

**Economies of Scale in Risk Identification:  
The Case of Foodborne Illness and Public Health**

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I consider economies of scale in risk identification that arise from a production process that relies upon noisy feedback from affected consumers and from random sampling of product prior to shipment. A model inspired by the stylized facts of foodborne illness and the United States public health surveillance system is introduced where plant-level failures that cause foodborne illness persist until identified and plants exert additional effort following the public exposure of a failure. I derive several comparative dynamics that isolate under what conditions industry consolidation might reduce consumer damages despite the fact that consolidation exposes more consumers to a single potential error and identify how various biological and regulatory parameters impact steady state consumer damages. The issue of allocating scarce regulatory testing dollars across large and small firms is also explored numerically. For the two scenarios explored it is optimal to allocate more of the fixed testing resources to large firms, though small firms have a larger percent of their product tested before product shipment.

## **Economies of Scale in Risk Identification: The Case of Foodborne Illness and Public Health**

Food safety is a concern of many consumers. Despite on-site government inspection and firm-established, government-approved quality control systems,<sup>1</sup> shipments of contaminated food occasionally enter the food distribution network. Once in the network, consumers may consume contaminated food and contract foodborne illnesses. The US Centers for Disease Control and Prevention (CDC) estimate that in 1998 there were 76 million illnesses, 325,000 hospitalizations and 5,000 deaths in the United States (Mead et al., 1999). For meat and poultry consumers alone Crutchfield et al. (1997) suggest that annual medical and productivity costs attributable to seven key foodborne pathogens to range from \$6.5 to \$34.9 million a year (1997 dollars).<sup>2</sup>

The emergence of large processors and the concentration of the food processing industry to fewer firms in larger plants have led many to question how such concentration will affect the ambient food safety risk faced by consumers. Researchers from CDC have charged that “Changes in food processing and distribution are resulting in more multistate outbreaks of foodborne infection,” (CDC 1998, page viii). One explanation that often follows is that as food flows through fewer plants, a single critical processing mistake may lead to shipment of contaminated product that will expose more consumers to the risk of foodborne illness than if production were diversified across more, smaller plants. All told it adds one more reason why increasing concentration and scale in food processing is looked upon unfavorably by many consumer groups.

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<sup>1</sup> As of January of 1998 all meat slaughter firms under USDA inspection were required to file Sanitation Standard Operating Procedures as part of the revised USDA regulatory program based upon the concepts of Hazard Analysis of Critical Control Points (HACCP).

<sup>2</sup> Compare this to 1996 statistics of 35 million motor vehicle accidents resulting in 6.1 million injuries with damages estimated at \$120.8 billion (Insurance Information Institute, New York, NY, Insurance Facts. Estimates based on official reports from a representative cross-section of states. Includes all motor vehicle accidents on and off the road and all injuries regardless of length of disability. Damages calculated as wage loss; legal, medical, hospital, and funeral expenses; insurance administrative costs; and property damage.

A growing literature has assessed the costs of providing food safety for certain industry segments and for proposed regulations (Jensen et al., 1998; Antle, 2000; Crutchfield et al., 1997; Unnevehr, et al. 1998). Issues surrounding economies of scale and food safety usually are aimed at the cost of implementing a HAACP plan at a firm level or identifying how some firms may experience increasing returns to scale in providing product safety (Antle, 2000).

This paper explores a slightly different question: How might aggregate consumer damages change under different degrees of industry concentration? High levels of processor concentration are judged suspiciously by consumer advocates because it exposes many individuals to a single failure in quality control. However, increased concentration may also yield some avenues to reduce aggregate consumer exposure to food safety risks via economies of scale. One possible source of such economies is that of failure identification. One morbid but relevant fact is that large-scale mistakes are easier to identify and correct, particularly in noisy systems such as the foodborne illness surveillance system. This, of course, is only advantageous if such identification helps correct a quality control problem in the processing or distribution system that would otherwise be persistent or if association of a disease outbreak with a firm's food yields extra vigilance by the violative firm in terms of future safety.<sup>3</sup>

Much of the foodborne illness suffered by consumers goes unidentified because, after ingestion of many foodborne pathogens, an 18 to 36 hour incubation period must pass before the effects of foodborne illness appear (FDA, 1992). Once such an illness does appear, its symptoms are often similar to other illnesses that affect the gastrointestinal tract. An unfortunate consequence of the time delay and indistinguishable symptoms is that only in severe cases, when

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<sup>3</sup> Plant-level problems are often identified after consumers are made ill or die. For example, in the case the Hudson Foods Inc., the Colorado Department of public health detected a cluster of illnesses that were traced to the consumption of hamburger from a violative Hudson Foods plant, which resulted in the recall of 25 million pounds of potentially tainted hamburger (CDC, 1998).

symptoms of the illness become dangerous or life-threatening, does the chance of correctly attributing the effects of the illness with its foodborne source become possible.

Even upon proper association of foodborne pathogen cause and effect, it is difficult to identify the correct source of the foodborne pathogen. Again, the wide and uncertain time frame for possible consumption of the causative agent and patients' imperfect recall of foods consumed create uncertainty in locating a pathogen's foodborne source. However, when several individuals suffer similar severe symptoms contemporaneously, public health officials can then compare the diets of the victims and more easily identify the source of the illness. Such identification will loosely be called an outbreak for the purposes of this paper.

Another source of economies of scale in quality control emerges from simple statistical sampling principals. Given a certain percentage of contaminated units in a lot, the probability that a fixed number of tested samples misses a systemic problem is about the same regardless of the number of units in the lot. For example, if a firm's or government's quality control plan involves randomly sampling units from a outgoing lot and testing each unit for a particular foodborne pathogen that only appears in 10 percent of the units, the probability that contaminated food is shipped to the public is about the same whether five units are sampled from a lot of 1,000, 10,000 or 100,000 units.

Decentralization of food processing has a very intuitive advantage: fewer individuals are exposed to any single quality control mistake. However, when quality problems are difficult to identify yet such identification affects future quality, processor concentration, via the economies of scale in quality control outlined above, may offset the advantages of processor diversification. To explore the issue of aggregate foodborne illness risk and processor concentration, this paper

introduces a model of the food distribution network, consumer foodborne illness and the public health surveillance system.

In the model, plant-level problems that cause shipment of food with dangerous levels of microbial infestation are identified following the identification of a foodborne illness outbreak. After a public identification, the processor exerts extra vigilance during the following period such that no damage will occur. Plant-level problems may also be identified random pre-consumption testing of outgoing food shipments. If quality control problems are identified via testing, no consumer damage occurs in that period but quality control problems may occur in the following period with same probability. If identified via an outbreak, the firm exerts greater quality control vigilance for several subsequent periods before returning to the same probability of quality control problems. I then study how economies of scale might impact regulatory decisions, particularly, how a regulator might minimize social damage by allocating a fixed testing budget across firms of different sizes.

### **The Food Distribution and Public Health Model**

$N$  consumers each demand a single unit of a particular processed food product each period.  $J$  firms, each operating a single processing plant of uniform size, supply equal shares of the total demand each period and total industry supply is independent of  $J$  (i.e., no monopoly power is exerted for small  $J$ ).

With proper calibration and maintenance of processing equipment, each unit shipped is free from contamination.<sup>4</sup> However, equipment is subject to failure that causes microbial

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<sup>4</sup> For many food-pathogen combinations, most final product contains a low level of microbial contamination that causes no harm when consumed at retail. It is only when the microbial load surpasses key thresholds that damage may occur, in which case the model could be restated such that the Bernoulli event of interest was surpassing this threshold.

contamination of the food. In each period each firm draws randomly from a Bernoulli distribution and failure occurs with probability  $p \in (0, 1)$ . If a failure occurs, the firm ships contaminated product in the current period and in all future periods until its problem is identified. When such a failure occurs a fixed fraction  $\alpha \in (0, 1)$  of the units shipped by the firm contains microbial contamination. These units are randomly distributed across the outgoing shipment and, hence, consumed by a fixed number of consumers selected randomly from the consuming population. Consumers are unable to detect contamination and consume all food by the end of the period.

