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**Review**

# Predicting and Preventing Mold Spoilage of Food Products

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## ABSTRACT

This article is a review of how to quantify mold spoilage and consequently shelf life of a food product. Mold spoilage results from having a product contaminated with fungal spores that germinate and form a visible mycelium before the end of the shelf life. The spoilage can be then expressed as the combination of the probability of having a product contaminated and the probability of mold growth (germination and proliferation) up to a visible mycelium before the end of the shelf life. For products packed before being distributed to the retailers, the probability of having a product contaminated is a function of factors strictly linked to the factory design, process, and environment. The in-factory fungal contamination of a product might be controlled by good manufacturing hygiene practices and reduced by particular processing practices such as an adequate air-renewal system. To determine the probability of mold growth, both germination and mycelium proliferation can be mathematically described by primary models. When mold contamination on the product is scarce, the spores are spread on the product and more than a few spores are unlikely to be found at the same spot. In such a case, models applicable for a single spore should be used. Secondary models can be used to describe the effect of intrinsic and extrinsic factors on either the germination or proliferation of molds. Several polynomial models and gamma-type models quantifying the effect of water activity and temperature on mold growth are available. To a lesser extent, the effect of pH, ethanol, heat treatment, addition of preservatives, and modified atmospheres on mold growth also have been quantified. However, mold species variability has not yet been properly addressed, and only a few secondary models have been validated for food products. Once the probability of having mold spoilage is calculated for various shelf lives and product formulations, the model can be implemented as part of a risk management decision tool.

Molds are able to grow within a wide range of water activity values ($a_w$), pH values, and temperatures by using a large number of substrates such as carbohydrates, organic acids, proteins, and lipids *(48)*. Consequently, molds are able to grow on acidic products such as fruits or fruits juices *(58)* and on foods with intermediate moisture levels such as bread and bakery products *(2)* where other microorganisms such as bacteria cannot grow. Molds also can grow in cereals, beverages, dairy products, and fermented products *(29)* and thus are associated with the spoilage of a wide range of foods (Table 1).

Mold spoilage of food products causes great economic losses *(17)*. These losses are very difficult to evaluate because they fluctuate depending on the location, the season, and the type of product spoiled. For instance, 1 to 3% losses were reported in the bakery industry *(62),* and 5 to 10% losses were reported for postharvest fruits treated with fungicides *(11)*. Economically, mold spoilage losses in western Europe cost more than €200 million per year in the bread industry *(61),* and food losses related to fungal spoilage in Australia have been reported to be more than $10 million per year *(97)*. Consequently, prevention of mold spoilage is important for the food industry and can be accomplished by limiting in-factory contamination and mold growth. However, the factors involved in mold contamination and growth must be identified and quantified. Modeling tools can be useful for describing and understanding mold behavior on food matrices, as previously done with bacteria.

Four scientific reviews of fungal modeling have been published. In 1997, Gibson and Hocking *(35)* summarized the few studies done at that time. Then Dantigny et al. *(17)* developed the basis of predictive mycology by identifying primary and secondary models applied to germination and mycelium proliferation of spoilage molds. Pardo et al. *(86)* and Garcia et al. *(30)* reviewed mycotoxin modeling, focusing on food product safety. The purpose of the present article is to complete the work presented in previous reviews by focusing on food product stability regarding spoilage by molds. This spoilage ability is defined here as the capacity for a contaminating mold to grow on the surface of a given food product before the end of the product's shelf life. The development of visible mycelia on the surface of a product leads to rejection of the product by the consumer *(37, 46)*. Mold growth includes two interconnected phenomena: spore germination and mycelium proliferation (Fig. 1).

During germination and proliferation, molds may produce exoenzymes (e.g., lipases, proteases, and carbohydrases). These exoenzymes can transform the sensorial properties

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TABLE 1. *Nonexhaustive list of spoilage fungi isolated in food products between 1999 and 2012*

| Food type | Food product | Mold | Reference(s) |
|---|---|---|---|
| Bakery | Bread | *Aspergillus flavus* | *82* |
| | | *Endomyces fibuliger* | |
| | | *Penicillium commune* | |
| | | *P. corylophilum* | |
| | | *P. palitans* | |
| | | *P. polonicum* | |
| | | *P. roqueforti* | |
| | | *P. solitum* | |
| | Bread | *Fusarium* sp. | *59* |
| | | *P. chrysogenum* | |
| | | *P. solitum* | |
| | | *P. roqueforti* | |
| | | *P. commune* | |
| | | *P. polonicum* | |
| | | *A. niger* | |
| | | *A. flavus* | |
| | Pastry | *A. candidus* | *18* |
| | | *A. niger* | |
| | | *Cladosporium cladosporioides* | |
| | | *Eurotium herbariorum* | |
| | | *Mucor circinelloides* | |
| | | *Paecilomyces variotii* | |
| | | *Penicillium chrysogenum* | |
| | | *P. digitatum* | |
| | | *Rhizopus oryzae* | |
| | Pastry | *P. chrysogenum* | *114* |
| | | *A. flavus* | |
| | | *C. cladosporioides* | |
| | | *Alternaria alternata* | |
| | Pudding | *E. chevalieri* | *89* |
| | | *E. repens* | |
| | Not specified | *E. amstelodami* | *2* |
| | | *E. chevalieri* | |
| | | *E. herbariorum* | |
| | Not specified | *E. repens* | *41* |
| | Not specified | *P. brevicompactum* | *71* |
| Dairy | Yogurt | *M. circinelloides* | *37, 38* |
| | | *A. niger* | |
| | | *P. spinulosum* | |
| | | *P. chrysogenum* | |
| | | *P. expansum* | |
| | | *P. commune* | |
| | | *P. corylophilum* | |
| | | *P. purpurogenum* | |
| | | *A. flavus* | |
| | | *F. oxysporum* | |
| | | *Rhizopus oryzae* | |
| | | *C. cladosporioides* | |
| | Cheese | *P. commune* | *82* |
| | | *P. discolor* | |
| | | *P. roqueforti* | |
| | Dairy caramel | *Wallemia sebi* | *90* |
| Fruit | Wine grapes | *Botrytis cinerea* | *51* |
| | | *P. expansum* | |
| | Satsumas | *P. digitatum* | *24* |
| | | *P. italicum* | |
| | Apples | *P. expansum* | *5, 58* |
| | Strawberries | *B. cinerea* | *57* |
| | Jam | *W. sebi* | *90* |
| Grain | Wheat flour | *P.* species | *59* |
| | | *A. flavus* | |
| | | *A. terreus* | |
| | | *P. citrinum* | |
| | Corn | *A. flavus* | *3, 111, 112* |
| | | *A. parasiticus* | |
| | | *F. verticilliodes* | |
| | | *F. proliferatum* | |
| | Peanuts | *A. parasiticus* | *32* |
| | Coffee | *A. ochraceus* | *32* |
| | Cocoa powder | *P. chrysogenum* | *89* |
| | Cereal | *A. ochraceus* | *87* |
| | Cereal | *A. ochraceus* | *59* |
| | Grain | *P. verrucosum* | *59* |
| Other | Flavored water | *P. glabrum* | *80* |
| | Chocolate | *A. flavus* | *18* |
| | | *M. racemosus* | |

