Acute pulmonary hemorrhage during isoflurane anesthesia in two cats exposed to toxic black mold (*Stachybotrys chartarum*)

Douglas R. Mader, MS, DVM, DABVP; Iwona Yike, PhD; Anne M. Distler, PhD; Dorr G. Dearborn, PhD, MD

Case Description—Acute pulmonary hemorrhage developed during isoflurane anesthesia in 2 Himalayan cats undergoing routine dental cleaning and prophylaxis.

Clinical Findings—The cats were siblings and lived together. In both cats, results of preoperative physical examinations and laboratory testing were unremarkable. Blood pressure and oxygen saturation were within reference ranges throughout the dental procedure. Approximately 15 to 20 minutes after administration of isoflurane was begun, frothy blood was noticed within the endotracheal tube. Blood was suctioned from the endotracheal tube, and the cats were allowed to recover from anesthesia.

Treatment and Outcome—1 cat initially responded to supportive care but developed a second episode of spontaneous pulmonary hemorrhage approximately 30 hours later and died. The other cat responded to supportive care and was discharged after 4 days, but its condition deteriorated, and the cat died 10 days later. Subsequently, it was discovered that the home was severely contaminated with mold as a result of storm damage that had occurred approximately 7 months previously. Retrospective analysis of banked serum from the cats revealed satratoxin G, a biomarker for *Stachybotrys chartarum*, commonly referred to as "toxic black mold."

Clinical Relevance—Findings highlight the potential risk of acute pulmonary hemorrhage in animals living in an environment contaminated with mold following flood damage. (*J Am Vet Med Assoc* 2007;231:731–735)

Two overtly healthy Himalayan cats from a single household were brought to the veterinary hospital for annual dental cleaning and prophylaxis. The cats were siblings and were housed strictly indoors. Cat 1 was a 6-year-old castrated male that weighed 3.6 kg (7.9 lb); cat 2 was a 5-year-old spayed female that weighed 2.9 kg (6.4 lb). The medical history of cat 1 was unremarkable, whereas in cat 2, lymphocytic-plasmacytic colitis had been diagnosed 2 years previously by means of endoscopic biopsy. As a result, both cats were fed a diet of lamb and green peas,[a] and clinical signs in cat 2 had been stable since the diagnosis had been made. Both cats received annual vaccinations and regular veterinary care. During a recent examination, both cats were noticed to have mild to moderate dental tartar with mild gingivitis.

Results of a complete physical examination, including pulmonary auscultation, prior to dental cleaning and prophylaxis were unremarkable, except that cat 2 was noticed to have stenotic nares. Results of preanesthetic laboratory testing were also unremarkable with the exception of high blood calcium concentrations (12.6 and 14.3 mg/dL in cats 1 and 2, respectively; reference range, 7.8 to 11.3 mg/dL).

Both cats had a history of being fractious and difficult to handle. Thus, no preanesthetic medications were administered, and both cats were anesthetized by placing them in an induction chamber and exposing them to 5% isoflurane. In each cat, anesthetic induction was smooth without evidence of struggling. Once the righting reflex was lost, each cat was removed from the induction chamber and intubated with a cuffed, 3.5-mm endotracheal tube. Lidocaine was not used for endotracheal intubation.

Cats were connected to a nonrebreathing anesthetic circuit, and anesthesia was maintained with 2% isoflurane. An IV catheter was placed in the left cephalic vein, and administration of lactated Ringer's solution was started at a maintenance rate. Anesthetic monitoring was performed with a pulse oximeter that used a rectal reflectance probe and with a blood pressure cuff applied to the tail. Cats were allowed to breathe spontaneously, but a sigh was performed manually approximately every 4 to 5 minutes, with maximum airway pressure between 10 and 15 cm H_2O. The cats were moved from left to right lateral recumbency as the procedure progressed.

Dental cleaning in cat 1 proceeded without incident until the procedure was almost complete. While polishing the teeth, the dental technician noticed frothy blood in the endotracheal tube and the procedure was halted. At this time, oxygen saturation measured by means of oximetry was > 95% and blood pressure was within ref-

From the Marathon Veterinary Hospital, 11187 Overseas Hwy, Marathon, FL 33050 (Mader); and the Department of Environmental Health Sciences, School of Medicine, Case Western Reserve University, Cleveland, OH 44106 (Yike, Distler, Dearborn).