The size of the population receiving contaminated product from firm  $j$  equals  $\alpha N/J$ . Call this firm  $j$ 's *vulnerable population* and assume that a fixed fraction of vulnerable consumers,  $1 - \beta \in (0, 1)$ , employ effective food handling and preparation techniques and do not become ill.<sup>5</sup> However,  $A = \beta \alpha N/J$  consumers, called firm  $j$ 's *afflicted population*, do become ill if contamination occurs. Each afflicted consumer draws randomly from another, independent Bernoulli distribution with parameter  $\theta \in (0, 1)$ . Those drawing a one incur an illness that results in severe damages while those drawing a zero incur an illness associated with mild damages. Aggregate consumer damage during a given period is defined as the number of consumers with mild damages plus  $\phi > 1$  times the number with severe damage.

Consumers with mild damages endure pain and cost and may even visit a doctor; during any such visit, however, the doctor fails to link the illness to a foodborne pathogen and the illness is not reported to public health officials. Those with severe damages suffer greater pain

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<sup>5</sup> Food preparers can often prevent foodborne illness in the face of contaminated food by thoroughly cooking food and employing other practices that remove the microbial pathogens. Assume the fraction that prevents foodborne illness is constant in any given period and, because consumers cannot observe microbial contamination, does not increase if a given unit of food is contaminated. However,  $\beta$  may change with increased public education campaigns.

and cost and may even die. Such incidents require medical attention or an autopsy; the attending physician links the illness to a foodborne pathogen and alerts public health authorities.

Upon receiving such a severe illness report, public health authorities analyze the patient's diet and form a list of foods that may have caused such an illness. Identification of the source is impossible with only one report;<sup>6</sup> however, upon obtaining  $y \geq 2$  reports in which victims consumed product from the same firm in the same period, authorities can trace the problem to the violative plant (hereafter, traceback).<sup>7,8</sup> Denote the probability of traceback conditioned on shipment of contaminated food as  $\gamma$ . Upon successful traceback, the plant is immediately notified of the problem and the necessary corrections are implemented. Due to high levels of public and shareholder scrutiny exerted after such a publicized failure, firm managers exert additional effort following the outbreak; after  $w \geq 1$  damage-free periods, random failures again occur with probability  $p$ .<sup>9</sup>

Plant-level quality control failures can also be identified and corrected by a second method. The second method involves randomly sampling units from each lot and testing each sampled unit for contamination before the lot is shipped to consumers; a regulatory agency may require and verify that the sampling and testing scheme is properly executed.<sup>10</sup> The firm draws  $z$  units from each period's lot; if no units test positive for contamination the product is shipped.

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<sup>6</sup> Typically officials don't have enough resources to investigate each suspicious food or don't have the authority to investigate a suspicious food source on the basis of a single report.

<sup>7</sup> Note that if two different companies both produce the same type of food with contamination in the same period, and both lots cause  $y-1$  serious illnesses, an outbreak is not identified and production practices are improved in neither plant even though the total number of serious illnesses meet or exceed the threshold of  $y$ .

<sup>8</sup> It may also be of interest to allow an outbreak to be identified by a cumulative count of serious illnesses through time; i.e., different but sequential lots from the same company that together account for  $y$  or more outbreaks in adjacent periods. The implications for this paper are that smaller plants are more likely to detect quality control failures via an outbreak, but it will take longer for such a signal to get back to the violative firm.

<sup>9</sup> For example following a December 1998 outbreak of listeriosis caused by hot dogs produced in a Zeeland, Michigan plant operated by Sara Lee, lawyers and scientists representing victims requested to inspect the site. Needless to say, very high effort was likely exerted by plant officials during this post-outbreak time period.

<sup>10</sup> Testing of meat products, both at the plant level and at the retail level, are performed as part of USDA's revamped regulatory approach.

The probability that contaminated product is shipped despite the testing regime (i.e., a Type-I quality-control error) is denoted as  $\delta$ .

If any of the  $z$  units tests positive for contamination, the entire lot is discarded or the entire lot is perfectly screened before shipment; in either case no consumer damage occurs. Reports of the positive test results are not so widely publicized as are reports of outbreaks; firm managers exert some additional effort following the positive test result. After  $v < w$  damage-free periods, random failures again occur with probability  $p$ . Hereafter  $v$  is normalized to zero; i.e., no extra vigilance is exerted the period following a problem identified via sampling. Figure 1 schematically depicts model dynamics.

### **Expected Steady-State Damages**

In a given period, the model begins in one of  $w+2$  states. Call state 1 a ‘clean start’ which follows a period with no damages or a period where contamination is detected via testing; equipment failures occur with probability  $p$ . Call state 2 a ‘dirty start’ which follows a period in which a failure occurred but was not identified either via testing or traceback; the previous quality failure persists. Call state 3 through state  $w+2$  ‘guaranteed clean 1’ through ‘guaranteed clean  $w$ .’ These periods follow sequentially after a period with an outbreak; no damages occur during any of these periods due to extra vigilance by the plant. The state transition matrix for the case where  $w=2$  and for the generalized case is:

$$\mathbf{P}|_{w=2} = \begin{bmatrix} 1-p+p(1-\delta) & p\delta(1-\gamma) & p\delta\gamma & 0 & 0 \\ 1-\delta & \delta(1-\gamma) & \delta\gamma & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 1 \\ 1 & 0 & 0 & 0 & 0 \end{bmatrix}$$

(1)

$$\mathbf{P} = \begin{bmatrix} 1-p+p(1-\delta) & p\delta(1-\gamma) & p\delta\gamma & 0 & \dots & 0 \\ 1-\delta & \delta(1-\gamma) & \delta\gamma & 0 & \dots & 0 \\ 0 & 0 & 0 & 1 & \dots & 0 \\ \vdots & \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & 0 & 0 & \dots & 1 \\ 1 & 0 & 0 & 0 & \dots & 0 \end{bmatrix} .$$

Solving (1) for the steady-state probabilities of each state yields:

$$\pi_1 = \frac{1-\delta(1-\gamma)}{1+p\delta-\delta(1-\gamma)+(w-1)p\delta\gamma},$$

(2)

$$\pi_2 = \frac{p\delta(1-\gamma)}{1+p\delta-\delta(1-\gamma)+(w-1)p\delta\gamma},$$

$$\pi_3 = \pi_4 = \dots = \pi_{w+1} = \pi_{w+2} = \frac{p\delta\gamma}{1+p\delta-\delta(1-\gamma)+(w-1)p\delta\gamma}$$

The expected steady-state damages for the consumers of firm  $j$  in a clean-start period are:

$$(3) D^1 = p\delta\alpha \frac{N}{J} \beta(1-\theta + \phi\theta).$$

Beginning with a clean start, expected damages occur only if the plant suffers a quality control failure, which occurs with probability  $p$ , and then only if testing fails to identify such a failure, which occurs with probability  $\delta$ . Upon such an unidentified failure,  $\theta$  percent of the afflicted population ( $\beta\alpha N/J$ ) will suffer from severe damages valued at  $\phi$  while  $1-\theta$  percent will suffer from mild damages whose value is normalized to 1.

For dirty-start periods expected damages are:

$$(4) D^2 = \alpha\delta \frac{N}{J} \beta(1-\theta + \phi\theta) = D^1 / p.$$

This differs from the expected damages with a clean start because the uncertainty surrounding whether the plant will suffer a quality control failure is removed; the failure is persistent.