of the food product by inducing off-flavors, discoloring, and producing toxins *(29)*. Contrary to off-flavors and discoloration, toxins are potentially dangerous to consumer health. Although investigations into the occurrence and toxicity of mycotoxins in food chains have been conducted, the total number of mycotoxins involved in mycotoxicoses is not known, and little progress has been made in developing kinetic models. Exoenzyme and mycotoxin production are not considered in this review, which is focused on only visible spoilage.

This review includes formulation, process and environmental factors influencing mold growth, the mathematical models used to quantify the influence of these factors on mold growth, and a probabilistic approach used to determine the product shelf life. Based on this information, management options to prevent mold spoilage are suggested.

## FORMULATION FACTORS INFLUENCING MOLD GROWTH

**Water activity.** First defined by Scott *(119)*, $a_w$ is related to the water available in the matrix and is generally the dominant factor in the control of food stability and spoilage *(97)*. Based on $a_w$, two types of molds can be defined: xerophilic (or xerotolerant) molds, which can grow at low $a_w$ (i.e., below 0.85) and nonxerophilic, which can grow at $a_w$ from 0.85 to 1 *(95)*. Pelhate *(91)* described xerophilic fungi as having higher growth rates below an $a_w$ of 0.95. Some *Eurotium, Wallemia,* and *Chrysosporium* species are xerophilic *(130)*, whereas some *Penicillium* and *Aspergillus* species are not *(47)*. In terms of effect on mold growth, $a_w$ is widely recognized as having the greatest impact on mold germination and hyphal proliferation *(96, 114)*. The $a_w$ depression leads to a decrease in the speed of both germination and proliferation until a minimum is reached, where neither germination nor proliferation occur. Germination and proliferation rates also depend on the nature of the solutes involved in the $a_w$ depression *(47)*. This

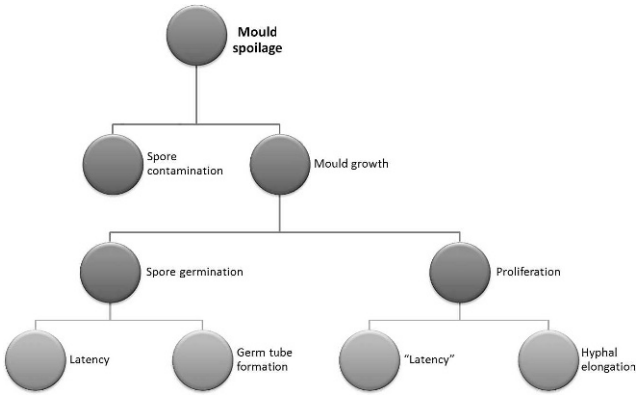


FIGURE 1. *Schematic representation of phenomena involved in food spoilage by molds.*

interaction is important in both laboratory media and food products.

**Hydrogen ion concentration (pH).** The pH in the range encountered in most food products has no significant direct effect on either germination (pH 3 to 6.5) *(116)* or mycelium proliferation (pH 3 to 8) *(132)*. However, pH may have indirect effects. First, it has an impact on the growth of the bacterial flora. When the pH is optimum (i.e., 4.5 to 8, depending on the physiology *(6)*) bacteria may grow in the matrix and compete with molds. This competition may induce a decrease in molds growth rate. Second, the pH affects the ratio of dissociated to undissociated potentially inhibitory organic acids in the food matrix. The undissociated forms of some organic acids generally have a greater inhibitory effect on mold growth than do the dissociated forms *(42)*.

**Preservatives.** To prevent food spoilage from microorganisms, the use of preservatives is almost routine for manufacturers. Among them, propionic acid, sorbic acid, benzoic acid, and their salts are efficient for inhibiting mold growth *(41)*. The weak acids are pH dependent because they exist in both dissociated and undissociated forms. The mode of action of weak acids is believed to be associated with the undissociated state. In this form, the acid is neutral and is able to penetrate the cell through the membrane. The acid then lowers the intracellular pH by releasing hydrogen ions and thus inhibits cell growth *(98)*. However, Stratford et al. *(121)* found that although acetic acid follows this pattern, sorbic acid does not induce intracellular acidification and a possible membrane-mediated mode of action may be involved.

The effect of weak acids on mold growth has been investigated by Guynot et al. *(41)* and Marin et al. *(66)* near neutral pH (6.5 to 7). They found that low concentrations of weak acids (0.025 and 0.05%) promote mold growth instead of inhibiting it. Under these conditions weak acids must be used at high concentrations to be effective.

**Lactic acid bacteria.** Lactic acid bacteria produce molecules such as organic acids, fatty acids, hydrogen peroxide, and bacteriocins that have an inhibiting effect on mold growth *(33)*. These bacteria also are generally recognized as safe for use in foods. Lactic acid bacteria can be used directly in processing of fermented food products, but the extraction and purification of the molecules of interest is plausible mostly when peptides are involved *(118)*. Biopreservation of food products through molds inhibition has been studied extensively *(15, 59, 69, 102, 106, 107, 129)*.

**Plant extract.** Plants contain numerous components and are great sources of new active molecules with antimicrobial properties *(78)*. Vapors of essential oils from plant extracts such as thyme oil have been effective for inhibiting growth of *Penicillium roqueforti, Penicillium corylophilum, Eurotium repens,* and *Aspergillus flavus (122)*. However, at effective concentrations essential oils may migrate in the product and change its aroma *(82)*. This change may not have an impact on consumer preferences for treated postharvest fruits but can be a problem in manufactured food products *(77)*. Although studies dealing with vapors of essential oils are numerous *(4, 36, 92, 99),* studies on nonessential oil plant extracts directly usable in food products are scarce *(102)*. Therefore, more research is needed to find new plant extracts that do not impact the organoleptic properties of food product but are effective for preventing mold spoilage.