Supported in part by a grant from the National Institutes of Health (R21 ES014653; DGD).

The authors thank Dr. Kevin Fitzgerald for assistance in obtaining serum samples from control cats.

Address correspondence to Dr. Mader.

erence limits. Before the procedure could be continued, additional blood was noticed in the endotracheal tube. Therefore, the procedure was terminated, and anesthesia was discontinued. A sterile red rubber catheter attached to a surgical vacuum unit was advanced down the endotracheal tube, and approximately 3 to 4 mL of bloody fluid was collected.

Thoracic radiography was performed while the cat recovered from anesthesia and revealed diffuse pulmonary infiltrates consistent with alveolar hemorrhage in the dorsocaudal lung fields (**Figure 1**). As a result, the cat was given vitamin K (15 mg, SC) and placed in a heated recovery cage; fluid administration was continued at a maintenance rate. The cat recovered from anesthesia without further incident. No more hemorrhage was seen, but the endotracheal tube was found to be covered with coagulated blood when removed. Following extubation, the cat seemed comfortable in the recovery cage and was clinically eupneic.

The events for cat 2 were similar to those for cat 1, with the exception that laser rhinoplasty was performed prior to initiation of dental cleaning. Again, frothy blood was noticed in the endotracheal tube near the completion of the dental cleaning. As with cat 1, the procedure was halted and the endotracheal tube was suctioned. Again, approximately 3 to 4 mL of bloody fluid was aspirated. Thoracic radiographs revealed diffuse alveolar infiltrates similar to those seen in cat 1.

Vitamin K (15 mg, SC) was administered, and cat 2 was placed in a recovery incubator for monitoring. The cat remained dyspneic, demonstrating open-mouth breathing, and supplemental oxygen bubbled through saline solution was administered.

Cat 1 was discharged later the same day. However, after returning home, the owners thought that the cat's breathing was labored and brought the cat back to the hospital. On reexamination, the cat appeared to be resting comfortably but moist rales were noticed bilaterally during auscultation over the dorsal lung fields. As a result, it was decided to hospitalize the cat overnight for monitoring. On the assumption that both cats had a condition similar to exercise-induced pulmonary hemorrhage in horses, for which there is no specific treatment,[1] it was decided to monitor the cats overnight while maintaining them in a quiet, low-stress environment and providing supplemental oxygen as needed.

The following morning (ie, day 2), both cats were resting comfortably. Cat 2 was removed from the oxygen cage and returned to a standard ward for continued monitoring. In cat 1, however, recheck radiography revealed that the infiltrates had either expanded or migrated from the dorsocaudal lung fields to the cranioventral lung fields (**Figure 2**). Later that afternoon, cat 1 became dyspneic and was transferred to an oxygen cage and supportive treatment was initiated. An IV catheter was placed, and administration of 0.45% NaCl with 5% dextrose was started at a maintenance rate. In addition, dexamethasone (0.5 mg/kg [0.23 mg/lb], IV), furosemide (6 mg/kg [2.7 mg/lb], IV), cimetidine (6 mg/kg, slow IV), and aminophylline (4 mg/kg [1.8 mg/lb], SC) were administered. The cat's condition stabilized with these treatments, and the cat appeared clinically to be eupneic, but approximately 7 hours later, the cat developed hemoptysis followed by respiratory and cardiac arrest. Attempts at resuscitation were unsuccessful.

