoge et al. 2004; OSMF 2003, 2005, 2006a, b, 2008; Tanielian et al. 2008; although this last study includes Air Force and Navy personnel), and Figure 3 shows that the PTSD rate drops by only 20% from the base case when we cut the median time lags in half. Moreover, if we drastically reduce the military time lag distribution so that it equals the civilian distribution (analysis not shown), the PTSD rate is reduced to 27%, which is still much higher than the PTSD rates reported in the recent surveys; note that some of the discrepancy between the surveys and our results is because many surveyed servicemembers will be exposed to additional combat stress after they are surveyed (i.e., in the current or a subsequent deployment). Hence, the twofold punchline of this study is that ignoring the time lag (i.e., assuming it is zero, as is implicitly done in the recent surveys) and the future stress exposure of those surveyed leads to a significant underestimation of the PTSD rate, and further data are required to improve the precision of the time lag. More specifically, there is a need for a large-scale longitudinal study that involves at least three or four time points, which would provide a better understanding of the time lag distributions and hence a more refined forecast of future PTSD cases.

As an aside, our PTSD estimates are also higher than those obtained from the Vietnam War, which is not surprising, given the higher deployment tempo in OIF. A study of Vietnam veterans 15 years after they left the military (Schlenger et al. 1992) estimates a PTSD rate of  $\approx 15\%$ . The National Vietnam Veterans Readjustment Study estimated that  $\approx 30\%$  of Vietnam veterans would develop PTSD during their lifetimes (Kulka et al. 1988), although a recent reevaluation of that study by Dohrenwend et al. (2006) estimated the value was closer to 20%.

When considering whether our results can be extrapolated to all OIF servicemembers, it is important to note that the MHAT studies focus on combat units (OSMF 2003, 2005, 2006a, b), and it would seem that servicemembers in these units may screen for PTSD at higher rates than the general population deployed to OIF. However, MHAT studies II, III, and IV (OSMF 2005, 2006a, b), which contain the bulk of the PTSD data used to calibrate

our model, state that their samples should be representative of the larger theater population. Furthermore, combat servicemembers screen for mental health concerns at lower rates than several other occupations (see Figure 3 in annex A of OSMF 2005). Despite this potential bias, our results are likely to be conservative—that is, they are likely to underestimate the true number of servicemembers that will experience PTSD—for several reasons. Our model assumes that someone who develops PTSD stays in that condition. There is some selection bias in that only working, nondisabled servicemembers were surveyed in the MHAT studies (Hoge 2005). Furthermore, the MHAT reports define a servicemember as screening for PTSD through a self-reported survey (OSMF 2006b), which has been validated in military settings (Bliese et al. 2008) and has been used in several other studies analyzing PTSD in OIF servicemembers (Hoge et al. 2004, Hotopf et al. 2006, Smith et al. 2008) and is likely a conservative definition (Hoge et al. 2004, OSMF 2006b, Tanielian et al. 2008). In addition, the stigma associated with mental health problems and the shifting incentives as servicemembers return to civilian life can lead to underreporting and delayed reporting (although we attempt to capture the latter factor with the time lag); see our discussion about time lag dynamics in §2.2. Because of the paucity of longitudinal data, it is difficult to estimate the time lag until symptoms develop, and we may be underestimating the right tail of the time lag distribution by ignoring the right censoring in the longitudinal Gulf War study (Wolfe et al. 1999 and §2 in the online appendix). If the continuation rate depends on a servicemember's exposure to stress or the ability to cope with stress, then servicemembers with PTSD will have higher attrition rates, causing their replacements to receive more combat exposure (§3.2 in the online appendix). Because of the increase in the number of waivers of enlistment standards and less pre-combat training for recent Army recruits (Thompson 2007), it seems plausible that these soldiers will be more vulnerable to PTSD than the soldiers surveyed in the MHAT studies. Our PTSD estimates do not include servicemembers who never deploy or servicemembers from the Air Force or Navy (Smith et al. 2008), all of whom experience some PTSD, albeit at reduced rates (Smith et al. 2008). Our estimates also do not include the  $>10^6$  government contractors participating in OIF, who may have a more difficult time accessing mental health services (Risen 2007).