## PROCESS AND ENVIRONMENTAL FACTORS INFLUENCING MOLD GROWTH

**Storage temperature.** Temperature is recognized as the second most important factor impacting mold growth after $a_w$. Interactions between temperature and $a_w$ also have a significant effect on fungal growth *(2, 68)*. Some species of molds are able to grow at moderate and high temperatures (thermotolerant molds). For instance, *A. flavus* and *Aspergillus niger* are able to grow between 8 and 45°C, with an optimum near 30°C *(97)*. These molds are involved in spoilage of shelf-stable food, especially in hot weather. Molds involved in spoilage of chilled food are more psychrophilic (i.e., capable of growing at low temperature). Certain species of *Penicillium, Cladosporium,* and *Fusarium* are able to grow between 0 and 5°C *(55)* or even at −7 to 0°C *(97)*.

**Heat treatment and mold heat resistance.** Few mold species are known to be heat resistant (i.e., molds that may survive pasteurizing processes). Most asexual spores (conidia) and vegetative cells of *Penicillium* and *Aspergillus* are killed after being heated for 5 min at 60°C *(126)*. The heat resistance of molds is thus associated with their thick-walled sexual spores (ascospores). Among the molds that produce ascospores, *Byssochlamys fulva, Byssochlamys nivea, Neosartorya fischeri, Talaromyces flavus, Talaromyces macrosporus,* and *Talaromyces avellaneus* are the most frequent species encountered in spoiled processed food *(128)*. These molds are commonly found in soil *(94)*. Consequently, they regularly spoil pasteurized foodstuffs (e.g., fruit juices and canned fruits) made of products readily contaminated by soil (e.g., fruits). To prevent these kinds of food from mold spoilage, manufacturers should take into consideration in their pasteurization processes the *D*-values and *z*-values either of the actual contaminants or of a model. *B. fulva* is a good model candidate because it has been

TABLE 2. *Main primary models applied to mold growth*

| Mold growth | Model | Reference(s) |
|---|---|---|
| Germination | Gompertz (eq. 1) | *2, 39, 50, 68, 85, 87* |
| | Logistic function (eq. 2) | *21, 22, 50, 110* |
| | Asymmetric model (eq. 3) | *20* |
| Mycelium proliferation | Baranyi model (eq. 4) | *3, 31, 32, 71, 72, 75, 83, 109, 111, 127, 128* |
| | Linear | *5, 13, 14, 18, 21, 44, 47, 51, 52, 57, 58, 60, 80, 85, 87, 88, 90, 110, 114, 115* |
| | Two-phase linear (eq. 5) | *37, 38, 70* |

reported to be the most heat-resistant mold *(113)*. Depending on the strain and the intrinsic matrix factors, its *D*-value is 1 to 12 min at 90°C and its *z*-value is 6 to 7.4°C *(9, 53, 113)*.

**Ethanol.** The inhibitory effect of ethanol has been reported for both mold germination *(24)* and mold proliferation *(18)*; ethanol either stops growth or delays it by disrupting the cell membrane *(49)*. During manufacture of food products, ethanol is used directly on the product (e.g., sprayed on the surface) or in the packaging atmosphere (ethanol vapor) *(23)*.

Dantigny et al. *(22)* found that at ethanol vapor concentrations >4%, conidia of *Penicillium chrysogenum* isolated from spoiled bakery products was inhibited and most of these cultures were arrested during the swelling stage (second stage of germination after activation *(27)*). However, the effect of ethanol was reversible under the experimental conditions.

For mold proliferation, Dantigny et al. *(18)* estimated minimal ethanol concentrations at which no growth would occur: close to 4% for *P. chrysogenum* and 3 to 5% for most of the other molds tested.

**Packaging.** The molds encountered in food spoilage are strict aerobes, i.e., oxygen is necessary for mold growth *(131)*. Consequently, limiting oxygen as a substrate for molds is an effective way to inhibit mold growth. However, some molds such as *P. roqueforti, Mucor plumbeus,* and some *Fusarium* are able to grow at atmospheric oxygen concentrations as low as 0.5% *(123)* or 2% *(108)*. Molds also are able to use oxygen from the matrix, not just from the atmosphere *(97)*. Therefore, oxygen depression in the atmosphere of packed food products must be combined with the toxic effect of another compound such as carbon dioxide or nitrogen *(120)*. In a study conducted with modified atmosphere packaging, proliferation of *A. niger, Eurotium amstelodami, P. chrysogenum,* and *Fusarium oxysporum* was completely inhibited under anaerobic conditions and was delayed under aerobic conditions (5% $O_2$) *(43)*. Growth of *Penicillium aurantiogriseum* was inhibited at $CO_2$ concentrations >70% in modified atmosphere packaging. Consequently, this packaging method is an effective way to prevent mold growth in packaged food products.

## MATHEMATICAL MODELS QUANTIFYING THE INFLUENCE OF FORMULATION, PROCESSING, AND ENVIRONMENTAL FACTORS ON MOLD GROWTH

**Primary models.** To quantify fungal development, both germination and mycelium proliferation data are analyzed, whereas for bacteria only growth is generally studied. Assessment of bacterial growth often consists of plating and counting CFUs, but other methods have been used to measure fungal development.

Germination is generally checked microscopically, and the number of germinated spores is plotted against time. A spore is considered germinated when the length of its longest germ tube is greater than the greatest dimension of the swollen spore *(16)*. Using primary models, the latency before germination ($\lambda_G$), the maximum percentage of germinated spores ($P_{max}$), and the germination time ($t_G$; the time for 50% of spores in the inoculum to germinate) are estimated. Three primary models are commonly used for describing germination (Table 2).

The modified Gompertz equation is

$$P_{(t)} = P_{max} \cdot \exp\left\{ -\exp\left[\frac{\mu_G \cdot e(1)}{P_{max}} \cdot (\lambda_G - t) + 1\right]\right\} \quad (1)$$

where $t$ (hours) is the time, $P_{(t)}$ is the percentage of germinated spores at time $t$, $P_{max}$ (percentage) is the asymptotic value at $t \rightarrow +\infty$, $\mu_G$ (percent per hour) is the slope term of the tangent line through the inflection point, and $\lambda_G$ (hours) is the geometrical latency ($t$ axis intercept of the tangent through the inflection point).

