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Research Note

Inhibitory Activity of Phosphates on Molds Isolated from Foods and Food Processing Plants

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ABSTRACT

Six commercial phosphates were evaluated for inhibition of the growth of 17 molds isolated from food sources. The assays were performed at neutral and natural (without pH adjustment) pH values, and the molds were streaked on plate count agar with added phosphates. Phosphate concentrations of 0.1, 0.3, 0.5, 1.0, and 1.5% (wt/vol) were used, and the MIC was determined. The resistance of molds to phosphates depended on the species. At a neutral pH, *Aspergillus ochraceus* and *Fusarium proliferatum* were resistant to all phosphates at all concentrations assayed, and *Byssochlamys nivea*, *Aureobasidium pullulans*, and *Penicillium glabrum* were most sensitive. The most inhibitory phosphates were those with chain lengths greater than 15 phosphate units and the highest sequestering power. At natural pH values (resulting from dissolving the phosphate in the medium), inhibitory activity changed dramatically for phosphates that produced alkaline or acidic pH in the medium. Phosphates with alkaline pH values (sodium tripolyphosphate of high solubility, sodium tripolyphosphate, and sodium neutral pyrophosphate) were much more inhibitory than phosphates at a neutral pH, but sodium acid pyrophosphate (acidic pH) had decreased inhibitory activity. The results indicate that some phosphates could be used in the food industry to inhibit molds linked to food spoilage.

Phosphates are additives widely used in the food industry (mainly meat and dairy processes) to protect flavor and increase yields because of their water retention and emulsifying capacities. They are generally recognized as safe, and in the meat industry they are used to enhance four major functional properties: increasing water binding in meats, enhancing emulsification, retarding oxidative rancidity and color deterioration, and enhancing cured-color development *(17)*. However, phosphates are not used specifically to inhibit microorganisms *(17)*, although some antimicrobial and antibotulinal activities have been reported *(24, 25, 27)*. More information about antimicrobial activity could allow expansion of the application of phosphates in the food industry. In several studies, phosphates have had inhibitory effects linked to the chelation of structurally essential metal ions *(6, 7, 12–14, 16, 22)*. Inhibition of bacterial growth by polyphosphates has been the subject of several investigations *(15, 18, 23)*, and in general gram-positive bacteria appear to be more sensitive than gram-negative bacteria to the effects of phosphates.

Very little is known about the effect of phosphates on mold growth, although there is evidence that phosphates may interfere with certain stages of mold metabolism, such as cell differentiation, sporulation, and production of toxins and antibiotics *(17)*. Molds are undesirable contaminants in the food industry, affecting raw materials and processed foods *(19, 20)*. The aim of this work was to obtain data on the inhibitory effect of commonly used food-grade phosphates on different molds associated with food processing plants.

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MATERIALS AND METHODS

Microorganisms and culture conditions. Seventeen molds (Tables 1 and 2) were isolated from air, surfaces of equipment, and processed food products in the region of Santa Fe, Argentina. Molds were grown on plates of malt extract agar with added chloramphenicol (100 mg/liter) and then purified according to the method of Basílico et al. *(3)*.

Phosphates. Six available food-grade phosphates (SUDAMFOS S.A., Argentina) with different chain lengths were used: polyphosphates A and B (15 to 20 phosphate units), sodium tripolyphosphate of high solubility (TAS), sodium tripolyphosphate (TRI; $Na_5P_3O_{10}$), sodium acid pyrophosphate (PAS; $Na_2H_2P_2O_7$), and sodium neutral pyrophosphate (N; $Na_4P_2O_7$).

