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Can composting destroy BSE prions?

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Defining the Fate of Prions During Composting of Specific Risk Material	Completed:	*March 2013*

Researchers:

Tim McAllister, Ph.D. mcallistert@agr.gc.ca
Tim McAllister, Ph.D. and Doug Inglis, Ph.D. (AAFC Lethbridge), Stephanie Czub, Ph.D. (University of Calgary), Kim Stanford, Ph.D. (AARD Lethbridge) Aru Balanchandran, Ph.D. (CFIA) and Brent Sellinger, Ph.D. (University of Lethbridge)

Background:

Bovine spongiform encephalopathy (BSE) is believed to be caused by misfolded prion proteins. On the rare occasion of an infected animal, the prions will be present in particular tissues, known as specified risk materials (SRM) of the animal. Proper disposal of carcasses and SRM is important to control the spread of BSE and maintain Canada's controlled BSE status with the OIE.

The majority of SRM in Canada are rendered and disposed of in CFIA-regulated landfills. The clay liners of these landfills are intended to bind prions and keep them in the landfill, but do not destroy prions. Determining whether composting can cost-effectively destroy prions would be very helpful in areas of Canada where deadstock pickup services or CFIA-approved landfills are not available.

Objectives:

1. Assess whether composting can degrade prions,
2. Establish a bioassay protocol to determine whether composting reduces prion infectivity, and
3. Use protein misfolding cyclic amplification (PMCA) to detect prions after composting

Composting can reduce the very rare chance of BSE prion infection by at least 90%, and therefore a viable method for the controlled disposal of SRM in Canada.

What they did:

Laboratory-scale composting: BSE studies were done in the lab as CFIA requires BSE prion research to be done in very secure facilities. BSE prions were composted with manure and wood shavings. A second set of lab-scale composters also contained feathers. Feathers were added to encourage the growth of microbes that can break down

difficult-to-degrade proteins like the keratin in feathers (and perhaps also prions). After 28 days, specialized lab techniques were used to investigate the extent of prion degradation.

Field-scale composting: To determine whether prions could be degraded under more farm-like conditions, scrapie prions and cattle hooves were composted in a biosecure field-scale facility for up to 230 days. Like feathers, hooves contain keratin which is very difficult to compost, so finding microbes that are attracted to hooves may help identify microbes that can also break down prions. Composted hooves were collected and bacterial and fungal communities that were involved in their breakdown were examined. Composted scrapie prions were used in bioassay studies (below).

Bioassays: Two trials examined whether composted prions can cause disease in lab animals, an approach that is considered the gold standard of assessing prion infectivity. This approach also saves time; mice and hamsters can develop prion diseases in months, compared to years in cattle. In one trial, non-composted prions were bound to membranes and fed to mice. In a second trial, prions from the field-scale composter were inoculated directly into the brains of hamsters.

What They Learned:

Laboratory-scale composting: Prions were spiked into and detected in the manure before composting began. After 14 days, the compost that contained no feathers had fewer prions than at the start, and no prions were found in the compost that contained feathers. After 28 days, no prions were found in either type of compost. The Western Blot test used to detect prions has a limit of detection that showed that 90 to 99% of the prions were destroyed during composting. Re-analyzing these samples with the more sensitive PMCA test indicated that 99.9% of the prions had been destroyed.

Field-scale composting: Microbial community analysis found bacterial biofilms and fungal populations worked together to break down hoof proteins and it seems likely that a similar approach is used to destroy prions during composting. Further study and characterization of these microbes may identify methods of speeding up the rate of prion breakdown in compost.

Bioassays: Surprisingly, feeding membrane-bound non-composted prions did not cause disease in mice, even 400 days after being consumed. Attachment to the membrane may have prevented the prions from being absorbed by the digestive tract. If prions are not absorbed, they cannot reach the brain and will not cause disease. It is well known that it takes far fewer prions to cause disease if they are injected directly into the brain as compared to if they are consumed in feed. The longer prions were in the field-scale composter, the longer it took Syrian hamsters to get sick after being implanted with those prions. Disease onset was delayed in hamsters implanted with prions composted for 14, 56 and 144 days, and hamsters implanted with prions composted for 230 days still had not developed the disease nearly a year after being implanted. This clearly demonstrates that composting reduces the infectivity of prions, and prolonged composting may render them non-infective.

What it Means:

Composting is unlikely to completely destroy all prions because of variability in compost conditions and microbial activity. However, long composting periods can reduce BSE prion infectivity by at least 90%. Adding feathers to the compost may further encourage the growth of microbes that degrade prions.

BSE is rare in Canada, so most SRM does not contain BSE prions to begin with. Considering that feeding prions failed to cause disease in animal bioassays and that composting prions greatly delayed the onset and reduced the occurrence of disease in cranially implanted hamsters, the chances of compost acting as a vector for prions is infinitesimally small. Even if a fraction of prions remained infective after composting, they would be greatly diluted within the compost. Subsequent spreading onto agricultural land would make it highly unlikely that any livestock would ever contact sufficient prions to develop BSE. With these considerations, composting is a viable method for the controlled disposal of SRM in Canada.

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For More Information Contact:
Beef Cattle Research Council
#180, 6815 - 8th St. NE
Calgary, AB T2E 7H7
Tel: (403) 275-8558 Fax: (403) 274-5686
info@beefresearch.ca

For More Information Visit:
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