The logistic function is

$$P_{(t)} = \frac{P_{max}}{1 + \exp[k(\tau - t)]} \quad (2)$$

where $t$, $P_{(t)}$, and $P_{max}$ have the same definitions as above, $\tau$ (hours) is the inflection point where $P = P_{max}/2$ (with $\tau$ equal to the germination time $t_G$), and $k$ (percent per hour) is related to the slope of the tangent line through the inflection point. By deriving, the latency before germination $\lambda_G$ is extrapolated.

The asymmetric model is

$$P_{(t)} = P_{max}\left[1 - \frac{1}{1 + \left(\frac{t}{\tau}\right)^d}\right] \quad (3)$$

In addition to the parameters defined above, $d$ is a shape parameter. By deriving, the latency before germination $\lambda_G$ is extrapolated.

Mycelium proliferation is most commonly assessed by plotting the visible radial growth (colony diameter) against time. Alternative proliferation assessment such as measurement of ergosterol (a fungal cell membrane component) *(65)* and fungal biomass dry weight *(67)* have been tested but remain of marginal use. Visible growth data are fitted by primary models to estimate the latency before proliferation ($\lambda$) and the radial proliferation rate ($\mu$). These models are either linear or nonlinear (Table 2). In the linear model, the

lag phase and the possible final asymptote are removed. A linear regression is then performed on the straight part of the curve. The slope obtained equals the radial proliferation rate μ, and the latency λ is estimated over the interception between the regression line and the *x* axis. This method might be considered arbitrary because the data selection is manual, often done after visual inspection of the data set. The most commonly used nonlinear models are presented below.

The Baranyi model *(7)* model is

$$\begin{cases} D_{(t)} = D_0 + \mu \cdot A_{(t)} - \ln\left\{1 + \dfrac{[\exp(\mu \cdot A_{(t)}) - 1]}{\exp(D_{\max} - D_0)}\right\} \\ A_{(t)} = t + \left(\frac{1}{\mu}\right) \cdot \ln[\exp(-\mu \cdot t) + \exp(-\mu \cdot \lambda) \\ \qquad\qquad - \exp(-\mu \cdot t - \mu \cdot \lambda)] \end{cases} \quad (4)$$

where $t$ is the time (days), $D_{(t)}$ (millimeters) is the colony diameter at time $t$, $D_0$ (millimeters) is the initial colony diameter, λ is the latency (hours) before the increase in the colony diameter, μ (millimeters per hour) is the radial proliferation rate, and $D_{\max}$ (millimeters) is the maximum colony diameter (often equal to the diameter of the culture dish).

The two-phase model is

$$D_{(t)} = \begin{cases} D_0, & t \le \lambda \\ D_0 + \mu \cdot (t - \lambda), & t > \lambda \end{cases} \quad (5)$$

with the same parameter definitions as above.

**Secondary models.** Secondary models have been developed to quantify the effects of formulation, processing, and environmental factors on mycelium proliferation kinetics and to some extent on germination kinetics. Because of a lack of specific models dedicated to molds, models developed first for bacteria have been applied to mold growth *(17)*. Three types of models are commonly used: polynomial, cardinal, and logistic regression (Table 3).

The polynomial models have been applied more often than the others, and the general expression is given in equation 6:

$$Y = a_0 + \sum_{i=1}^{3} a_i \cdot x_i + \sum_{i=1}^{3} \sum_{j=1}^{3} a_{ij} X_i X_j \quad (6)$$

where $Y$ is the kinetic response (e.g., radial proliferation rate, μ), the coefficients $a_0$ and $a_i$ are the estimated parameters, and $X_i$ and $X_j$ are the variables (e.g., $a_w$ and temperature).

Based on this equation, Gibson et al. *(34)* developed a model to describe the effect of $a_w$ on the radial proliferation rate (μ) of *A. flavus*. They found that the logarithm of μ follows a hyperbolic shape with the square root of $(1 - a_w)$. This model was the first secondary model dedicated to molds (equation 7):

$$\ln(\mu) = a_0 + a_1 \cdot \sqrt{1 - a_w} + a_2 \cdot (1 - a_w) \quad (7)$$

where μ is the radial proliferation rate, $a_0$, $a_1$, and $a_2$, are constants to be estimated, and $a_w$ is the water activity.

Another polynomial model was used by Panagou et al. *(84)* to describe the effect of $a_w$, temperature, and pH on *Monascus ruber*. They modified the linear Arrhenius-Davey equation, which was originally applied to bacterial growth (equation 8):

$$\ln(\mu) = a_1 + a_2 \cdot \mathrm{pH} + a_3 \cdot \mathrm{pH}^2 + a_4 \cdot a_w + a_5 \cdot a_w{}^2 + \frac{a_6}{T} + \frac{a_7}{T^2} \quad (8)$$

where μ is the radial proliferation rate, $a_i$ ($i$ from 1 to 7) are model parameters, and $T$ is the temperature (Kelvin).

Because of potential colinearity among variables and because the constants estimated from polynomial models have no biological meaning, some researchers have moved to cardinal models. These models include cardinal values of extrinsic and intrinsic factors (e.g., $a_w$, pH, and temperature). Three cardinal values are generally involved for each factor: a minimum ($Y_{\min}$) below which no growth occurs, a maximum ($Y_{\max}$) above which no growth occurs, and an optimum ($Y_{\text{opt}}$) where the growth rate is maximal. The first model that was developed based upon the idea of cardinal values was the Ratkowsky et al. *(100)* square root model. First applied to describe the effect of temperature on bacterial growth, it was then transposed to mold proliferation and extended to $a_w$ by Tassou et al. *(124)* (equation 9):

$$\sqrt{\mu} = b \cdot (T - T_{\min}) \cdot \{1 - \exp[c \cdot (T - T_{\max})]\} \cdot \sqrt{(a_w - a_{w\,\min}) \cdot \{1 - \exp[d \cdot (a_w - a_{w\,\max})]\}} \quad (9)$$

where μ is the radial proliferation rate and $T$ is the temperature (degrees Celsius). The coefficients $a_{w\ \min}$, $a_{w\ \max}$, $b$, $c$, and $d$ are estimated parameters.