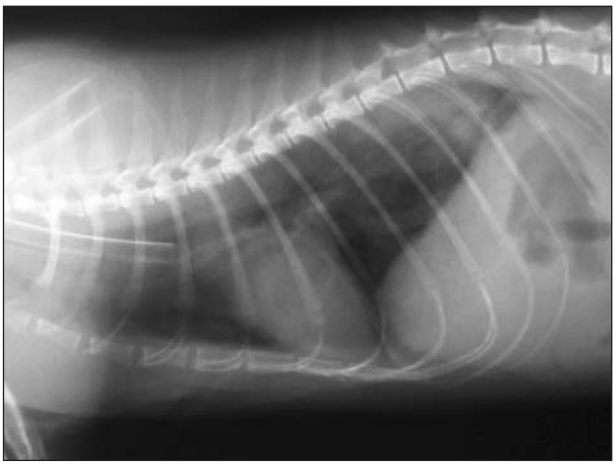

Figure 1—Lateral radiographic view of the thorax of a cat (cat 1) that developed acute pulmonary hemorrhage during isoflurane anesthesia for routine dental cleaning and prophylaxis. Notice the pulmonary infiltrates in the dorsocaudal lung fields.

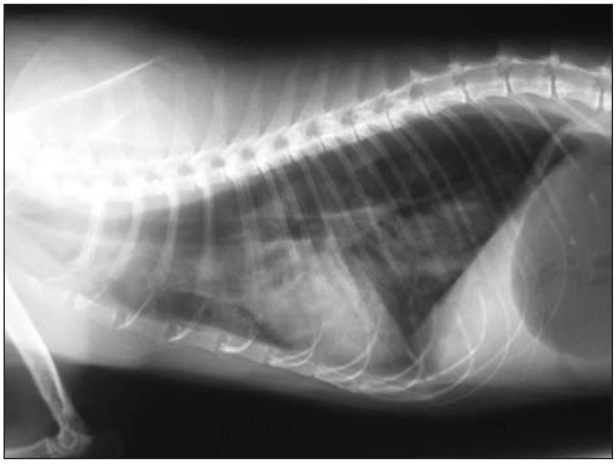

Figure 2—Lateral radiographic view of the thorax of the cat in Figure 1 obtained approximately 24 hours after the episode of pulmonary hemorrhage. Notice the redistribution of the infiltrates to the caudoventral and cranioventral lung fields. Also notice the gas in the stomach, indicative of aerophagia.

The initial treatment for cat 2 was identical to that for cat 1. The PCV was 32% on day 3, compared with a PCV of 36% prior to anesthesia, and the total solids concentration was 6.4 g/dL, compared with a concentration of 6.8 g/dL prior to anesthesia, suggesting that there had been minimal loss of blood into the lungs. In anticipation of hospital discharge, treatment was changed to oral administration of prednisone (5 mg, q 24 h) and theophylline (25 mg, q 24 h), although IV administration of furosemide was continued. Prothrombin time (8.5 seconds; reference range, 6 to 11 seconds) and partial thromboplastin time (15.8 seconds; reference range, 10 to 25 seconds) were within reference limits, as was the platelet count (230,000 platelets/µL; reference range, 200,000 to 500,000 platelets/µL). On day 4, the cat's PCV was again 32%, the cat was taking food when offered by hand, and the cat appeared eupneic. Therefore, the cat was discharged.

Cat 2 was reexamined on day 6. Results of thoracic auscultation were unremarkable, and no infiltrates were seen on thoracic radiographs. On day 9, the owner reported that the cat was doing well and breathing normally but had a finicky appetite. The poor appetite was attributed to treatment with theophylline, and drug administration was discontinued. At this time, the PCV was 34%. A CBC revealed leukocytosis (33,000 WBCs/µL; reference range, 5.5 to 19.5 WBCs/µL) characterized by neutrophilia (21,000 neutrophils/µL; reference range, 2,500 to 12,500 neutrophils/µL), lymphocytosis (9,360 lymphocytes/µL; reference range, 400 to 6,800 lymphocytes/µL), monocytosis (2,080 monocytes/µL; reference range, 150 to 1,700 monocytes/µL), and basophilia (150 basophils/µL; reference range, 0 to 100 basophils/µL). The platelet count was 214,000 platelets/µL. The cat's condition appeared stable, and the cat was not febrile (rectal temperature, 38.4°C [101.2°F]). Therefore, the leukogram was attributed to a combination of steroid administration and resolving inflammation in the lungs. On the basis of these findings, it was decided to begin decreasing the prednisone dosage.