Our model also does not include servicemembers deployed to Afghanistan in Operation Enduring Freedom (OEF). Forces have been deployed to Afghanistan since 2001 and deployment data (SIAD 2008b) do exist, as well as casualty data (SIAD 2008a) and limited PTSD data (OSMF 2008). For most of

the duration of OIF, servicemembers deployed to Afghanistan in OEF were exposed to less combat and screened for PTSD at lower rates than troops deployed to OIF (Hoge et al. 2004, 2006; Tanielian et al. 2008). However, in the months at the end of our study there has been a trend of fewer casualties in OIF and more casualties in OEF (SIAD 2008a). Including OEF would have required a simultaneous deployment model to both theaters and possible estimation of more PTSD parameters with limited additional data. To get a sense of the relative magnitude of OIF and OEF, we note that there have been 13.0 times as many troops wounded and 8.5 times as many troops killed in OIF as there have been in OEF since the start of OIF in March 2003 (SIAD 2008a), and on average there have been 8.1 times more troops deployed per month to OIF than to OEF (SIAD 2008b). These figures suggest that the number of PTSD cases from OEF is roughly an order of magnitude less than the number of OIF cases (i.e., a total of  $\approx 30,000$  cases among soldiers and Marines).

Given that the earliest withdrawal date would appear to be no earlier than February 2009, given our sensitivity analyses (§3.2 and §3.3 in the online appendix), and given that the withdrawal itself may take longer than 13 months and may be incomplete, we predict that there will be at least 300,000 soldiers and Marines who develop PTSD and that on the margin, there are  $\approx 20,000$  new cases for every year that the war is prolonged. Although it is not possible to put a confidence interval on these estimates, our sensitivity analyses suggest that these figures are likely to be within  $\pm 30\%$ .

### 4.3. Effects of Time Lag and Multiple Deployments

Although our model was unable to tease out the form of the dose-response function or the value of the recuperation rate, its dynamic aspects allow us to understand the impact of the time lag until symptoms and of multiple deployments; in contrast, a traditional logit model—with independent variables such as the total exposure to combat, number of deployments, and deployment lengths—would require the PTSD status of each individual servicemember (this is not publicly available) and could not account for the impact of the time lag, which is crucial for developing an accurate estimate of the eventual PTSD rates. The time lag is shorter after a servicemember separates from the military (Milliken et al. 2007), which may be caused by a variety of factors, including the reluctance of active servicemembers to self-report PTSD symptoms, the two-year time window for VA health benefits after separation from the military, and the difficulties of transitioning back to civilian life. Consequently, our model predicts that, in

aggregate, reserves develop symptomatic PTSD  $\approx 1$ –2 years before active servicemembers, which is not inconsistent with recent VA data that members of the National Guard and Army Reserve have accounted for more than half of the suicides among OIF veterans (Associated Press 2008). Our model also predicts that among servicemembers who screened positive on their second deployment in the MHAT studies,  $>75\%$  were exposed to PTSD-generating stress during their first deployment.

There are many issues and concerns regarding health, experience, morale, family life, etc. that need to be balanced when determining deployment schedules. From the narrow confines of our model, multiple deployments can be viewed from the individual servicemember's viewpoint or from the perspective of the military as a whole. When viewed from the servicemember's viewpoint, the likelihood of a random servicemember developing PTSD after  $m$  months of service in the absence of recuperation between deployments is  $\approx 1 - e^{-0.028m}$  for Army soldiers and  $\approx 1 - e^{-0.040m}$  for Marines (see Equation (8)). Similarly, superimposing the average dose rate (i.e., stress from combat) and the deployment length on to the horizontal axis of the dose-response curve (Figure 2) shows the increased risk for PTSD incurred by multiple deployments with no recuperation (although the effect is larger for the Poisson curve than the flatter probit curve) and highlights one of the hazards of carrying out a prolonged war with a volunteer military. Even if there is full recuperation, the PTSD rate increases from 0.24 to 0.39 to 0.64 when comparing Marines who deployed for 1, 2, or  $\geq 4$  deployments, respectively. However, multiple deployments reduce the total number of PTSD cases, because of the concave nature of the dose-response curve: In the extreme hypothetical example where OIF had utilized an involuntary draft to increase the number of troops to the point where there were no multiple deployments, the number of PTSD cases increases by  $>30\%$ , that is, by  $>100,000$  cases. Moreover, this increase may be significantly underestimated because the thresholds for draftees would likely be lower than for volunteers (e.g., due to self-selection and motivation).