To get a step further in including cardinal values of factors to build a secondary model, Rosso et al. *(104)* proposed a cardinal model with inflection. Initially set to describe the effect of temperature, it was next applied to other factors ($a_w$ and pH), and a general form of the cardinal model with inflection was proposed (equations 10 and 11) *(103, 105)*:

$$r = r_{\text{opt}} \cdot CM_n(Y, Y_{\min}, Y_{\text{opt}}, Y_{\max}) \quad (10)$$

$$CM_n(Y, Y_{\min}, Y_{\text{opt}}, Y_{\max}) = \begin{cases} Y \le Y_{\min}, & 0 \\ Y_{\min} < Y < Y_{\max}, & \dfrac{(Y - Y_{\min})^n \cdot (Y - Y_{\max})}{(Y_{\text{opt}} - Y_{\min})^{n-1} \cdot \{(Y_{\text{opt}} - Y_{\min}) \cdot (Y - Y_{\text{opt}}) - (Y_{\text{opt}} - Y_{\max}) \cdot [(n-1) \cdot Y_{\text{opt}} + Y_{\min} - nY]\}} \\ Y \ge Y_{\max}, & 0 \end{cases} \quad (11)$$

where $Y$ is the factor (e.g., $a_w$, temperature, and pH), $Y_{\min}$, $Y_{\text{opt}}$, and $Y_{\max}$ are the minimum, optimum, and maximum values, respectively, for growth of this factor, $n$ is a shape parameter, $r$ is the kinetic parameter (e.g., μ), and $r_{\text{opt}}$ is the optimal value of $r$ in the product considering the factors studied.

Cardinal models that describe the effect of a compound (e.g., weak acid and ethanol) from its concentration also are used. The reparameterized Monod-type equation *(18)*

TABLE 3. *Secondary models describing the effect of formulation, processing, and environmental factors on different steps in mold growth*

| Model | Mold growth | Parameter[a] | Factor | Reference(s) |
|---|---|---|---|---|
| Polynomial (eqs. 6–8) | Germination | $\lambda_G$, $\mu_G$ | $a_w$, $T$[b] | *2* |
| | | $t_G$ | $a_w$, $T$ | *90* |
| | | % at $t$ = 25 days | $a_w$, $T$, pH | *117* |
| | | $t_{G90\%}$ | $a_w$, $T$, pH | *116* |
| | | $\lambda_G$, $\mu_G$ | $a_w$, $T$ | *85, 87* |
| | | % at $t$ = 4, 8, and 24 h | $a_w$, $T$ | *15* |
| | Mycelium proliferation | $\mu$ | $a_w$ | *34, 88, 105, 128* |
| | | $\mu$ | $a_w$, $T$ | *2, 15, 57, 58, 87, 90, 111, 112* |
| | | $\mu$ | $a_w$, $T$, [acid] | *63* |
| | | $\mu$ | $a_w$, $T$, pH | *84* |
| | | $\mu$ | $T$ shock, pH shock | *79* |
| | | $\lambda$, $\mu$ | $a_w$, $T$ | *32* |
| | | $\lambda$, $\mu$ | $T$, [acid] | *74* |
| | | $\lambda$, $\mu$ | $a_w$, pH | *127* |
| Gamma-type with cardinal values (eqs. 9–14) | Germination | $t_G$ | [ethanol] | *22* |
| | | $\lambda_G$, $\mu_G$ | $T$ | *39* |
| | Mycelium proliferation | $\mu$ | pH | *72* |
| | | $\mu$ | $a_w$ | *114* |
| | | $\mu$ | $T$ | *88* |
| | | $\mu$ | [ethanol] | *18* |
| | | $\mu$ | $a_w$, $T$ | *3, 32, 51, 83, 124* |
| | | $\mu$ | $a_w$, $T$, pH | *70, 80, 84* |
| | | $\lambda$, $\mu$ | $a_w$ | *64* |
| | | $\lambda$, $\mu$ | $T$ | *37, 38* |
| Logistic (eq. 15) | Mold growth | *G*/*NG* | $a_w$, $T$, inoculum size | *31* |
| | | *G*/*NG* | $a_w$, $T$ | *3, 32, 83* |

[a] $\lambda_G$, latency before germ tube formation during germination (hours); $\mu_G$, germination rate (percent per hour); $t_G$, germination time (time to germinate 50% of viable spores; hours); $t_{G90\%}$, time to germinate 90% of viable spores (hours); $\mu$, mycelium proliferation rate (millimeters per day); $\lambda$, latency before mycelium proliferation (hours); *G*/*NG*, growth/no growth boundary.
[b] $T$, temperature.

(equation 12) and the modified asymmetric model *(52)* (equation 13) have been developed specifically for molds. Here, the cardinal value is either the MIC of the compound ($MIC_i$) or the concentration where $\mu$ is divided by 2 ($C_{i50}$).

$$r = r_{opt}\frac{C_{i50}\cdot(MIC_i - C_i)}{C_{i50}\cdot MIC_i - 2C_{i50}\cdot C_i + MIC_i\cdot C_i} \quad (12)$$

$$r = r_{opt}\frac{1}{1+\left(\frac{C_i}{C_{i50}}\right)^d} \quad (13)$$

where $r$ and $r_{opt}$ have the same definition as above, $C_i$ is the concentration of the compound $i$, and $d$ is a shape parameter.

Cardinal models are also used when more than one factor must be modeled. In that case, the effects of the factors are assumed to be multiplicative. The secondary model built upon this approach is the gamma concept *(133)*. The general form of this model is presented in equation 14:

$$\mu = \mu_{opt}\cdot\gamma(a_w)\cdot\gamma(T)\cdot\gamma(pH) \quad (14)$$

In equation 14, each gamma term is parameterized such that its value ranges between zero and 1. At zero, the inhibition due to this factor is maximal, at 1 the inhibition is null. In the absence of inhibition, the growth rate of bacteria or the proliferation rate of molds, is at its maximal value, $\mu_{opt}$. Various functions for each gamma term have been developed for describing the effect of $a_w$, temperature, and pH on bacterial growth rate.

The general pattern of this model is flexible; it allows inclusion of a large set of mathematical expressions for each gamma term (Table 3) and addition of new gamma terms, for example to describe the effect of an inhibitory compound *(52)*.

Another type of secondary model is the growth/no-growth boundary model. Experimentally, the approach is simpler than that for other cases; the operators must record whether growth (value 1) or no growth (value 0) is obtained at a certain combination of extrinsic and intrinsic factors and after a certain time. To determine the growth/no-growth interface over the factors considered, the data obtained are fitted to a logistic regression *(101)*, often linked to a polynomial model (equation 15) *(8)*:

$$\text{logit}(P) = \ln\left(\frac{P}{1-P}\right) = a_0 + \sum_{i=1}^{3} a_i\cdot x_i + \sum_{i=1}^{3}\sum_{j=1}^{3} a_{ij}X_iX_j \quad (15)$$

where $P$ is the probability of growth (range of 0 to 1), the coefficients $a_i$ are estimated parameters, and the $X_i$ values are the independent factors (e.g., $a_w$, temperature, and pH).