Damages associated with ‘guaranteed clean’ states are zero. Multiplying the state-contingent expected damages by the respective steady-state probabilities and summing across the  $J$  equally sized firms yields expected industry-wide steady-state damages of:

$$(5) JE[D] = \frac{JD^1}{1 + p\delta - \delta(1 - \gamma) + (w - 1)p\delta\gamma}.$$

### **Economies of Scale in Traceback and Sampling**

The conditional probabilities of identifying plant-level quality control failures through traceback ( $\gamma$ ) and sampling ( $1 - \delta$ ) are the linchpins to model’s dynamics because of the differential damage paths that follow each type of identification. As I show below, each mechanism features a type of economies of scale with regard to failure identification.

Consider the mechanics of traceback. If a type-I error has occurred (contaminated product is shipped to consumers) the conditional probability that exactly  $x$  consumers served by plant  $j$  become seriously ill in the event of contamination is denoted:

$$p_{\theta}(x | A) = \binom{A}{x} (\theta)^x (1 - \theta)^{(A-x)}$$

where  $\binom{A}{x} = \frac{A!}{x!(A-x)!}$ . In the event of product contamination, the expected number of serious

illnesses caused by firm  $j$  equals  $A\theta$  and the expected number of mild illnesses equals  $A(1 - \theta)$ .

The conditional probability that an outbreak will be identified and that traceback successfully locates and corrects the equipment failure at firm  $j$  is:

$$(6) \quad \gamma = \sum_{i=y}^A p_{\theta}(i | A) = 1 - \sum_{i=0}^{y-1} p_{\theta}(i | A) = 1 - \sum_{i=0}^{y-1} \binom{A}{i} (\theta)^i (1-\theta)^{(A-i)}$$

That is, an outbreak is identified if  $y$  or more individuals becomes seriously ill. To calculate this probability we merely sum the probabilities of any particular number of serious illnesses from  $y$  to  $A$  occurring. As indicated by the second equality in equation (6), this probability equals 1 minus the probability that fewer than  $y$  individuals become seriously ill.

The following proposition highlights some key insights concerning the means of problem identification available within the food distribution and public health surveillance model given the definition for  $\gamma$  outlined above. Proofs are available in Appendix A.

**Proposition 1:** In the event of a plant-level quality control failure at firm  $j$  the probability that the problem will be corrected is, *ceterus paribus*:

- (a) Decreasing in  $y$ , the outbreak identification threshold;
- (b) Increasing in  $\alpha$ , the percent of units within a contaminated lot that may cause illness;
- (c) Increasing in  $N$ , the total population served by all firms;
- (d) Increasing in  $\beta$ , the fraction of vulnerable consumers who are afflicted by illness;
- (e) Decreasing in  $J$ , the number of firms serving all consumers;
- (f) Increasing in  $\theta$ , the probability illness results in severe damages;
- (g) Independent of  $\phi$ , the relative magnitude of damages related to severe illnesses to those related to mild damages.

Each part of the proposition follows from simple intuition. Because outbreak identification, traceback, and correction of a quality control are synonymous in this model,

Proposition 1(a) states the obvious: if it requires more severely ill people to identify an outbreak, the conditional probability of correcting a quality control problem decreases.

Propositions 1(b) - 1(e) all build from evaluation of  $d\gamma/dA$ : how the size of a each plant's afflicted population affects  $\gamma$ . Increasing the afflicted population can never diminish the possibility of identifying an outbreak if  $y$  is fixed. Hence, increasing  $\alpha$ ,  $\beta$  or  $N$  makes the afflicted population larger and improves the probability of identifying an outbreak when contamination occurs, while increasing the number of firms,  $J$ , shrinks the per-firm afflicted population and diminishes the probability of having at least  $y$  people severely ill.

The intuition behind Proposition 1(f) is quite similar to Propositions 1(b) – 1(e): given all else equal, more severely ill people can never decrease the probability of  $y$  or more individuals becoming severely ill and inducing traceback. Also note that  $\phi$ , the damages incurred by the severely ill relative to the mildly ill, has no effect on the probability of traceback in this simple model.<sup>11</sup>

Now consider the effectiveness of sampling during a period when the equipment fails. Given that  $\alpha$  percent of units are contaminated and randomly distributed across the lot and  $z$  units are randomly sampled and perfectly tested, the probability of a Type-I quality-control error is:

$$\delta = \prod_{i=0}^{z-1} \left[ 1 - \frac{\alpha N}{(N - iJ)} \right].$$

A simple example is illustrative. Suppose each firm produces 100 units ( $N/J = 100$ ) and draws one sample each period. If an equipment failure causes 25% of units to be contaminated, there is a one in four chance the failure is identified; 75% of the time the firm will ship

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<sup>11</sup> The model could easily be altered to allow  $y$  to decrease with  $\phi$ ; that is an outbreak would be identified with fewer cases if each case was relatively more damaging.

contaminated product. If a second sample were drawn the probability equals  $(75/100)*(74/99) = 0.5606$  or the probability that the first sample missed a contaminated unit times the probability that a second sample was not contaminated.

Economies of scale are obvious when one repeats this example for a firm with the same failure rate that produces 1000 units. The probability of shipping contaminated product when only one sample is drawn is 0.75 ( $750/1000$ ). If a second sample is drawn it equals  $(750/1000)*(749/999) = 0.5623$ . Even for greater effort, say 10 samples, the probabilities of shipping bad product for the smaller and larger firm are 0.049 and 0.055, respectively. This minimal difference is amazingly robust to the difference in size of firms and in the probability of failure. In short the larger firm can provide similar levels of safety as the smaller firm with the same testing effort.

More formally, consider the following proposition:

**Proposition 2:** The probability of shipping contaminated product given contamination has occurred,  $\delta$ , is:

- (a) strictly decreasing in  $z$ , for  $z = 0, 1, 2, \dots, n-1$
- (b) non-increasing in  $J$ ,
- (c) decreasing in  $\alpha$  for  $z < N/J$ ,
- (d) non-decreasing in  $N$ .

Part (a) states the obvious: drawing more samples for pathogen testing reduces the chance of shipping contaminated product.

Part (b) is subtler than (a), but also straightforward. Spreading the same aggregate production over more firms means each firm ships smaller lot sizes. If the number of samples drawn from each lot does not change but each lot is smaller, the firm is less likely to ship

contaminated product because the same sample size gives broader testing coverage. For example, drawing 10 samples from a lot of 500 versus a lot of 400 means a lower probability of a Type-I error.

To gain an appreciation of the magnitude of the differentials dealt with in part (b), consider a non-trivial example in which adding another firm would have the greatest impact on the probability of shipping contaminated product. The example involves four consumers, one processing plant,  $\alpha = .5$ <sup>12</sup>, and  $z = 2$ . Consider the effect of adding another processing plant that also draws two pre-consumption samples. The conditional probability of shipping contaminated product when only one firm exists was  $(1-2/4)*(1-2/3) = 1/6$  while the probability with two firms is  $(1-2/4)*(1-2/2)=0$  (i.e., all product is tested before reaching the consumer). Adding another firm decreases  $\delta_{j,z}$  by  $1/6$  in this example and serves as the greatest improvement possible in non-trivial situations.

Part (d) follows with logic similar to that of part (b), only that there are now more total consumers and the same number of firms, hence lot size increases and the opposite result ensues.

Part (c) simply states that if a quality control failure causes a greater percentage of the lot to be contaminated, contaminated lots are less likely to be shipped. This poses a peculiar public-private tradeoff. Quality control failures that cause nearly the entire lot to contain high levels of pathogens will ensure that dangerous lots will be identified before consumption with minimal sampling effort. However, the larger the percentage of the lot that is contaminated, the higher the cost associated with reprocessing contaminated product to a safe level or the lower the probability that the any part of the entire lot might be salvaged for any valuable use.

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<sup>12</sup> If  $\alpha = 1.0$ , one sample would identify a contaminated lot while if  $\alpha=0.0$ , no amount of sampling would identify a contaminated lot. Setting  $\alpha=0.5$  yields the largest effect for the example.