Inhibition assays. Plate count agar (PCA) was used as the basal medium *(15)* to which various concentrations of phosphates (0.1, 0.3, 0.5, 1.0, and 1.5%, wt/vol) were added. Stock phosphate solutions (5%, wt/vol) were prepared in distilled water and sterilized by filtration (0.45-μm-pore-diameter membranes; Millipore, Bedford, Mass.) *(13)*. PCA-phosphate media were prepared using distilled water and the necessary volumes of stock solutions to reach the final phosphate concentrations needed. Media were used at two pH conditions: neutral and natural (without pH adjustment)

TABLE 1. *Phosphate inhibitory activity on molds isolated from food sources (neutral pH)*[a]

Organism	Phosphates (%, wt/vol)[b]: A 0.1	A 0.3	A 0.5	A 1.0	B 0.1	B 0.3	B 0.5	B 1.0	TAS 0.5	TAS 1.0	TAS 1.5	PAS 0.5	PAS 1.0	PAS 1.5	TRI 0.5	TRI 1.0	TRI 1.5	N 0.5	N 1.0	N 1.5
Aspergillus ochraceus	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
Fusarium proliferatum	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
Absidia corymbifera	+	+	+	+	+	+	±	−[c]	+	+	+	+	+	+	+	+	+	+	+	+
Fusarium sp.	+	+	+	±	+	+	+	±	+	+	+	+	+	+	+	+	+	+	+	+
Cladosporium sphaespermum	+	+	+	−[c]	+	+	±	−[c]	+	+	±	+	+	±	+	+	+	+	+	+
Penicillium chrysogenum	+	+	±	−[c]	+	+	±	−[c]	+	+	±	+	+	+	+	+	±	+	+	+
P. roquefortii	+	+	+	−[c]	+	+	±	−[c]	+	+	+	+	+	+	+	+	+	+	+	+
P. commune	+	+	+	+	+	+	±	−[c]	+	+	±	+	+	±	+	+	±	+	+	+
Aspergillus niger	+	+	±	−[c]	+	±	−[c]	−	+	±	−[c]	+	±	−[c]	+	±	−[c]	+	+	±
Trichoderma viride	+	±	−[c]	−	+	−[c]	−	−	+	±	−[c]	+	+	±	+	±	−[c]	+	±	−[c]
Epicoccum nigrum	±	−[c]	−	−	±	−[c]	−	−	+	±	−[c]	+	+	±	+	+	±	+	+	−[c]
Rhizopus stolonifer	+	−[c]	−	−	±	−[c]	−	−	+	−[c]	−	+	−[c]	−	+	−[c]	−	+	±	−[c]
Aspergillus candidus	+	±	−[c]	−	+	±	−[c]	−	+	±	−[c]	+	±	−[c]	±	−[c]	−	+	+	±
Phoma glomerata	±	−[c]	−	−	+	−[c]	−	−	+	±	−[c]	+	±	−[c]	+	±	−[c]	+	+	−[c]
Byssochlamys nivea	±	−[c]	−	−	±	−[c]	−	−	±	−[c]	−	±	−[c]	−	±	−[c]	−	±	−[c]	−
Aureobasidium pullulans	+	−[c]	−	−	+	−[c]	−	−	−[c]	−	−	±	−[c]	−	−[c]	−	−	+	−[c]	−
Penicillium glabrum	+	−[c]	−	−	+	−[c]	−	−	±	−[c]	−	+	−[c]	−	±	−[c]	−	+	−[c]	−
% molds inhibited	17.6	47.0	59.0	76.4	17.6	53.0	82.3	88.2	17.6	52.9	70.6	11.8	41.2	64.7	23.5	47.0	64.7	5.9	29.4	53.0

[a] +, growth (relative growth index [RGI], 0.8 to 1); ±, weak growth (RGI, 0.2 to 0.8); −, no growth (RGI, <0.2).

[b] A and B, polyphosphates (15 to 20 phosphate units); TAS, sodium tripolyphosphate, high solubility; PAS, sodium acid pyrophosphate; TRI, sodium tripolyphosphate; N, sodium neutral pyrophosphate.

[c] MIC.