**Concluding remarks on mathematical models.** Many primary and secondary models, often based upon those developed for pathogenic bacteria in the last 30 years, have been applied to

mold growth (Tables 2 and 3). Among them, few have been successfully validated with foods, with the exception of bakery products *(72)* and fruit juices *(83)*. However, biological strain diversity has not been quantified adequately, i.e., biological variability has not been characterized in detail.

Whatever the model mathematical expression, even if satisfactory results have been reported with the factor storage temperature and $a_w$, a model for quantifying the influence of pH seems to be more difficult to develop. For instance, with a gamma-type model Nevarez et al. *(80)* reported odd parameter values such as a $pH_{min}$ of $-2.13$. Despite these limitations, existing mathematical models can be incorporated in a general probabilistic framework to predict the shelf life of a food product.

## MODELING FUNGAL SHELF LIFE OF A FOOD PRODUCT

In terms of shelf life, there is a difference between the ''use by'' date and the ''best before'' date *(81)*. When food must be eaten within a certain period for health or safety reasons, a ''use by'' date is required. However, a ''best before'' date is applicable to food when deterioration affects consumer acceptance without impacting health and safety. Such deterioration includes rancidity, texture change, flavor loss, and microbial spoilage. In this review, the shelf life considered is that dictated by the ''best before'' date as it relates to fungal spoilage.

Fungal spoilage leads to product rejection by the consumer and consequently to severe economic losses for food companies. Consumer rejection depends on the product shelf life because mold proliferation is a dynamic phenomenon that varies with time and depends on the processing and formulation factors controlling mold growth. Consequently, the shelf life of a product potentially spoiled by mold will be a function of the processing and formulation characteristics, and the type of processing and formulation of a product depends on the shelf life.

**Mold spoilage assessment: probabilistic model framework.** Mold spoilage results when a product is contaminated with fungal spores that germinate and form a visible mycelium before the end of the shelf life (ESL). Mathematically, the spoilage can be expressed as a combination of probabilities: probability of being contaminated and probability of fungal growth (germination and proliferation) to a visible mycelium before the ESL (equation 16):

$$\Pr(\text{mold spoilage}) = \Pr(\text{in-factory contamination}) \times \Pr(\text{mold growth before ESL}) \quad (16)$$

where Pr is the probability function; Pr(in-factory contamination) is the prevalence of contaminating fungal spores, and Pr(mold growth before ESL) is the capacity of spores to germinate in or on the food product and then the ability of germinated spores to form a visible mycelium on the product before the ESL.

Use of a probabilistic framework to describe microbial food safety and stability is relatively common in public health risk assessment, at least with pathogenic bacteria. Risk-based food safety management must take into consideration the probability that the level of bacteria will exceed a given food safety objective, i.e., the maximum pathogen level allowed at the time of consumption, e.g., 2 log CFU/g for *Listeria monocytogenes* in deli meats in Europe *(28)*. This probability is calculated as a function of process, formulation, and distribution characteristics and the ability of the bacteria to grow under these formulation and distribution conditions.

Each term in equation 16 can be considered a statistical model, with a response (the probability) and the factors of variation. These factors are introduced in the model as single values when a deterministic approach is preferred or as a probability distribution function when a probabilistic approach is chosen. For mold spoilage assessment, there is variability in some process and environmental factors. For example, storage temperature varies with region, season, and consumer storage habits, and mold heat resistance characteristics vary with mold species or strain. Storage temperature and heat resistance characteristics should be then introduced in the assessment model as probability distribution functions. In contrast, formulation factors such as $a_w$, pH, or preservative concentrations may be considered fixed, i.e., chosen by the manufacturer, although small variations around the fixed points may be taken into account as uncertainty in the model.

In the food safety domain, many examples of quantitative microbial risk assessments based on probabilistic models have been published *(12, 54, 76, 125)*. By building on this experience, it seems feasible to develop a probabilistic modeling approach focusing on mold contamination and growth.

**Mold spoilage assessment: contamination during product manufacture.** For foods whose manufacture includes a heat treatment process, molds are generally eliminated during pasteurization. Raw materials also should be monitored to prevent direct reintroduction of contaminants during manufacture. However, molds are commonly found in manufacturing environments. Airborne spores have been reported as the most frequent route of contamination during food manufacturing *(56)*. Air-to-food transfer can occur through dust particles or via aerosols, especially during product packaging *(10, 26, 93)*.

In equation 16, Pr(in-factory contamination) corresponds to the prevalence of fungal spores in the product before the product is released to the market. For products that are packed before being distributed to retailers, this probability is a function of factors strictly linked to the factory design, processing, and environment, which can be summarized into three modules (equation 17):

$$\Pr(\text{in-factory contamination}) = f([\text{outside}] \text{ and } [\text{plant}] \text{ and } [\text{product}]) \quad (17)$$

For the [outside] module, microbial load in the air environment outside the factory is dependent upon many factors, including season, outside temperature, and the presence of crop fields and other factories nearby. For the [plant] module, inside the factory the concentration of

spores in the plant air depends upon the microbial load outside, the filtering efficacy of the heating, ventilation, and air-conditioning system, and the frequency of air changes within the production hall. The relative humidity and temperature also affect microbial survival on equipment surfaces and dissemination within the plant, and mold levels can be increased when there is excessive condensate and poor drying capacity. When air plant quality is monitored on a regular basis, data collected can be used for validating the [plant] module or as inputs in the [product] module. For the [product] module, the quantity of spores per product is obtained by multiplying the level of spores in the plant air ([plant]) by the microbial settling velocity, the exposure time (e.g., period between pasteurization and packaging), and the surface of exposure *(25)*. This value is likely to be low (e.g., 1 spore) or even very low (e.g., 0.01 spore). In such a case, it is interpreted as the Esperance of a Poisson distribution: E([product]).

Consequently, the probability of a contaminated product, Pr(in-factory contamination), is directly derived from the cumulative distribution function as follows (equation 18):

$$\Pr(\text{in-factory contamination}) = 1 - \Pr(\text{product free of spores}) = 1 - e^{-[\text{product}]} \quad (18)$$

**Mold spoilage assessment: germination and mycelium proliferation.** Once the product is contaminated, rejection of the product by the consumer will depend upon the ability of the fungal spores to germinate and form a visible mycelium before the ESL.

Two scenarios are possible. In scenario 1, the mold contamination on the product is scarce, the spores are spread over the product, and it is unlikely that more than a few spores will be settle on the same spot. In such a case, models applicable for a single spore should be applied and when possible can be extended to a few spores. In scenario 2, the mold contamination on the product is high, and the germination and mycelium proliferation must be analyzed at the population level.