### 4.4. Supply vs. Demand

Our primary motivation for forecasting future PTSD incidence is to enable the VA system to plan for adequate supply of PTSD care. Unfortunately, when mapping from future PTSD incidence to future demand for VA mental health services, there are several factors that are considerably more uncertain than our estimates in Figure 1 for the number of OIF servicemembers and veterans who will develop PTSD each month. First, only some servicemembers will

be referred to the VA for a mental health evaluation on separation from the military. The Government Accountability Office reports that only 22% of OIF/OEF veterans at risk for PTSD were referred by DOD's health-care providers for a mental health evaluation; this reveals the tension between the VA's attempts at early intervention for rehabilitation and the DOD's retention goals (Bascetta 2006). Even if a veteran is screened for—and diagnosed with—PTSD, there is no guarantee that he will seek treatment. Indeed, the majority of the general U.S. population with mental health problems does not receive treatment (because of stigma or to lack of access and benefits; Kessler et al. 2005, Wang et al. 2005), and veterans may be no different. Even if veterans receive treatment, some of them may receive it in the private sector. Finally, as mentioned earlier, PTSD symptoms can come and go over long periods of time, and full remission is occasionally achieved after initial symptoms in the absence of treatment (Kessler et al. 1995). There are currently not ample data to estimate these other factors and hence to reliably convert future PTSD incidence into demand for VA mental health services. However, if all these other factors occur in a time-homogeneous manner, then Figure 1, combined with recent demand for VA mental health treatment by OIF veterans, can lead to crude estimates for future demand.

As discussed in Atkinson et al. (2008) and Wein (2009), the available supply and demand data suggest cause for concern; Atkinson et al. (2008) also provide a very crude demand-versus-supply analysis. Despite the uncertainties in our PTSD rates and in the factors raised in the previous paragraph, we believe that our analysis justifies making two policy recommendations: 100% of servicemembers should be evaluated by the VA for PTSD on separation from the military, and rapid evidence-based care should be provided to those servicemembers requiring treatment. Early identification and treatment of PTSD may lessen the severity of the condition, and if left untreated, PTSD can lead to comorbidities such as substance abuse and severe depression (Prigerson et al. 2002). A recent study concludes that the evidence is sufficient to conclude the efficacy of exposure therapies in the treatment of PTSD, but inadequate to determine the efficacy of a variety of pharmacotherapies and other psychotherapies (Institute of Medicine 2007). A recent cost analysis estimates that evidence-based PTSD care, which provides complete remission in an estimated 30%–50% of cases (Friedman 2006), would pay for itself within two years, largely by reducing the loss of productivity (Tanielian et al. 2008). The VA's response to workload increases and capacity shortages for mental health care has been to reduce the

intensity of service per patient (i.e., fewer patient visits per year, despite no improvements in treatment technology that would warrant this) (Rosenheck and Fontana 2007), which does not bode well for returning veterans with PTSD, who typically require 3–6 months of intensive treatment if there are no comorbidities (National Center for PTSD 2007). To create surge capacity during this crucial time window of troop withdrawal, the government may need to train and compensate mental health professionals in the private sector.

#### 4.5. Conclusion

We provide an integrative modeling approach that links rates of PTSD to troop deployment patterns and combat exposure during deployments. The incorporation of a time delay into the model reveals that raw survey data of active servicemembers during OIF is likely to significantly underestimate the number of PTSD cases ultimately generated. The model and analysis provide a starting point for further refinement of both the model and the parameter values as new data become available. Although it is tempting to employ the model to predict PTSD rates for various types of deployment schedules (e.g., frequent 6-month deployments versus infrequent 12-month deployments), we believe this is premature. Such a comparison would require an accurate estimate of the recuperation rate  $\theta$ , which could not be obtained from our analysis with the existing data.

## 5. Electronic Companion

An electronic companion to this paper is available as part of the online version that can be found at <http://mansci.journal.informs.org/>.

#### Acknowledgments

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