TABLE 2. *Phosphate inhibitory activity on molds isolated from food sources (natural pH)*[a]

Organism	Phosphates (%, wt/vol)[b]: TAS 0.5	TAS 1.0	TAS 1.5	PAS 0.5	PAS 1.0	PAS 1.5	TRI 0.5	TRI 1.0	TRI 1.5	N 0.5	N 1.0	N 1.5
Aspergillus ochraceus	+	+	+	+	+	+	+	+	±	+	+	−
Fusarium proliferatum	+	+	+	+	+	+	+	+	±	+	+	±
Absidia corymbifera	+	+	±	+	+	+	+	+	−[c]	+	−[c]	−
Fusarium sp.	+	−[c]	−	+	+	+	+	−[c]	−	+	±	−[c]
Cladosporium sphaespermum	+	±	−[c]	+	+	+	+	±	−[c]	±	−[c]	−
Penicillium chrysogenum	+	±	−[c]	+	+	+	+	±	−[c]	+	−[c]	−
P. roquefortii	+	−[c]	−	+	+	+	+	−[c]	−	+	±	−[c]
P. commune	+	−[c]	−	+	+	+	+	−[c]	−	+	±	−[c]
Aspergillus niger	±	−[c]	−	+	+	+	±	−[c]	−	±	−[c]	−
Trichoderma viride	−[c]	−	−	+	+	+	−[c]	−	−	−[c]	−	−
Epicoccum nigrum	−[c]	−	−	+	+	+	−[c]	−	−	−[c]	−	−
Rhizopus stolonifer	−[c]	−	−	+	+	+	−[c]	−	−	−[c]	−	−
Aspergillus candidus	±	−[c]	−	+	+	+	±	−[c]	−	±	−[c]	−
Phoma glomerata	−[c]	−	−	+	+	+	−[c]	−	−	−[c]	−	−
Byssochlamys nivea	−[c]	−	−	+	+	+	−[c]	−	−	−[c]	−	−
Aureobasidium pullulans	−[c]	−	−	+	+	+	−[c]	−	−	−[c]	−	−
Penicillium glabrum	±	−[c]	−	+	+	±	−[c]	−	−	−[c]	−	−
% molds inhibited	52.9	82.3	88.2	0	0	5.9	52.9	82.3	100	58.8	88.2	100

[a] Natural pH indicates that no pH adjustment was made. +, growth (relative growth index [RGI], 0.8 to 1); ±, weak growth (RGI, 0.2 to 0.8); −, no growth (RGI, <0.2).

[b] A and B, polyphosphates (15 to 20 phosphate units); TAS, sodium tripolyphosphate, high solubility; PAS, sodium acid pyrophosphate; TRI, sodium tripolyphosphate; N, sodium neutral pyrophosphate.

[c] MIC.

(Table 3). Neutral pH values were obtained by addition of 6 N solutions of NaOH or HCl, according to the phosphate used. Mold conidia suspensions were prepared by a standard method *(2)* in peptone water (0.1%, wt/vol) + Tween 80 (0.01%, wt/vol), streaked onto PCA-phosphate plates according to the ecometric method *(1),* and incubated for 7 days at 28°C. Results were expressed as relative growth indices (RGIs). These indices were calculated as the number of streaks (from a total of 25) grown on a plate of PCA-phosphate medium divided by the number of streaks grown on a plate of PCA medium (control). Among the phosphate concentrations assayed, the MICs were determined from plates where no growth was observed (RGI < 0.2).

TABLE 3. *Physicochemical characteristics of the phosphates used in this study*

Phosphate[a]	pH of plate count agar–phosphate (%, wt/vol) media 0.1	0.3	0.5	1.0	1.5	Sequestering power (g Ca/100 g phosphate)[b] pH 7.0	Natural pH
A	6.81	6.79	6.72	6.59	6.45	15	15
B	6.81	6.78	6.68	6.64	6.59	15	15
TAS	7.00	7.13	7.17	7.35	7.48	12	13
PAS	6.17	5.77	5.55	5.17	5.04	3	NP[c]
TRI	7.04	7.27	7.44	7.42	7.64	12	13
N	7.28	7.78	7.86	8.14	8.31	5	8

[a] A and B, polyphosphates (15 to 20 phosphate units); TAS, sodium tripolyphosphate, high solubility; PAS, sodium acid pyrophosphate; TRI, sodium tripolyphosphate; N, sodium neutral pyrophosphate.

[b] Values for 1.2% phosphate (data provided by the manufacturer). Natural pH indicates that no pH adjustment was made.

[c] NP, not provided.