Both scenarios are mathematically detailed below. However, with mold contamination coming from the air and good hygienic practices in place in the factory, the probabilistic model framework based on a single spore or a few spores spread all over the product is probably more appropriate for calculating mold spoilage and designing product shelf life.

For a single spore, theoretically the germination is described by two parameters: latency before germination and germination rate. However, so far mathematically for a single spore, only the time to germination (latency + germination time) has been determined *(110)*. Proliferation is described with one parameter, the proliferation rate (equations 4 and 5) because there is no latency before hyphal elongation; it starts without any delay after the germination of the spore. Morphologically, germination and elongation of a spore are two successive and continuous processes *(131)*.

Consequently, the probability of mold growth before ESL corresponds to the probability of one spore germinating ($G$) and of hyphal elongation ($E$) up to a visible spot ($V$) before ESL (equation 19):

$$\Pr(\text{mold growth before ESL}) = \Pr_{1\ \text{spore}} G \cap E \text{ up to } V, \text{ before ESL} \quad (19)$$

To solve equation 19 for various processing, formulation, and environmental conditions, primary model and secondary models applied to the three parameters (latency before germination, germination rate, and proliferation rate) are required. These secondary models are similar to those listed in Table 3.

The model system might be simplified (one or two parameters removed) when a high correlation between latency before germination and germination rate or between germination rate and hyphal elongation rate can be established. Gougouli and Koutsoumanis *(38, 39)* pointed out that the temperature effect on proliferation rate and germination rate was relatively similar (same $T_{opt}$ value and close $T_{min}$ and $T_{max}$; equations 10 and 11). However, this similarity must be confirmed for other controlling factors; Abellana et al. *(2)* found that the observed $a_w$ minima for germination of some *Eurotium* species were lower than those for mycelium proliferation.

When a few spores are spread all over the product, the probability of mold growth before the ESL corresponds to the probability of at least one spore germinating and of hyphal elongation up to a visible spot before ESL (equation 20):

$$\Pr(\text{mold growth before ESL}) = 1 - \left(1 - \Pr_{1\ \text{spore}} G \cap E \text{ up to } V, \text{ before ESL}\right)^{\text{number of spores}} \quad (20)$$

The same secondary model system (with three, two, or one parameter of interest) as used for the one-spore equation system is required. This scenario of one or few spores is likely to be more appropriate for calculating mold spoilage and designing shelf life.

However, when the mold contamination on the product is high and multiple spores are aggregated on one spot, the germination and mycelium proliferation must be analyzed at the population level. Many studies at the population level have been conducted. Generally, researchers have developed models for describing either germination (latency and germination rate; equation 1) or germination time ($t_G$, equations 2 and 3) or mycelium proliferation rate. In theory, based upon the understanding of the germination and elongation process at the individual spore level, a fourth parameter is not needed. However, with large numbers of spores in the inoculum, a latency before mycelium proliferation (Fig. 1) sometimes also must be analyzed with a secondary model.

This additional parameter might be directly connected to germination. Dantigny et al. *(21)* found that the time to germination of more than 99% of the viable spores of *Mucor racemosus* corresponded to the latency before mycelium proliferation. This experiment was conducted on laboratory medium under optimal conditions with a standardized spore

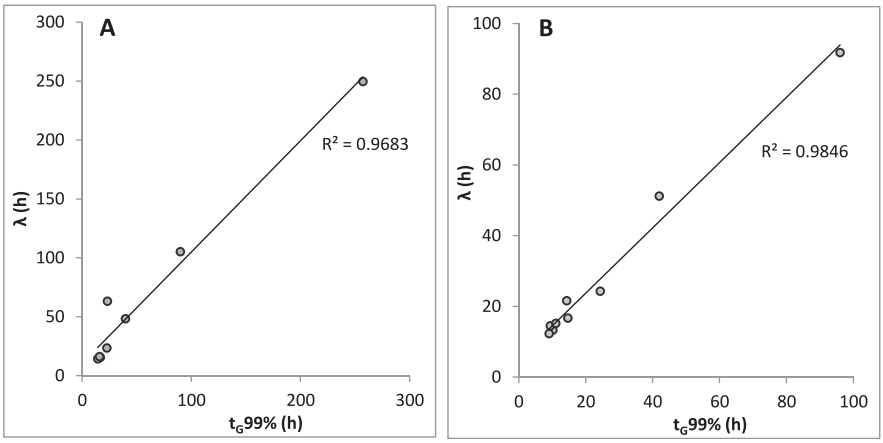


FIGURE 2. *Comparison between the latency before mycelium proliferation (λ) and the time to germinate 99% of the spores ($t_G$99%) for* Penicillium expansum *(A) and* Aspergillus niger *(B) at different temperatures (38, 39).*

inoculum. To investigate this parameter further, published data were reanalyzed. Gougouli and Koutsoumanis *(39)* established the spore germination percentage over the time for *Penicillium expansum* and *A. niger*. In 2010 the same team working with the same two strains estimated the latency before hyphal elongation, based on primary and secondary models describing the mycelium proliferation *(38)*. It was then possible to make a direct comparison between the time to obtain 99% spore germination and the latency before hyphal elongation (Fig. 2). The general pattern seems to indicate a high colinearity (the longer the time to obtain 99% germination, the longer the latency) or even an equality between these two phenomena (slope of 0.97 and 0.98 for *P. expansum* and *A. niger*, respectively).

If this pattern is confirmed, the probability of mold growth before the ESL corresponds to the probability of germination of 99% of the spores ($G_{99\%}$) combined with hyphal elongation up to a visible spot before the ESL (equation 21):

$$\Pr(\text{mold growth before ESL}) = \left(\Pr_{\text{aggregated spores}}\ G_{99\%} \cap E \text{ up to } V, \text{ before ESL}\right) \quad (21)$$

The probability of germination of 99% of the spores should be deduced from a single-cell level observation in which the latency before germination and the germination rate are studied as a function of the processing, formulation, and environmental factors. Variables at the population level should be derived from variables observed at a single-cell level when they are independent and normally distributed *(110)*. However, under some suboptimal conditions Judet et al. *(50)* found that distribution of the germination times does not necessarily follow a perfect symmetrical shape and then not a normal distribution. With these two contradictory results, it is difficult to make a definitive conclusion; the mathematical pathway from single-spore level to population level should be investigated further before using this information to estimate the shelf life.