The owner called on day 12 to report that the cat had an improved appetite but had vomited 3 times that morning, with the last vomitus having a tinge of blood. On examination, the cat was lethargic but still eupneic. The owner did not want to hospitalize the cat for IV fluid therapy, so lactated Ringer's solution was administered SC, as well as a dose of cimetidine (15 mg). The cat was discharged, and the owner was advised to administer amoxicillin-clavulanic acid (62.5 mg, PO, q 12 h) and cimetidine (50 mg, PO, q 8 h).

On day 14, the owners took the cat to another hospital for a second opinion. The following day (day 15), the owners chartered a plane to take the cat for a third opinion at yet another hospital. The cat died later that evening, several hours after the flight.

Gross necropsy findings for cat 1 included severe, diffuse pulmonary edema and hemorrhage in both lungs. Edema fluid was evident in the bronchi and bronchioles, and a small amount of blood-tinged fluid was seen in the trachea and nasal passages. There were no other gross abnormalities and no other evidence of hemorrhage anywhere else in the cat. Histologic examination of lung tissue revealed that the pulmonary architecture had been completely effaced by hemorrhage, fibrin deposition, and large numbers of mixed inflammatory cells. Bacterial culture of a lung specimen yielded a pure growth of *Pasteurella* spp.

Necropsy of cat 2 revealed bronchopneumonia; bacterial culture of lung specimen yielded β-hemolytic *Streptococcus* spp and *Klebsiella pneumoniae*. The pathologist indicated that the lung lesions appeared to be acute (ie, of ≤ 3 days' duration) and secondary to aspiration. No other evidence of hemorrhage was seen on the gross examination. Microscopically, lung specimens included multifocal areas where bronchioles and surrounding alveoli were partially or completely filled with fibrin, large numbers of bacteria, and abundant cellular debris.

Serum that had been collected and banked from each cat was sent to the Mary Ann Swetland Center of Environmental Health at the Case Western Reserve University School of Medicine for mold toxin analysis. Samples were analyzed for mycotoxin-serum protein adducts by means of adduct detection.[2] In brief, following exhaustive digestion of serum proteins with pronase, affinity chromatography was performed with polyclonal anti–satratoxin G antibodies that selectively bind macrocyclic trichothecenes and their amino acid and peptide adducts. Adducts were eluted and identified by means of matrix-assisted, laser desorption, time-of-flight mass spectrometry.

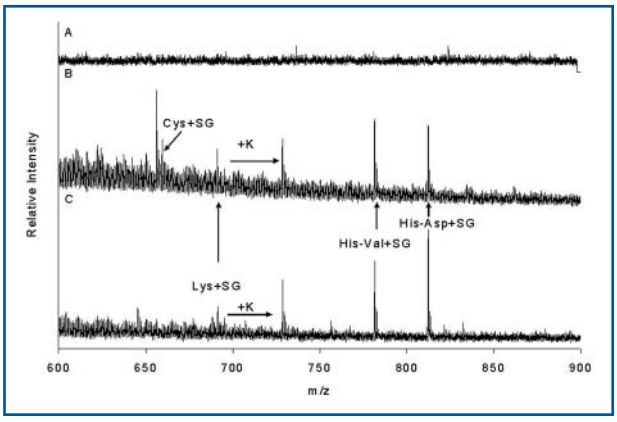

Figure 3—Results of matrix-assisted, laser desorption, time-of-flight mass spectrometry for satratoxin G adducts in pronase digests of serum samples from a healthy control cat (A) and from 2 cats (cat 1 [B] and cat 2 [C]) that developed acute pulmonary hemorrhage during isoflurane anesthesia for routine dental cleaning and prophylaxis. Cys+SG = Cysteine–satratoxin G adduct. Lys+SG = Lysine–satratoxin G adduct. His-Val+SG = Histidyl valine–satratoxin G dipeptide adduct. His-Asp+SG = Histidyl aspartic acid–satratoxin G dipeptide adduct. +K = Lysine–satratoxin G adduct with bound potassium.

Serum samples from both cats were positive for satratoxin G adducts, biomarkers for the toxin produced by *Stachybotrys chartarum,* also known as "toxic black mold." Cysteinyl–satratoxin G and lysyl–satratoxin G adducts were found in the serum of cat 1 (**Figure 3**). Similarly, the adduct of lysine with satratoxin G was detected in the serum of cat 2. Lysine–satratoxin G adducts containing potassium were detected in serum from both cats, along with 2 dipeptidyl–satratoxin G adducts.

