March 26, 2022

Division of Reclamation, Mining and Safety

1313 Sherman Street

Room 215

Denver, CO 80203

Dear Sirs,

Just this morning I was made aware of yet another sneaky attempt to open a gravel mine less than half a mile from my home. We attended a meeting held at the Milliken Town Hall last August, and attended because people were notified by volunteers who went door to door to publicize it. It was attended by the owners of Loveland Ready-Mix but with the exception of a few postcards, they did not publicize it. There were approximately 50 people at that meeting and numerous other neighbors signed a petition objecting to this facility. There was substantial community resistance displayed at this meeting and some hostility from those attending from Loveland Ready-Mix. The resistance was due, in my observation, to two issues. The first is obvious. Silica poisoning is an ugly thing. The second was the addition of a large number of big trucks, (estimated by the company to be 80 to 100 a week) which will come in on a small county road, 48 ½, be filled up and travel to their facility in Loveland. The estimation is not written in stone and they are not held to that number.

We have a nice community at Mad Russian, with families, growing children, retired people and Veterans with health issues due to their service. All of them will be harmed by this facility (see OSHA article and letter from Dr. Syzek enclosed). There is little question in the short amount of research I have done that silica poisoning affects not just the workers, but those in the surrounding area. This will have an affect on the health of all of us in the area, our landscaping, gardens (see article from Utah re cherry orchards) pets, and our general quality of life. I wrote to a physician in Salida, CO who fought a similar attempt made in their area. While it was ultimately turned down for the size of the roads, Dr. Syzek shared the research he had done with information on the affects of silica poisoning on a community. He was adamant that there would be significant health problems for those living nearby. The owners of Loveland Ready-Mix do not live in this community, so their mine will enrich them at our expense.

The notification they promised us at the community meeting never happened. Not one attempt was made by them to contact the community that would be most affected. They did have contact information, but never used it. They seemed to feel that posting signs on county road 48 ½ was enough, even though only those who live directly across the road would likely see them. There is no access to Mad Russian Subdivision from that road. They posted a notice in the Greeley Tribune. We do not live in Greeley. It would appear obvious that they did as much as they possibly could to keep this information about applying for permits to begin work as secretive as possible. They also suggested at the community meeting that there would be "only" 80 to 100 trucks each week and that they would not use the already overcrowded Highway 257 to deliver the product to their facility. This is hogwash. We did hear from more than one actual truck driver who stated that truck drivers choose the shortest route

between point A and point B, and that is likely not a narrow dirt road. Highway 257 is already currently overwhelmed by huge oil trucks and other trucks hauling heavy equipment, and this is obvious to us 24 hours a day, 7 days a week. They noted that they would leave the property in lovely condition....25 years from now, with nice ponds and vegetation. You will excuse me for not being excited, but I will be 75 this year and it would likely be nice for someone else, but not for me. I am sure too that the children who would likely suffer long term health consequences from the silica (an elementary school is approximately ¾ of a mile away) and their suggestion that they will water all this dust down in dry Colorado is subject to question. There is a golf course immediately to the north of this proposed facility. A sure win for that golf course. The only people who stand to profit are the owners of Loveland Ready Mix. The rest of us will receive only negative affects; health issues, the loss of vegetation due to dust coating the foliage, the loss of property values, and the affects on the wildlife, some of it either endangered or threatened.

I sincerely hope that you will look at the aggregate negative affects on the people living in this area, and see that the value of our lives, homes and simple quality of life has more value than a gravel pit and that we deserve protection from a company who went a long way towards covering up this mine, and evading any good neighbor policies. I have enclosed printouts of the letter from Dr. Syzek, the report on the affects of Silica poisoning from OSHA, and a newspaper article from Utah about the affects on a 200 year-old cherry orchard caused by a gravel mine in close proximity. Another Mad Russian advocate has passed on the names of those who signed in opposition to this gravel pit, and additional information about the adverse health effects. Weld County has a tendency to approve anything a property owner decides to do with their property, no matter the effects on others in the area. I cannot enclose the dismay and anger of the people in Mad Russian Subdivision who have been lied to and blindsided by this company, but I hope you can try to put yourselves in our place. I was born and raised in Colorado and remember a time when the lives and well being of the citizens were more valued than money. I sincerely hope that is still true.