### Comparative Dynamics of Damages without Product Testing

First, consider how marginal changes in key parameters affect expected steady-state damages when the industry is not regulated via any pre-market product testing protocol ( $\delta = 1$ ). A recurrent theme is that changes in expected steady-state damages come from two sources: a damage response, which captures changes in expected damages for a given state (clean or dirty), and a probability response, which captures changes in the steady-state probability for the various states. The damage response captures effects felt through changes in  $D^1$  and  $D^2$  while the probability response captures the effect through the steady-state probabilities  $\pi_i$ 's. Parametric changes will often increase expected damages via one response channel and decrease them via the other, though this is not uniformly true. For example, consider the following proposition:

**Proposition 3:** Per-firm expected steady-state damages are:

- (a) Increasing in  $y$ , the threshold for outbreak identification and traceback, and
- (b) Increasing in  $p$ , the probability of contamination incidence.

*Proof:* Part (a) is established by calculating the change in damages associated with increasing the  $y$  by one:

$$(7) \quad \begin{aligned} \frac{dE[D]}{dy} &= J\{E[D | y+1] - E[D | y]\} \\ &= JD^1 \frac{(\gamma_y - \gamma_{y+1})}{(p + \gamma_y)(p + \gamma_{y+1})} > 0 \end{aligned}$$

where, by (A2), the numerator is positive, hence establishing the sign of the inequality. The result in (b) is established by calculating the partial differential of steady-state damages with respect to  $p$ :

$$\begin{aligned}
(8) \quad \frac{\partial E[D]}{\partial p} &= J \left\{ \frac{\partial D^1}{\partial p} \frac{1}{p + \gamma} + D^1 \frac{\partial}{\partial p} \left[ \frac{1}{p + \gamma} \right] \right\} \\
&= J \left\{ \frac{\gamma D^2}{(p + \gamma)^2} \right\} > 0
\end{aligned}$$

In the case of Proposition 3(a) changes in  $y$  only appear in the probability response. That is, the expected damages for a given period and a given initial state are independent of outbreak identification threshold. Consumers suffer the same amount of damages regardless of  $y$ ; only the probability of traceback is affected. Indeed, by (A2), we know that increasing the number of severe damage victims needed to initiate traceback reduces the probability of traceback and, hence, the steady-state occurrence of the clean state. Because the clean state is associated with fewer expected damages, total expected damages increase as the identification threshold increases.

The negative of expression (7) captures the advantage of improved foodborne illness surveillance systems and suggests a means that public policy might affect  $y$ . For example, before 1995 the US Centers for Disease Control and Prevention (CDC), the public agency charged with coordinating outbreak investigation at the national level in the United States, relied on a passive surveillance system that aggregated reports gathered by state-level authorities who were called upon to investigate groups of illnesses thought to have a common origin. This allowed for a certain level of outbreak identification and traceback ability. In 1995 CDC shifted to an active surveillance system that regularly tests a random sample of stool specimens collected from patients visiting a doctor for general gastrointestinal illnesses in one of several geographic catchment regions. While primarily meant as a means to improve baseline measurement of the incidence of key foodborne pathogens in the general population, the system undoubtedly improves the probability of traceback in a fashion similar to lowering  $y$ .

In the case of changes in  $p$  addressed by Proposition 3(b), both the damage response and the probability response are positive. That is, a marginal increase in the incidence of contaminated lots has a non-negative effect on the damages suffered in both states and has a positive effect on the probability that the higher-expected-damages dirty state will occur. Hence the damage response and the probability response reinforce one another.

The US Department of Agriculture and the US Food and Drug Administration both mandate meat and seafood processing plants under their regulation to file and maintain quality control plans that meet agency standards. The regulatory mechanics, in part, involve certain design standards that aim to lower the incidence of pathogenic contamination (lower  $p$ ), limit the spread of pathogens within contaminated lots (lower  $\alpha$ ) and reduce the concentration of pathogens in a given serving of product (which might lower  $\theta$ ). By Proposition 3(b), if such regulation decreases  $p$  it will, *ceteris paribus*, unambiguously decrease aggregate damages caused by firms.

However, the following propositions suggest the *ceteris paribus* effect of  $\alpha$  and  $\theta$  on total damages is not so clear.

**Proposition 4:** Per-firm expected steady-state damages are:

- (a) increasing in  $\alpha$ , *ceteris paribus*, increasing in  $N$ , *ceteris paribus*, and increasing in  $\beta$ , *ceteris paribus*, if,

$$(9) \quad \frac{d\gamma}{dA} < \frac{\gamma_A + p}{A}, \text{ and}$$

- (b) increasing in  $\theta$ , *ceteris paribus*, if,

$$(10) \quad \frac{\partial \gamma}{\partial \theta} < \frac{(\phi - 1)}{(1 - \theta + \phi \theta)} (p + \gamma_A)$$

*Proof:* See calculations in Appendix A.

Consider Proposition 4(a) in which  $\alpha$ ,  $\beta$  or  $N$  is increased just enough to cause the per-plant afflicted population,  $A$ , to increase by 1 person. There is a direct damage response: more people will suffer damages when contamination occurs. There is also an offsetting probability response: the dirty state will be less likely because outbreak identification has improved. As the inequality in (9) suggests, when the marginal improvement in outbreak identification,  $d\gamma/dA$ , is small, the direct damage response will dominate. A small afflicted population,  $A$ , a high probability of contamination,  $p$ , and a large probability of outbreak identification also help insure that damages increase with  $\alpha$ ,  $\beta$  or  $N$ . However, if the afflicted population is large, the probability of contamination is small, the improvement in outbreak identification is large and the current probability of outbreak identification is small, expected steady-state damages could decrease with a greater percentage of contaminated units.

Similarly, in Proposition 4(b), increasing the percent of ill consumers who suffer severe damages has a direct damage response effect – the expected damage for those who fall ill is greater – and an indirect probability effect – higher severe damage counts improve the probability of traceback and decrease the probability of a dirty start. If the improvement in traceback probability,  $\partial\gamma/\partial\theta$ , is small or the ratio of damages for severe and mild illnesses,  $\phi$ , is large, the direct damage response dominates and total expected damages increase with greater severity of illness. However if increased severity dramatically improves traceback and damages from severe illness are not too much greater than from mild illness, increased severity could reduce steady-state damages.

The results from Proposition 4 can also speak another growing concern of industry regulators and public health officials: the growing virulence of certain pathogens. For example, the pathogen *E. coli* O157:H7 emerged during the early 1980's as a 'new' malevolent strain of

the bacteria commonly found in animal digestive tracts. Other public health fears include growing resistance by some pathogens to typical food preparation techniques and processing treatments. Growing virulence could increase  $\theta$ ,  $\phi$ , or effectively decrease  $\beta$  while growing resistance to processing treatments could affect  $p$ ,  $\alpha$  and  $\theta$ . In systems that rely upon outbreaks as the main source of identification, growing virulence may be a mixed blessing. Even if total damages do increase, one must account for any offsetting improvements in the steady-state damages incurred due to improved traceback.

Finally, consider the following proposition that explores the effect of adding one more firm to serve the same number of customers.

**Proposition 5:** Increasing the number of firms, ceteris paribus, increases the total expected steady-state damages.

*Proof.* Calculate the expected damages for an industry with  $J+1$  and  $J$  firms, adjusting for the fact that per firm afflicted population will decrease by  $k = \alpha\beta N/(J+1)$  and the probability of traceback,  $\gamma_A$  will differ by afflicted population size. Taking differences:

$$\begin{aligned}
 \frac{dE[D]}{dJ} &= (J+1)E[D | A-k] - JE[D | A] \\
 &= (J+1)(A-k)\hat{D}\frac{1}{p+\gamma_{A-k}} - J\hat{D}\frac{1}{p+\gamma_A} \\
 (11) \quad &= J\hat{D}\left[\frac{1}{p+\gamma_{A-k}} - \frac{1}{p+\gamma_A}\right] \\
 &= J\hat{D}\left[\frac{(\gamma_A - \gamma_{A-k})}{(p+\gamma_{A-k})(p+\gamma_A)}\right] > 0
 \end{aligned}$$

where  $\hat{D} = p(1 - \theta + \phi\theta)$  and the numerator of the expression in the final line of (12) is positive by (A7).