To validate findings for the 2 affected cats, serum samples from 6 healthy blood-donor cats living in a mold-free environment in Denver, Colo, were also tested. None of the 6 control cats had satratoxin G adducts in their serum.

Discussion

The 2 cats in the present study had acute pulmonary hemorrhage. To our knowledge, acute pulmonary hemorrhage has not been reported previously in the veterinary literature, with the exception of exercise-induced pulmonary hemorrhage in horses. The exact etiology of exercise-induced pulmonary hemorrhage is unknown, but it is presumed to be caused by hypertension associated with vigorous exercise.[1] In humans, acute pulmonary hemorrhage can be associated with cardiac and vascular malformations, infectious or neoplastic processes, milk protein allergies, immune vasculitis, and trauma.[3] Physicians from 30 states reported

138 cases of unexplained pulmonary hemorrhage in infants from 1993 through 1997,[3,4] with 40 cases occurring to date in the Cleveland area.

A thorough review of the procedures that led up to the hemorrhage episodes in the 2 cats described in the present report failed to reveal any immediate cause for the pulmonary hemorrhage. In both cats, results of preoperative physical examination and laboratory testing were unremarkable, other than slightly high serum calcium concentration; anesthetic induction was associated with minimal stress; endotracheal intubation was performed with sanitized, disinfected tubes; anesthesia was maintained with a nonrebreathing circuit; fluids were administered at a maintenance rate; heart rate, blood pressure, and oxygen saturation were continuously monitored; and dental procedures were performed with sterilized instruments. There were no problems with hemorrhage during catheter placement or collection of blood for preanesthetic testing. To our knowledge, hypercalcemia is not associated with hemorrhage, although hypocalcemia can be associated with hemorrhage in some cases.[5] Importantly, no problems were encountered in a cat that underwent anesthesia and dental prophylaxis immediately before these 2 cats or in a cat that underwent anesthesia and exploratory laparotomy immediately after. The procedure was begun in cat 2 even after complications developed in cat 1 because pulmonary hemorrhage is not known to be a complication associated with isoflurane anesthesia and the cat seemed to be recovering.

In a study[6] involving examination of tracheal wash fluid from cats, hemosiderosis was reportedly identified in 61 of 96 (64%) cats, with findings varying from mild (30%) to marked (10%). Cats in that study, however, had a variety of underlying disease conditions, including heart failure, trauma, infection, foreign body migration, lung lobe torsion, pulmonary embolism or infarction, neoplasia, and bleeding diathesis. In cats, nonspecific hemorrhage has been associated with probable intestinal malabsorption of vitamin K, rodenticide toxicosis, virulent feline calicivirus infection, and the Chediak-Higashi syndrome.[7-10] With the exception of the latter, history and physical examination findings, along with subsequent postmortem findings, ruled out these conditions as possible causes of the pulmonary hemorrhage that developed during anesthesia in the 2 cats in the present report.

In humans, pulmonary hemorrhage has been associated with exposure to the mold *S chartarum*, [3,11-15] and pulmonary hemorrhage during anesthesia has been directly linked with exposure to *S chartarum*.[3] Seven months prior to anesthesia of the 2 cats described in the present report, the region had experienced a devastating hurricane with severe wind damage and flooding. Many houses were destroyed by winds and floods or had severe damage to their roofs, resulting in substantial mold contamination of walls and ceilings. On questioning, owners of the 2 cats described in the present report indicated that their house did have minor flood damage as a result of the storm. Subsequent investigation revealed mold infiltration in their walls resulting from a roof leak that was so substantial as to require replacement of the walls.