RESULTS

The resistance of molds to phosphates was dependent on species (Tables 1 and 2). At neutral pH (Table 1), *Aspergillus ochraceus* and *Fusarium proliferatum* were resistant to all the phosphates and concentrations assayed, whereas *Byssochlamys nivea, Aureobasidium pullulans,* and *Penicillium glabrum* were the most sensitive. The most inhibitory phosphates were those with chain lengths greater than 15 phosphate units (polyphosphates A and B); they were able to inhibit (including ± and negative results) the growth of 76.4% and 88.2% of molds, respectively, at a concentration of 1%. The other phosphates at this concentration inhibited only from 29.4% (N) to 52.9% (TAS) of molds. All molds were inhibited by 1.5% phosphates A and B (data not shown), but none were inhibited by concentrations lower than 0.5% for phosphates TAS, PAS, TRI, and N (data not shown).

At natural pH (Table 2), the inhibitory activity of phosphates changed dramatically, except for phosphates A and B, whose solutions had pH values close to neutrality (Table 3). The sequestering power (grams of Ca per 100 grams) of phosphates A and B was the highest of the phosphates assayed and had the same value at neutral and natural pHs (Table 3). PAS was acidic in solution (Table 3) and lost the inhibitory activity it had at pH 7.0 with no inhibition of

molds at the highest concentration of 1.5%. This phosphate also had poor sequestering power (Table 3). In contrast, TAS, TRI, and N had increased inhibition against molds. For TAS (1.5%), the percentage of molds inhibited increased from 70.6% (pH 7.0) to 88.2% (pH 7.5), whereas TRI and N were the most inhibitory, with inhibition of all molds at 1.5%. This increase in the inhibitory activity may be related to the higher sequestering power exhibited by these three phosphates at natural pH (alkaline solutions), especially for N (Table 3). All molds were capable of normal growth on PCA adjusted to pH values in the range 5.0 to 8.5.

DISCUSSION

The antimicrobial activity of phosphates has received relatively little attention. The inhibitory activity of phosphates may be related to factors such as changes in pH or withdrawal of metal cations through chelation *(24).* Gram-positive bacteria are more susceptible than gram-negative bacteria to inhibition by various pyro- and polyphosphates *(4, 15, 18, 26, 29),* and young cultures of both gram-positive and gram-negative bacteria are more sensitive than cultures grown for 24 h or more *(18).*

The mechanisms of inhibition of gram-positive bacteria by phosphates have been studied. The addition of certain polyvalent metal ions, such as Mg^{2+}, Ca^{2+}, and Fe^{2+}, can reverse the inhibitory effects of polyphosphates on microorganisms *(6, 7, 13, 16, 28).* This suggests that the capacity of phosphates for complexing metal ions (sequestration), which are essential for cell life, may be responsible for the antimicrobial activity.