In this model, regardless of the number of firms, the same amount of contaminated product reaches the mouths of consumers. This can be seen in the simplification made from line 2 to line 3 of (11):  $(J+1)(A - k) = JA$  by the definition of  $k$ . Altering the number of firms merely affects the number of lots from which the contaminated product originates and, hence, the probability that an outbreak will be identified.

When traceback is the only mechanism by which quality control failures can be rectified and when small failures are inherently difficult to identify due to a noisy detection system, large mistakes that cause great damage in the near term may be more beneficial. Hence, centralizing production decreases expected steady-state production as the gains from improved detection unambiguously dominate losses from larger mistakes.

### **Comparative Dynamics of Damages with Product Testing**

First note that results concerning differentials of  $\gamma$  with respect to key parameters established in the previous section, where products were not tested, still hold; however, the probability of identifying an outbreak is now conditional upon both contamination occurring and contaminated product eluding detection during sampling.

**Proposition 6:** Expected steady-state damages are decreasing in the number of pre-consumption samples drawn.

*Proof:* Calculate the change in damages associated with increasing the number of pre-consumption samples by one:

$$(12) \quad \frac{dE[D]}{dz} = \left[ \frac{p\delta_{z+1}\alpha\beta(1-\theta+\phi\theta)}{1+\delta_{z+1}[p-1+\gamma+(w-1)p\gamma]} - \frac{p\delta_z\alpha\beta(1-\theta+\phi\theta)}{1+\delta_z[p-1+\gamma+(w-1)p\gamma]} \right]$$

where  $\delta_z \leq 1$  is the probability of a Type-I error associated with  $z$  samples and the denominators of both terms inside the square brackets are positive. The expression in brackets simplifies to:

$$(12') \quad \frac{(\delta_{z+1} - \delta_z)p\alpha\beta(1 - \theta + \phi\theta)}{\{1 + \delta_{z+1}[p - 1 + \gamma + (w - 1)p\gamma]\}\{1 + \delta_z[p - 1 + \gamma + (w - 1)p\gamma]\}} < 0 \text{ thus proving the}$$

inequality.

The effects of an increase in the probability of contamination and an increase in the threshold for identifying an outbreak are unambiguously positive, just as they were when no testing was performed. The following proposition is stated without proof:

**Proposition 7:** Expected steady-state damages are increasing in  $p$ , the probability of contamination and  $y$ , the threshold for outbreak identification and traceback.

As was the case when no testing was performed, the effects of changes in the breadth of contamination within a lot ( $\alpha$ ), the size of the consumer base ( $N$ ), and the relative severity of illness ( $\theta$ ) are ambiguous.

**Proposition 8:** Expected steady-state damages are

(a) increasing in  $\alpha$  if:

$$(13) \quad 1 + \delta_\alpha(p - 1 + \gamma_A) > A\{[(\delta_{\alpha+\varepsilon} - \delta_\alpha)(p - 1)] + [\delta_{\alpha+\varepsilon}\gamma_{A+1} - \delta_\alpha\gamma_A]\}, \text{ and}$$

(b) increasing in  $N$  if:

$$(14) \quad 1 + \delta_N(1 - p + \gamma_A) > A\{(\delta_{N+g} - \delta_N)(1 - p) + (\delta_{N+g}\gamma_{A+1} - \delta_N\gamma_A)\}$$

where  $\varepsilon$  is defined such that  $\varepsilon$  such that  $(A+1) = (\alpha + \varepsilon)N/J$  and  $g$  is defined such that  $(A+1) = \alpha(N + g)/J$ .

Proof. Appendix B.

Increasing  $\alpha$  has an ambiguous effect on total damages. It has an obvious direct damage response: an increase of  $\alpha$  by  $\varepsilon$  increases the afflicted population per plant by one person. When contaminated product is ingested, greater damages are expected. However, increasing  $\alpha$  has two probability responses that reduce expected damages. First, increasing  $\alpha$  reduces the chance that

contaminated product leaves the plant ( $d\delta/d\alpha < 0$ ) because fixed sampling is more likely to detect contaminated lots. Second, it improves the chance that, if a Type I error does occur, the public health surveillance system will trace the problem back to its source ( $d\gamma/d\alpha > 0$ ). These two probability effects may not always be fully felt, however, because of the differential damage reductions that accrue from the different means of identifying the problem. Recall, identification via outbreak guarantees no damages the following period. Therefore, there could exist a set of parameters for which increasing  $\alpha$  induces fewer contaminated shipments, hence fewer outbreak identifications and larger total damages because extra vigilance is invoked less often. Note also, that as the effect of  $\alpha$  on  $\gamma$  approaches zero, expected damages are unambiguously increasing. In other words, the damage reduction effect caused through fewer Type I errors as  $\alpha$  increases is not enough to offset the direct damage response.

Part (b) is also ambiguous. As in part (a), there are both damage response effects and probability response effects, though, in this case, the individual effects through  $\delta$  and  $\gamma$  competing rather than reinforcing. Increasing  $N$  by  $g$  makes detection more difficult with pre-consumption testing as lot size increases, but it makes outbreak identification more likely. However, substituting detection in the plant with detection in the field may be beneficial given post-outbreak vigilance. Inspection of expression (14) suggests that as  $\delta$  and  $\gamma$  become more sensitive to  $N$ , damages are more likely to decrease with population growth. If, however, changes in population yield no changes in either  $\delta$  or  $\gamma$ , expected damages will increase with population size.

**Proposition 9:** Expected damages are increasing in  $\theta$  if:

$$(15) \quad \frac{\partial \gamma}{\partial \theta} < \frac{(\phi - 1) + \delta p(\phi - 1) - \delta(1 - \gamma)(\phi - 1)}{\delta(1 - \theta + \phi\theta)}.$$

*Proof.* Partial differentiation of expected damages with respect to  $\theta$  yields:

$$(16) \quad \frac{\partial E[D]}{\partial \theta} = \frac{p\delta A[(\phi-1)((1+\delta(p-1+\gamma))-\delta(1-\theta+\phi\theta))\frac{\partial \gamma}{\partial \theta}]}{(1+\delta(p-1+\gamma))^2}.$$

The denominator is positive; hence the sign of the term in square brackets in the numerator dictates the sign of the result. Rearrangement of the numerator yields (15).

As was the case with no testing, the total effect of  $\theta$  on expected damages is ambiguous and depends upon the potential improvement in traceback from increased severity versus the direct effect of greater severity. Note that, unlike analyses involving  $\alpha$  or  $N$ , severity has no effect on  $\delta$ .

The partial derivative of traceback probability with respect to the conditional incidence of severe illness is positive and must be less than the expression on the right-hand side of (15). The first term in the numerator of the right-hand side is the direct damage response: more people suffer the damages associated with severe illness than with mild illness ( $\phi-1$ ). The second term in the numerator adds the fact that, dynamically, greater outbreak identification will cause more clean starts in which  $\delta p$  percent of cases will lead to greater marginal damages. The third term decreases the numerator and accounts for the fact that, with improved traceback, fewer dirty starts will occur where  $\delta(1-\gamma)$  suffer damages with certainty. All the terms in the numerator are normalized on the per-person expected damages associated with a dirty start.