Stachybotrys chartarum spores contain several classes of mycotoxins, most notably the trichothecene satratoxins G and H, which are potent protein synthesis inhibitors.[16-18] In animals, these toxins appear to cause capillary fragility, which leads to pulmonary hemorrhage,[12] and capillary fragility was the most likely cause of the pulmonary hemorrhage in the 2 cats described in the present report. *Stachybotrys chartarum* can also produce various phenylospirodrimanes,[19] a cyclosporine,[20] a hemolysin,[21,22] and proteinases.[23,24] In addition to their action as protein synthesis inhibitors, the trichothecenes are immunosuppressive,[25] as are the phenylspirodrimanes and cyclosporine. Together, the trichothecenes and hemolysin produced by *S chartarum* can cause cell injury and death, leading to local destruction of the alveolar capillary wall. Collagen-degrading proteinases produced by *Stachybotrys* spores can possibly also cause acute structural damage to the alveolar capillary wall.[24] As a result, the pulmonary capillaries would be at risk for stress failure whenever pulmonary capillary pressure or capillary transmural pressure was high.[26]

Although cat 1 appeared to recover from the initial pulmonary hemorrhage that occurred during anesthesia, a second episode occurred the next day and the cat died. However, the postmortem examination was not performed until 3 days after cat 1 died. Thus, the *Pasteurella* spp cultured from lung specimens was likely an oral cavity contaminant that had proliferated in the interim.

Cat 2 initially responded to treatment, but its condition deteriorated after the cat was discharged and returned to its contaminated environment. One of the owners reportedly was a smoker, and this may have contributed to the worsening of the cat's condition once it returned home, as tobacco smoke can trigger alveolar hemorrhage.[14] This cat died shortly after being taken by airplane for a second opinion. It is unknown what, if any, role alterations in atmospheric pressure during the flight might have had.

In human infants with acute pulmonary hemorrhage secondary to fungal or smoke exposure, treatment includes removing them from the contaminated environment and administration of corticosteroids.[15] Similarly, in animals, corticosteroids are thought to reduce the inflammation associated with stachybotrytoxicosis.[12,14] Cat 2 received dexamethasone while hospitalized and prednisone after discharge. Thus, even though a definitive diagnosis of *Stachybotrys* toxicosis had not been made, the treatment followed guidelines reported for treatment of humans following presumptive exposure to mold.

Findings for the 2 cats described in the present report highlight a potential risk for animals living in an environment contaminated with mold following flood damage. Importantly, there was no clinical evidence of a problem in these cats prior to the hemorrhagic episodes during anesthesia. Knowledge of possible flood or mold exposure should alert the anesthetist to the possibility of this complication. If hemorrhage occurs, anesthesia should immediately be discontinued and treatment with corticosteroids should be initiated.

a. IVD, Royal Canin, St Charles, Mo.