Long-chain phosphates exert a greater inhibitory effect on bacterial growth than do phosphates with shorter chains *(12, 18, 26),* possibly because the sequestering activity increases with chain length *(17)*; however, data on effects on molds are still scarce *(7, 11, 21).* Food raw materials and products can be contaminated with spores or conidia and mycelial fragments from the environment. Contamination can occur at different stages of production and fungal growth occurs only under favorable conditions, which vary among species *(19, 20).* One or more fungal species used in this study are commonly linked to spoilage of dairy foods *(3, 5, 8–10, 19, 20)*; meat and processed meats; dried-, concentrated-, salt-, and heat-processed acid foods; fruits; cereals; nuts and oil seeds *(19, 20).* In our study, there was greater inhibition of molds from food sources when polyphosphates A and B (with the highest chain lengths and sequestering power, 15 g Ca/100 g) were used (1.5%, wt/vol) at a neutral pH. Conversely, sodium tripolyphosphates (TAS and TRI, 12 g Ca/100 g) and sodium pyrophosphates (PAS and N, 3 to 5 g Ca/100 g), with shorter chains lengths, had lower inhibitory activity (53 to 70.6% of molds inhibited with 1.5% phosphate). These results indicate that the anti-mold inhibitory activity of phosphates at pH 7 may be related directly to chain length and sequestering power. Post et al. *(21),* using only three molds and four phosphates of different chain lengths, found inhibition of mold growth at 5% phosphate and pH 5.6; sodium tetraphosphate was the most effective, followed by polyphosphate (SPG) and sodium tripolyphosphate. Lebron et al. *(11)* found that SPG was more inhibitory than tetrasodium pyrophosphate against germination of *Aspergillus flavus* and *Aspergillus parasiticus* conidia. At 1% phosphate, there was visible growth after 3 days (tetrasodium pyrophosphate) and 6 days (SPG) for both molds, whereas at 2% phosphate, spore germination was inhibited until day 9. Lebron et al. did not report the pH values of phosphate-containing media. Our results demonstrated that the pH of the medium had a marked influence on the inhibitory activity of phosphates. When the inhibition assays were carried out at natural pH (resulting from dissolving phosphate in the medium), PAS had acidic pH values and lost the inhibitory capacity it demonstrated at pH 7.0. Conversely, solutions of TAS, TRI, and N produced alkaline pH values (7.17 to 8.31 for 0.5 to 1.5% phosphate), which noticeably increased their inhibitory activity against molds. This increased activity may be a consequence of increased capacity to sequester divalent cations at alkaline pH values, because the growth of molds was normal when the medium pH was adjusted to similar values using NaOH. Solutions of phosphates A and B had pH values near neutrality, and their inhibitory activities were identical to those exhibited at pH 7. The effect of pH was studied by Jen and Shelef *(6)* for *Staphylococcus aureus* 196E, using 0.5% sodium tripolyphosphate and sodium hexametaphosphate (SPG). They also observed an increased inhibition by phosphates at higher pH values (7.4 to 9) and hypothesized that chelators became increasingly dissociated and the quantity of complex cations increased. As a result, essential minerals became unavailable for bacterial growth and, in turn, inhibition was enhanced. Fungal cell walls possess anionic polymers such as chitin, chitosan, and glycoproteins, which are involved in the passive uptake of metals *(7).* Thus, the inhibition of molds by phosphates could be attributed to the removal of essential metal cations from unique cation-binding sites located in fungal cell walls. The results of this study suggest that some phosphates could be used in the food industry to inhibit molds linked to food spoilage at concentrations similar to or lower than those used when phosphates are applied as additives *(17).*

REFERENCES

1. Anonymous. 1987. Testing methods for use in quality assurance of culture media. *Int. J. Food Microbiol.* 5:291–296.
2. Association of Official Analytical Chemists. 1984. Official methods of analysis of the Association of Official Analytical Chemists, 14th ed. Association of Official Analytical Chemists, Washington, D.C.
3. Basílico, J. C., M. Z. de Basílico, C. Chieratti, and C. G. Vinderola. 2001. Characterization and control of thread mould in cheese. *Lett. Appl. Microbiol.* 32:419–423.
4. Chen, T. C., J. T. Culotta, and W. S. Wang. 1973. Effect of water and microwave energy precooking on microbial quality of chicken parts. *J. Food Sci.* 38:155.
5. Hocking, A. D., and M. Faedo. 1992. Fungi causing thread mould spoilage of vacuum packaged Cheddar cheese during maturation. *Int. J. Food Microbiol.* 16:123–130.
6. Jen, C. M., and L. A. Shelef. 1986. Factors affecting sensitivity of *Staphylococcus aureus* 196E to polyphosphates. *Appl. Environ. Microbiol.* 52:842–846.
7. Knabel, S. J., H. W. Walker, and P. A. Hartman. 1991. Inhibition of *Aspergillus flavus* and selected gram-positive bacteria by chelation