Finally, I consider the impact of industry structure:

**Proposition 10:** The change in expected steady-state damages when existing consumption demand is met by adding another plant is increasing in the number of firms if:

$$(17) \quad \gamma_A - \gamma_{A-k} > \frac{\delta_{J,z} - \delta_{J+1,z}}{\delta_{J,z} \delta_{J+1,z}}.$$

*Proof.* Calculate the difference in damages between  $J+1$  and  $J$  firms:

$$(18) \quad \frac{dE[D]}{dJ} = (J+1)E[D | A-k] - JE[D | A]$$

$$= \tilde{D} \left[ \frac{(J+1)(A-k)\delta_{J+1,z}}{1 + \delta_{J+1,z}(p-1 + \gamma_{A-k})} - \frac{JA\delta_{J,z}}{1 + \delta_{J,z}(p-1 + \gamma_A)} \right].$$

Using the fact that  $(J+1)(A-k) = JA$  by the definition of  $k$ , one can rearrange the terms in square brackets to yield the expression in (18).

The sign is ambiguous and will depend upon the relative effects made to the two modes of identifying contaminated product. The left-hand side is positive and reflects the fact that, with production more decentralized, it is more difficult to identify an outbreak and perform traceback. The right-hand side is also positive and reflects that, with smaller lots leaving each plant, Type I errors are made less often. Note that if Type I errors rarely occur ( $\delta$ 's are small and drive the denominator of the right-hand side of (17) toward zero) damages are very likely to decrease with decentralization. The intuition is that possible reductions in damages due to improved outbreak identification are trivial because very little contaminated product ever reaches consumers.

Note the irony evident from comments made by small firms during the HACCP regulatory comment period: strong regulations, including a high degree of mandatory pre-consumption testing, were more likely to drive small shops out of business and leave a more concentrated industry. Hence, requiring a small  $\delta$  through regulation increases the likelihood that decentralization would yield fewer damages but may decrease the likelihood that decentralization would ever occur.

The ambiguity is not surprising given the competing effects in this model. More firms make on-site inspection marginally more effective but marginally decreases the chances of identification via outbreak.

## Optimal Distribution of Regulatory Effort

Consider a processing industry comprised of  $J$  plants that fall into two distinct categories: large and small. Regulators affect ambient safety by spending a fixed budget to test product before it leaves plant. Assume that there are no fixed costs for testing at a plant and that the budget allows for  $\bar{z}$  total samples to be tested each period. In light of the economies of scale identified above, how should fixed testing resources be allocated across plants to minimize expected damages from foodborne illness?

The non-continuous nature of the constrained minimization problem does not lend itself to analytical procedures. Hence, several pertinent examples are developed and the regulator's problem is solved numerically. Two base cases are considered where 40 tests per period must be allocated across firms that serve a population of 2,000 people.

The first set of parameters is chosen to elucidate the circumstances of a pathogen like *campylobacter* or *salmonella*, which are moderately prevalent in the food supply, but rarely cause severe damages. Table 1 outlines the exact parameter assumptions. Half of the consumers are supplied by a single large firm while the remaining consumers are equally divided among either 4 or 10 smaller firms. For simplicity, I assume large and small firms feature the same model parameters, though situations where smaller firms are more or less prone to quality control problems could easily be investigated. Two outbreak identification thresholds are considered,  $y = 2$  and 4.

When 10 small firms exist, total consumer damage is minimized when 10 samples are drawn at the single large firm and three samples are drawn at each of the smaller firms. This represents a one percent sampling rate for the large firm (10 samples from 1000 units shipped)

and a three percent sampling rate at each small firm. Alternatively, while the large firm represent 50 percent of market share it receives on 25 percent of the regulator's resources.

When four firms split the 1,000 consumers not supplied by the one large firm, consumer damages are minimized by focusing more total regulatory resources on the one large firm (12 samples per period or a 1.2 percent sampling rate) while seven samples are taken at each of the four smaller firms (a 2.8 percent sampling rate). This highlights the fact that the regulator can more regularly rely upon the ex post regulatory structure, the public health surveillance system, to help identify problems at the one large plant while ex ante regulatory steps in the form of sampling are applied more intensely at the smaller plants.

The bottom third of the Table 1 explores the robustness of this result to alterations in the effectiveness of the public health surveillance system. Indeed, when the threshold number of cases for identifying an outbreak doubles from two to four, the allocation of sampling effort is unchanged for this set of parameters.

A second set of parameters reflect the stylized facts of pathogens such as *e coli* or *listeria*, which are found less frequently in food but cause considerable damage if ingested. For the same industry structure of one large firm and four smaller firms, the optimal regulatory allocation is less clear, with several allocations yielding similar levels of societal damages. The lowest level of damages occurs when half of the samples are drawn at the one large firm (sampling rate of 2.0 percent) and five samples are drawn at each of the four smaller firms (sampling rate of 2.0 percent), suggesting that the higher ex post costs of the illness offset the desirability of using the public surveillance system as a means for locating persistent foodborne illness problems in the large plant.

## Summary and Conclusions

This paper considers the interaction of two emerging issues facing policy makers today: the increased occurrence and public perception of food safety problems and the concentration of the food processing and distribution industries. As food processing and distribution becomes concentrated among fewer and fewer firms, any quality control mistakes that introduce microbial pathogens into the food supply expose more people to possible consumption of this food and cost and suffering attributed to foodborne illnesses. With such a multiplier effect present, it may be tempting to add food safety to the list of reasons for controlling concentration in the food processing industry.

However, there may exist certain economies of scale in producing food with low levels of microbial contamination. This paper explores two possible avenues for such economies.

The first derives from the fact that large mistakes are easier to spot in a noisy system. If the identification of foodborne illness outbreaks help identify and correct firm-level quality control failures then, through time, the benefits of greater concentration offset the advantages of diversification. As the means for identifying firm-level failures relies less heavily on public illness outbreaks, the advantages of concentration for aggregate consumer food safety also decreases.

The second derives from the economies of scale inherent in statistical sampling protocol. It is shown that, for a given percent of contaminated units within a shipment of food, the marginal benefit of additional random sampling and testing is about the same regardless of the size of shipment. Therefore a fixed number of samples more reliably assures that failures are identified when sampling from one big shipment rather than several small shipments. However, if a fixed sampling budget must be divided among small and large firms and the key pathogen of

interest is moderately prevalent but not severe in its consequences, social damages are minimized when more total samples are taken at larger firms, though this represents a smaller sampling rate than for small firms. Damage minimizing sampling rates are more similar when the pathogen causing the damage is less prevalent but causes more severe damages.

While the model provides some interesting and intuitive results surrounding economies of scale in the provision of quality assurance, it would be interesting to enrich the model in several respects. For example, failure probabilities and industry structure are exogenous features of the present model. In reality firms may exert additional effort to lower the probability of failure and, in fact, this might be tied to existing industry structure. Furthermore, industry structure is likely to evolve over time, for example, as some firms exit after the incidence of a foodborne illness outbreak.

Also, the traditional effects of industry structure have been ignored in this analysis. In particular, the presence of a more concentrated industry may imply lower industry output which holds implications for social damages. Also, an industry earning monopoly profits might better facilitate legal ex post remedies for food safety incidents as it is easier to trace foodborne illness problems to the only firm producing a particular type of food and the firm may have a larger pool of resources, which mitigates the judgment-proof weakness of many liability based rules.