References

1. Birks EK, Durando MM, McBride S. Exercise-induced pulmonary hemorrhage. *Vet Clin North Am Equine Pract* 2003;19:87–100.
2. Yike I, Distler AM, Ziady AG, et al. Mycotoxin adducts on human serum albumin: biomarkers of exposure to *Stachybotrys chartarum*. *Environ Health Perspect* 2006;114:1221–1226.
3. Tripi PA, Modlin S, Sorenson WG, et al. Acute pulmonary hemorrhage in an infant during induction of general anesthesia. *Paediatr Anesth* 2000;10:92–94.
4. Etzel RA, Dearborn DG. Pulmonary hemorrhage among infants with exposure to toxigenic molds: an update. In: Johanning E, ed. *Bioaerosols, fungi, and mycotoxins: health effect, assessment, prevention and control*. Albany, NY: Boyd Printing Co Inc, 1999;79–83.
5. Rossaint R, Cerny V, Coats TJ, et al. Key issues in advanced bleeding care in trauma. *Shock* 2006;4:322–331.
6. DeHeer HL, McManus P. Frequency and severity of tracheal wash hemosiderosis and association with underlying disease in 96 cats: 2002–2003. *Vet Clin Pathol* 2005;34:17–22.
7. Edwards DF, Russell RG. Probable vitamin K-deficient bleeding in two cats with malabsorption syndrome secondary to lymphocytic-plasmacytic enteritis. *J Vet Intern Med* 1987;1:97–101.
8. DuVall MD, Murphy MJ, Ray AC, et al. Case studies in second-generation anticoagulant rodenticide toxicities in nontarget species. *J Vet Diagn Invest* 1989;1:66–68.
9. Hurley KE, Pesavento PA, Pedersen NC, et al. An outbreak of virulent systemic feline calicivirus disease. *J Am Vet Med Assoc* 2004;224:241–249.
10. Cowles BE, Meyers KM, Wardrop KJ, et al. Prolonged bleeding time of Chediak-Higashi cats corrected by platelet transfusion. *Thromb Haemost* 1992;67:708–712.
11. Tripi P, Thomas S, Dearborn DG. Management of general anesthesia in infants and children with a history of idiopathic pulmonary haemorrhage. *Paediatr Anaesth* 2002;12:243–247.
12. Yike I, Dearborn DG. Pulmonary effects of *Stachybotrys chartarum* in animal studies. In: Strauss D, ed. *Advances in applied microbiology, sick building syndrome*. St Louis: Elsevier, 2004; 241–273.
13. Yike I, Rand TG, Dearborn DG. Acute inflammatory responses to *Stachybotrys chartarum* in the lungs of infant rats: time course and possible mechanisms. *Toxicol Sci* 2005;84:408–417.
14. Dearborn DG, Smith PG, Dahms BB, et al. Clinical profile of thirty infants with acute idiopathic pulmonary hemorrhage in Cleveland. *Pediatrics* 2002;110:627–637.
15. Hart MA, Dearborn DG. Diffuse alveolar hemorrhage (pulmonary hemosiderosis). In: Burg F, Ingelfinger J, Polin R, et al, eds. *Current pediatric therapy*. 18th ed. Philadelphia: WB Saunders Co, 2006;1455–1457.
16. Jarvis BB, Salemme J, Morais A. *Stachybotrys* toxins. *Nat Toxins* 1995;3:10–16.
17. Feinberg B, MacLaughlin CS. Biochemical mechanism of action of trichothecene mycotoxins. In: Beasley VR, ed. *Trichothecene mycotoxicosis: pathophysiologic effects*. Vol 1. Boca Raton, Fla: CRC Press, 1989;27–36.
18. Jarvis BB, Sorenson WB, Hintikka E-L, et al. Studies of toxin production by isolates of *Stachybotrys atra* and *Memnoniella echinata* isolated from homes associated with pulmonary hemosiderosis in infants. *Appl Environ Microbiol* 1998;64:3620–3625.
19. Miyazaki W, Tamaoka H, Shinohara M, et al. A complement inhibitor produced by *Stachybotrys*, sp K-76, a new species of fungi imperfecti. *Microbiol Immunol* 1980;24:1091–1108.
20. Sakamoto K, Tsujii E, Miyauchi M, et al. M FR901459, a novel immunosuppressant isolated from *Stachybotrys chartarum* (*atra*) No. 19392. *J Antibiot (Tokyo)* 1993;46:1788–1798.
21. Vesper SJ, Dearborn DG, Elidemir O, et al. Quantification of siderophore and hemolysis from *Stachybotrys chartarum* strains, including a strain isolated from the lung of a child with pulmonary hemorrhage and hemosiderosis. *Appl Environ Microbiol* 2000;66:2678–2681.
22. Vesper SJ, Magnuson ML, Dearborn DG, et al. Initial characterization of the hemolysin, stachylysin, from *Stachybotrys chartarum*. *Infect Immun* 2001;69:912–916.
23. Kordula T, Banbula A, Macomson J, et al. Isolation and properties of Stachyrase A, a chymotrypsin-like serine proteinase from *Stachybotrys chartarum*. *Infect Immun* 2002;70:419–421.
24. Yike I, Rand T, Dearborn D. The role of fungal proteinases in pathophysiology of *Stachybotrys chartarum*. *Mycopathologica* 2007;164:in press.
25. Pestka JJ, Bondy GS. Mycotoxin-induced immune modulation. In: Dean JH, Luster MI, Munson AE, et al, eds. *Immunotoxicology and immunopharmacology*. New York: Raven Press Ltd, 1994;163–181.
26. West JB, Mathieu-Costello O. Structure, strength, failure, and remodeling of the pulmonary blood-gas barrier. *Annu Rev Physiol* 1999;61:543–572.