An alternative is to deduce the probability of germination of 99% of the spores from a primary model. As far as we know, no primary model had been designed to do this (no model with a $G_{99\%}$ parameter), and some researchers have stated that the models presented in equations 1, 2, or 3 are not suitable for this purpose *(20)*. Consequently, the last alternatives are to determine the time needed to germinate 99% of the spores experimentally by direct observation of standardized inoculum spots under the microscope or to develop a new predictive model.

Whichever alternative is chosen, a secondary model describing $G_{99\%}$ as a function of the processing, formulation, and environmental factors during manufacture is required. This model should be slightly different from most of those already described (Table 3) because it includes a different response ($G_{99\%}$ instead of latency before germination, germination rate for one spore, or germination time) but is relatively similar to the model developed for $t_{G90\%}$ *(116)*.

In contrast, hyphal elongation at the population level cannot be observed directly because the mycelium created from more than 100 spores forms an inextricable web *(45)*. However, in cases of contamination, the mycelium proliferation will extend mainly from the external bound of the inoculums spot, and the proliferation rate, μ, will be then deduced from the primary models listed in Table 3.

The probability of mold growth (germination plus proliferation) up to a visible mycelium before the ESL will be derived from the hyphal elongation and more precisely from the mycelium diameter. The mycelium diameter is a statistical response described by a normal law as $N(D_{(t)}, \varepsilon)$, where ε is the residual error and $D_{(t)}$ is obtained by the primary and secondary models mentioned earlier.

The normal distribution, $N(D_{(t)}, \varepsilon)$, can be calculated at $t = 0, 1, \ldots,$ ESL, i.e., at any time from zero to the ESL and compared with a visible limit (equation 22):

$$\Pr(\text{mold growth before ESL}) = \Pr\left\{N\left[D_{(t)},\varepsilon\right] \geq \text{visible limit}\right\} \quad (22)$$

This calculation is often too complex to be solved analytically. In that case, numerical methods are required.

To conclude the mold spoilage quantitative assessment, the scenario based on one or a few spores contaminating the product is likely to be the most realistic at least for processed foods. However, whatever the contamination scenario chosen, mathematical primary and secondary models must be handled with care before being incorporated into a probabilistic framework for estimating the food shelf life.

Spore germination and mycelium proliferation are successive and therefore not independent events. The choice of the statistical parameters (and associated secondary models) to describe these events when assessing mold spoilage and suggesting management options for preventing mold growth (e.g., to run what-if scenarios in a Monte Carlo simulation procedure) is essential.

**Management options for preventing mold spoilage in food products.** Management options for preventing mold spoilage are based on factors that influence either spore contamination or mold growth and that can be controlled by the risk managers as processing and formulation settings. However, some factors that influence mold growth cannot be considered controlled by the manufacturer. For instance, the conditions of spore production have an effect on mold growth. Conidia of *P. chrysogenum* produced at 0.95 $a_w$ had a lower germination time (i.e., time for 50% of the spore inoculum to germinate) and a higher proliferation rate than those conidia produced at 0.99 $a_w$ *(50)*. Likewise, the older a spore is, the later the germination occurs *(19)*. These two factors (spore production condition and spore age) cannot be assimilated into management options.

When preventing food products from becoming spoiled by mold, in-factory contamination is the major factor to be controlled, and considerable costs are involved in assuring control of recontamination operationally through adherence to hygienic good manufacturing practices and hazard analysis critical control point programs. Reaching a contamination rate of zero during food manufacturing is rarely feasible. However, reducing the rate to an acceptable threshold remains conceivable. Heating, ventilation, and air-conditioning systems in factories have a multitude of purposes, but their role in filtering the air and controlling the temperature is critical to controlling risks of contamination by both fresh and recirculated air contaminants. Risk managers can decide on the temperature, the filtering efficacy, and the air change frequency to improve the performance of the heating, ventilation, and air-conditioning systems. Simpler plant designs such as in-plant positive pressure systems also have been effective for reducing air mold contamination, although they must be installed where the product is exposed to the air, for example in the packaging area *(40)*.

The microbial air plant quality (the [plant] module in equation 17) depends also upon the equipment surfaces on which mold may survive or be disseminated within the plant, e.g., when there is excessive condensate. Standardized cleaning and disinfection programs and good manufacturing practices have been reported to be sufficient for preventing mold survival on surfaces *(56)*.

In addition to controlling the prevalence of contamination of the product at its release to the market, mold growth also should be prevented, which might be done by appropriate design of processing factors and formulation. Generally, the controlling factors of interest are $a_w$, temperature, and the presence of preservatives. The use of lactic acid bacteria or plant extracts might be solutions to consider. In addition to these controlling factors based upon product formulation, the choice of a specific packaging type might help to prevent mold growth.

Unfortunately, not all of the controlling factors have been studied and incorporated into ready-for-use secondary models. Intraspecies variability also has not been studied extensively, model validation is lacking for many food products. The risk associated with mold contamination and growth cannot be calculated whatever the scenario, i.e., whatever the factor or the strain, without this basic information.

For a specific product, when the main contaminating mold species are known secondary models can be developed for the controlling factors of interest. In that case, the probabilities of mold spoilage at a given ESL and at various levels of the factors can be calculated and compared. Consequently, risk reduction measures can be suggested on the basis of the probabilistic model outputs, which might be presented in isocontour plots *(1)* showing equal probability lines (1 in 1,000, 1 in 10,000, 1 in 100,000, etc.) for different processing and formulation conditions at a given ESL, similar to what has been done with pathogenic bacteria *(73)*. From current factory processing and formulation settings, management options for reducing the risk of mold spoilage are designed by moving to the most stringency domain of the graph.

## CONCLUSIONS

Mold spoilage is a major problem in the food industry, resulting in significant losses of popular foods such as bakery products and postharvest fruits. To prevent consumer rejection of product spoiled by mold, it is important to assess the probability for a given product to be microbiologically spoiled before the end of its shelf life. This probability depends upon the product in-factory contamination level and the ability of the mold spores to grow during the distribution and storage supply chain steps. Germination and mycelium proliferation have been described by primary and secondary predictive models adapted from models developed during the last 30 years with bacteria in the field of predictive microbiology. Consequently, the probability of germination and then mycelium proliferation before the ESL can be quantified as a function of storage temperature, $a_w$, and the presence of commonly used preservatives (e.g., potassium sorbates). With a risk of mold spoilage quantified, the product formulation can be designed to achieve a given shelf life, and vice versa the shelf life can be adjusted based on the product formulation.

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