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Table 1. Optimal Sampling of Large and Small Plants for High Prevalence, Low Damage Pathogen

# of Samples Drawn at . .		Damages incurred by the 1,000 Consumers Served by ..			Total Societal Damages	
		# of Small Firms	The One Large Firm	Each Smaller Firm		One Large Firm
<i>Outbreak identification threshold (y) = 2</i>						
10		0	4	27291	10525	37816
		10	3	2297	17975	20272
		20	2	260	26972	27232
		30	1	0	47398	47398
		40	0	0	136293	136293
4		0	10	36338	2026	38364
		4	9	7139	3730	8668
		8	8	2500	2483	4983
		12	7	755	3324	4079
		16	6	521	4488	5009
		20	5	260	6010	6270
		24	4	261	10801	11062
		28	3	0	14574	14574
		32	2	0	23769	23769
	36	1	0	26866	26866	
<i>Outbreak identification threshold (y) = 4</i>						
4		0	10	36338	2026	38364
		4	9	7139	1717	8856
		8	8	2500	2617	5117
		12	7	755	3181	3936
		16	6	521	6017	6538
		20	5	260	7735	7995
		28	3	0	19698	19698

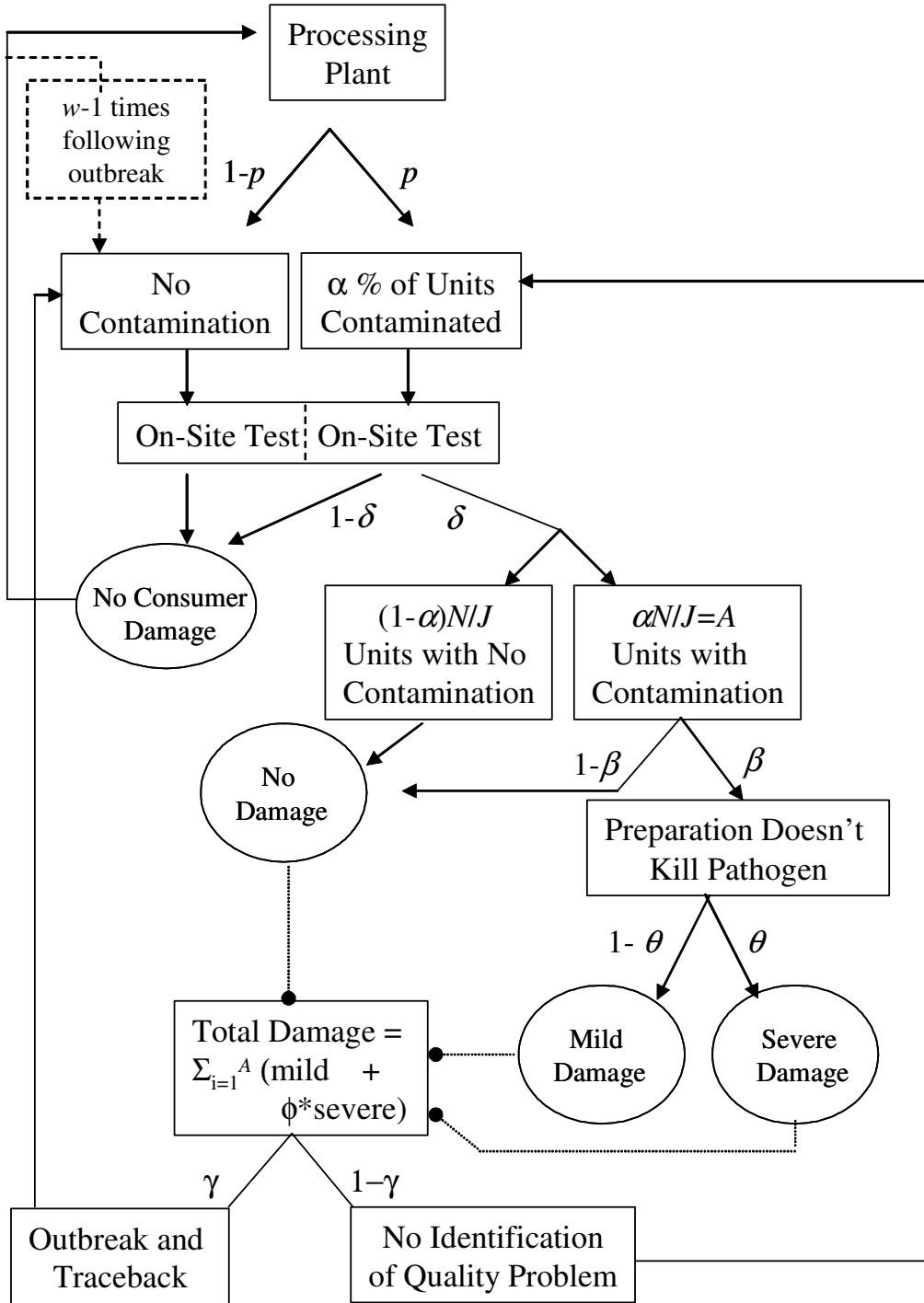
\*Parameters used in simulation are  $N = 2000$  (1,000 served by the one large firm and 1,000 equally divided among the smaller firms),  $p=0.1$ ,  $\alpha = 0.25$ ,  $\beta = 0.1$ ,  $\theta = 0.1$ ,  $\phi = 2.0$ .

Table 2. Optimal Sampling Between Large and Small Plants for Low Prevalence, High Damage Pathogen

		Damages from the 1,000 Consumers Served by			Total Societal Damages
# of Mom & Pop Firms	# of Samples Drawn at . .	Each Mom & Pop Firm	The One Big Firm	Mom & Pop Firms	
	4	0	10	96558	28161
4		9	51757	29853	81610
8		8	28209	37929	66138
12		7	23860	43507	67367
16		6	21051	46802	67853
20		5	12707	51711	64418
24		4	7506	62280	69786
32		2	2623	67755	70378
40		0	1042	81310	82352

\*Parameters used in simulation are  $N = 2000$  (1,000 served by the one large firm and 1,000 equally divided among the smaller firms),  $p=0.1$ ,  $\alpha = 0.10$ ,  $\beta = 0.1$ ,  $\theta = 0.9$ ,  $\phi = 10.0$ ,  $y = 4$ .

Figure 1. Flow Chart of Food Distribution and Public Health Surveillance Model



## Appendix A: Proof of Propositions 1 and 3

Recall the definition of  $\gamma$  is:

$$(A1) \quad \gamma = \sum_{i=y}^A p_{\theta}(i | A) = \sum_{i=y}^A \binom{A}{i} (\theta)^i (1-\theta)^{(A-i)} = 1 - \sum_{i=0}^{y-1} \binom{A}{i} (\theta)^i (1-\theta)^{(A-i)}.$$

To prove 1(a) calculate:

$$(A2) \quad \frac{d\gamma}{dy} = \sum_{i=y+1}^A p_{\theta}(i | A) - \sum_{i=y}^A p_{\theta}(i | A) = -\binom{A}{y} (\theta)^y (1-\theta)^{(A-y)} = -p_{\theta}(y | A) < 0,$$

which is clearly negative.

To prove 1(b)-1(e), note that  $A = \alpha\beta N/J$ , hence

$$(A3) \quad dA/d\alpha > 0,$$

$$(A4) \quad dA/dN > 0,$$

$$(A5) \quad dA/d\beta > 0, \text{ and}$$

$$(A6) \quad dA/dJ < 0.$$

Now let  $\gamma_{A+j} \equiv \sum_{i=y}^{A+j} p_{\theta}(i | A+j)$  and calculate:

$$\begin{aligned} \frac{d\gamma}{dA} &= \gamma_{A+1} - \gamma_A \\ &= 1 - \sum_{i=0}^{y-1} \binom{A+1}{i} (\theta)^i (1-\theta)^{(A+1-i)} - \left(1 - \sum_{i=0}^{y-1} \binom{A}{i} (\theta)^i (1-\theta)^{(A-i)}\right) \\ &= \sum_{i=0}^{y-1} \left[ \frac{A!}{i!(A-i)!} (\theta)^i (1-\theta)^{(A-i)} - \frac{(A+1)!}{i!(A+1-i)!} (\theta)^i (1-\theta)^{(A+1-i)} \right] \\ &= \sum_{i=0}^{y-1} \left[ \frac{A!(A+1-i)! (\theta)^i (1-\theta)^{(A-i)} - (A-i)!(A+1)! (\theta)^i (1-\theta)^{(A+1-i)}}{i!(A-i)!(A+1-i)!} \right] \\ &= \sum_{i=0}^{y-1} \left[ \frac{A!(A+1-i)(A-i)! (\theta)^i (1-\theta)^{(A-i)} - (A-i)!(A+1)(A)! (\theta)^i (1-\theta)^{(A-i)} (1-\theta)}{i!(A-i)!(A+1-i)!} \right] \\ &= \sum_{i=0}^{y-1} \left[ \frac{A!(A-i)! (\theta)^i (1-\theta)^{(A-i)}}{i!(A-i)!(A+1-i)!} (A+1-i - (A+1)(1-\theta)) \right] \\ &= \sum_{i=0}^{y-1} \left[ \frac{p_{\theta}(i | A)(A-i)!}{(A+1-i)!} (\theta(A+1) - i) \right] \\ &= \sum_{i=0}^{y-1} \left[ \frac{p_{\theta}(i | A)(A-i)!}{(A+1-i)(A-i)!} (\theta(A+1) - i) \right] \\ (A7) \quad &= \sum_{i=0}^{y-1} p_{\theta}(i | A) \left[ \frac{\theta(A+1) - i}{(A+1-i)} \right] \geq 0 \end{aligned}$$