of essential metal cations by polyphosphates. *J. Food Prot.* 54:360–365.
8. Kure, C. F., and I. Skaar. 2000. Mould growth on the Norwegian semihard cheeses Norvegia and Jarlsberg. *Int. J. Food Microbiol.* 62:133–137.
9. Kure, C. F., I. Skaar, and J. Brendehaug. 2004. Mould contamination in production of semi-hard cheese. *Int. J. Food Microbiol.* 93:41–49.
10. Kure, C. F., Y. Wasteson, J. Brendehaug, and I. Skaar. 2001. Mould contaminants on Jarlsberg and Norvegia cheeses from four factories. *Int. J. Food Microbiol.* 70:21–27.
11. Lebron, C. I., R. A. Molins, H. W. Walker, A. A. Kraft, and H. M. Stahr. 1989. Inhibition of growth and aflatoxin production of aspergilli in medium containing phosphates. *J. Food Prot.* 52:4–6.
12. Lee, R. M., P. A. Hartman, D. G. Olson, and F. D. Williams. 1994. Bactericidal and bacteriolytic effects of selected food-grade phosphates, using *Staphylococcus aureus* as a model system. *J. Food Prot.* 57:276–283.
13. Lee, R. M., P. A. Hartman, D. G. Olson, and F. D. Williams. 1994. Metal ions reverse the inhibitory effects of selected food-grade phosphates in *Staphylococcus aureus. J. Food Prot.* 57:284–288.
14. Lee, R. M., P. A. Hartman, H. M. Stahr, D. G. Olson, and F. D. Williams. 1994. Antibacterial mechanism of long-chain polyphosphates in *Staphylococcus aureus. J. Food Prot.* 57:289–294.
15. Loessner, M. J., S. K. Maier, P. Schiwek, and S. Scherer. 1997. Long-chain phosphates inhibit growth of *Clostridium tyrobutyricum* in processed cheese spreads. *J. Food Prot.* 60:493–498.
16. Maier, S. K., S. Scherer, and M. Loessner. 1999. Long-chain polyphosphate causes cell lysis and inhibits *Bacillus cereus* septum formation, which is dependent on divalent cations. *Appl. Environ. Microbiol.* 65:3942–3949.
17. Molins, R. A. 1991. Phosphates in food. CRC Press, Boca Raton, Fla.
18. Molins, R. A., A. A. Kraft, D. G. Olson, and D. K. Hotchkiss. 1984. Recovery of selected bacteria in media containing 0.5% food grade poly- and pyrophosphates. *J. Food Sci.* 49:948–949.
19. Northolt, M. D., J. C. Frisvad, and R. A. Samson. 1995. Occurrence of food-borne fungi and factors for growth, p. 243–285. *In* R. Samson, E. Hoekstra, J. Frisvad, and O. Filtenborg (ed.), Introduction to food-borne fungi, 4th ed. Centraalbureau voor Schimmelcultures, Baarn, The Netherlands.
20. Pitt, J. I., and A. D. Hocking. 1997. Fungi and food spoilage. Blackie Academic and Professional, London.
21. Post, F. J., W. S. Coblentz, T. W. Chou, and D. K. Salumke. 1968. Influence of phosphate compounds on certain fungi and their preservative effects on fresh cherry fruit (*Prunus cerasus*, L.). *Appl. Microbiol.* 16:138–142.
22. Post, F. J., G. B. Krishnamurty, and M. D. Flanagan. 1963. Influence of sodium hexametaphosphate on selected bacteria. *Appl. Microbiol.* 11:430–435.
23. Shelef, L. A., and J. A. Seiter. 1993. Indirect antimicrobials, p. 539–569. *In* P. M. Davidson (ed.), Antimicrobials in foods, 2nd ed. Marcel Dekker, Inc., New York.
24. Sofos, J. N. 1986. Use of phosphates in low-sodium meat products. *Food Technol.* 40:52–68.
25. Sofos, J. N., and F. F. Busta. 1980. Alternatives to the use of nitrite as an antibotulinal agent. *Food Technol.* 34:244–251.
26. Suárez, V. B., J. A. Reinheimer, O. Dorbessan, and M. Rivera. 2003. Actividad antimicrobiana de polifosfatos usados en la industria alimentaria. Presented at II Congreso Argentino de Microbiología de Alimentos. Santa Fe, Argentina, 24 to 26 September 2003.
27. Tompkin, R. B. 1983. Indirect antimicrobial effects in foods: phosphates. *J. Food Saf.* 6:13–27.
28. Zaika, L. L., O. Scullen, and J. Fanelli. 1997. Growth inhibition of *Listeria monocytogenes* by sodium polyphosphate as affected by polyvalent metal ions. *J. Food Sci.* 62:867–869.
29. Zessin, K. G., and L. A. Shelef. 1988. Sensitivity of *Pseudomonas* strains to polyphosphates in media systems. *J. Food Sci.* 53:669.