To verify that (A7) is positive, notice that the final expression is a weighted summation of the probability function. The probability mass is distributed such that half or more is located at the mean,  $A\theta$ , or below. The weighting function,  $[\theta(A+1) - i]/[A+1-i]$ , is positive for  $i < (A\theta + \theta)$  and negative elsewhere. Hence, summing over  $i$  will never yield a negative outcome. That is, increasing the population exposed to contaminated

food from each firm will, *ceteris paribus*, never decrease the probability of identifying an outbreak. Given that  $\alpha$ ,  $N$  and  $J$  appear in  $\gamma$  only through  $A$ , and given the relations in (A3)-(A5), the Propositions 1(b)-1(d) are proven.

To prove Proposition 1(e) calculate:

$$(A8) \quad \begin{aligned} \frac{\partial \gamma}{\partial \theta} &= -\sum_{i=0}^{y-1} p_{\theta}(i|A) \left( \frac{i}{\theta} - \frac{A-i}{1-\theta} \right) = \sum_{i=0}^{y-1} p_{\theta}(i|A) \frac{A\theta - i}{\theta(1-\theta)} \\ &= \frac{1}{\theta(1-\theta)} \sum_{i=0}^{y-1} p_{\theta}(i|A) (A\theta - i) > 0 \end{aligned}$$

To verify the sign, note that in the second line of (A7) the summed term is strictly positive so long as the threshold for identifying an outbreak is less than or equal to the expected number of severe illnesses,  $A\theta$ . As the threshold for identification increases beyond the average, the summation terms grow increasingly negative. However, even if it required that everyone in the affected population become seriously ill (i.e.,  $y = A$ ), we see that the second line in (A7) approximates the first central moment of the conditional probability and the last expression then simplifies as follows:

$$(A8') \quad \begin{aligned} \left. \frac{\partial \gamma}{\partial \theta} \right|_{y=A} &= \frac{A\theta}{\theta(1-\theta)} \sum_{i=0}^{A-1} p_{\theta}(i|A) - \frac{1}{\theta(1-\theta)} \sum_{i=0}^{A-1} p_{\theta}(i|A) i \\ &= \left( \frac{A\theta}{\theta(1-\theta)} (1 - p_{\theta}(A|A)) \right) - \left( \frac{1}{\theta(1-\theta)} (A\theta - p_{\theta}(A|A)A) \right) \\ &= \frac{p_{\theta}(A|A)A}{\theta} > 0 \end{aligned}$$

Hence, proposition 1(e) is proven. Proposition 1(f) is obvious from inspection of  $\gamma$ .

For Proposition 3(a), consider an increase in either  $\alpha$  or  $N$  that increases the per-firm afflicted population from  $A$  to  $A+1$ . Calculate the change in damages as:

$$(A9) \quad \begin{aligned} \frac{dE[D]}{dA} &= J \left\{ \frac{(A+1)\hat{D}}{p(1-\gamma_{A+1}) + \gamma_{A+1}} - \frac{A\hat{D}}{p(1-\gamma_A) + \gamma_A} \right\} \\ &= J\hat{D} \left\{ \frac{A(\gamma_{A+1} - \gamma_A)(p-1) + p(1-\gamma_A) + \gamma_A}{[p(1-\gamma_{A+1}) + \gamma_{A+1}][p(1-\gamma_A) + \gamma_A]} \right\} \end{aligned}$$

where the expression is positive if the numerator in the second line of (A9) is positive or

if  $A(1-p)\frac{d\gamma}{dA} < \gamma_A + p(1-\gamma_A)$  as was to be shown.

For Proposition 3(b), partially differentiate  $E[D]$  with respect to  $\theta$ :

$$(A10) \quad \begin{aligned} \frac{\partial E[D]}{\partial \theta} &= \frac{\partial D^1}{\partial \theta} \frac{1}{p(1-\gamma) + \gamma} + D^1 \frac{\partial}{\partial \theta} \left[ \frac{1}{p(1-\gamma) + \gamma} \right] \\ &= \frac{pA(\phi-1)}{p(1-\gamma) + \gamma} - \frac{pA(1-\theta + \phi\theta)(1-p)\frac{\partial \gamma}{\partial \theta}}{[p(1-\gamma) + \gamma]^2} \end{aligned}$$

which is positive if  $(1-p)(1-\theta + \phi\theta)\frac{\partial \gamma}{\partial \theta} < (\phi-1)[p + \gamma(1-p)]$ .

## Appendix B: Proof of Proposition 8

For Proposition 8 (a), consider the effect on damages from increasing  $\alpha$  by a positive amount  $\varepsilon$  such that  $(A+1) = (\alpha + \varepsilon)\beta N/J$ . Calculate the difference in damages:

$$(B1) \quad \frac{dE[D]}{d\alpha} = \frac{(A+1)\tilde{D}}{1 + \delta_{\alpha+\varepsilon}(p-1 + \gamma_{\alpha+\varepsilon})} - \frac{A\tilde{D}}{1 + \delta_{\alpha}(p-1 + \gamma_{\alpha})}$$

where  $\tilde{D} = p(1 - \theta + \phi\theta)$ ,  $\delta_{\alpha+\varepsilon} = \prod_{i=0}^{z-1} \left[ 1 - \frac{(\alpha + \varepsilon)N}{N - iJ} \right]$ ,  $\gamma_{\alpha+\varepsilon} = \gamma_{A+1}$ , and  $\gamma_{\alpha} = \gamma_A$ . This expression is positive if:

$$(B1') \quad 1 + \delta_{\alpha}(p-1 + \gamma_A) > A\{[(\delta_{\alpha+\varepsilon} - \delta_{\alpha})(p-1)] + [\delta_{\alpha+\varepsilon}\gamma_{A+1} - \delta_{\alpha}\gamma_A]\} \text{ as was to be proved.}$$

For Proposition 8 (b), consider the effect on damages from increasing  $N$  by a positive amount  $g$  such that  $(A+1) = \alpha\beta(N + g)/J$ . Calculate the difference in damages:

$$(B2) \quad \frac{dE[D]}{dN} = \frac{(A+1)\tilde{D}}{1 + \delta_{N+g}(p-1 + \gamma_{A+1})} - \frac{A\tilde{D}}{1 + \delta_N(p-1 + \gamma_A)},$$

where  $\delta_{N+g} = \prod_{i=0}^{z-1} \left[ 1 - \frac{\alpha(N + g)}{N + g - iJ} \right]$ . This expression is positive if:

$$(B2') \quad 1 + \delta_N(1 - p + \gamma_A) > A\{(\delta_{N+g} - \delta_N)(1 - p) + (\delta_{N+g}\gamma_{A+1} - \delta_N\gamma_A)\} \\ \text{as was to be proved.}$$