Sincerely,

Sheila P Grotzky

Kenneth L. Grotzky

115 Birdie Dr, Milliken, CO 80543 970-587-6408

March 26, 2022

Division of Reclamation, Mining and Safety
1313 Sherman Street
Room 215
Denver, CO 80203

Dear Sir or Madam,

I am writing to strongly urge you to not approve the proposed Dunn Pit Mine (DPM) on the edge of the town of Milliken. Placing a gravel mine only a few hundred feet from a neighborhood will likely have huge health consequences to the residents. Property values and tax revenues will be adversely affected. The significant added congestion of already crowded local roads will only add to air pollution already made worse by the dust from the mine and damage the roads. Loveland Ready-Mix Concrete(LRM) has not been an honest actor in its dealings with the surrounding towns and residents.

The location of this proposed mine is only a few hundred feet west from residential dwellings in the Mad Russian neighborhood which is only separated by a golf course. As has been well studied gravel mines produce crystalline silica (CS), which is a known carcinogen. CS enters the lungs and cannot be expelled by the body. Health effects of CS can range from Silicosis, lung cancer, tuberculosis and increased lung irritation. With dust storms already a common occurrence in Mad Russian, being down wind form the proposed LRM mine would obviously be a health hazard for everyone living in the neighborhood and likely for the entire town of Milliken of which the Mad Russian lies on the northern tip of the town. As much as the wind blows out of the west, there is no realistic amount of water that would make the dust levels safe at all times for residents near by.

From the figures that we received from LRM at a meeting on the matter several months back, there will be 64 truckloads a day, three times every week. On just the initial sight, over 10 years, this would equate to almost 100,000 truckloads, adding additional pollution to the air and destroying the local roads. Diminished property values will lead to lower tax revenues for local governments to pay for paving and repairing the roads.

As mentioned in the previous paragraph, property values are likely to be adversely affected by the existence of DPM. Open pit mines do not tend to be positive aspects of scenery such as a golf course or views of the mountains would be, and with their existence do tend to lower the value of nearby real estate. Toxic clouds of dust loaded with CS being dispersed over an entire town would not help real estate values either if everyone if getting sick. If real estate values go down so does tax revenue to local governments that have to pay for the roads.

OVER →

Of great concern to my neighbors and myself is the total lack of transparency that has been shown by LRM. We were told in the September meeting that they would notify us of the permitting process and let us know when we could submit comment. To my knowledge neither my neighbors nor myself have gotten any notification of anything and the decision date is only a few days away from the date of this letter. It seems as this is dishonest and it is an effort by LRM to ram this through. As secretive as LRM has been it raises many red flags.

With all of the adverse health and financial concerns the only logical choice is to not permit the DPM. With the high price of real estate now it would seem that it would be in the best interest of all parties involved for LRM to sell the land and move away from town were they could still mine and not affect the health and lives of Milliken's residents. Thank you for your time

Sincerely,
Morgan Grotzky
115 Birdie Dr.
Milliken,CO

6/15/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. One of the more alarming aspects is from the American Lung Association. It is titled Health effects for the population living near a cement plant: **Understanding Silicosis**. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

Kenneth W Helgeson — Printed

Kenneth W Helgeson — Signature

Beverly K. Helgeson

Over

6/15/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. One of the more alarming aspects is from the American Lung Association. It is titled Health effects for the population living near a cement plant: **Understanding Silicosis**. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

Michael Sage

Printed Signature

6/15/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. One of the more alarming aspects is from the American Lung Association. It is titled Health effects for the population living near a cement plant: **Understanding Silicosis**. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

Brandon Heinel

Printed

Signature

1977 BIRDIE DR.

6/15/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. One of the more alarming aspects is from the American Lung Association. It is titled Health effects for the population living near a cement plant: **Understanding Silicosis**. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

Michael S Buckley — 1997 BIRDIE DR
Printed — Signature

DANIELLE 720 425 9797

6/15/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. One of the more alarming aspects is from the American Lung Association. It is titled Health effects for the population living near a cement plant: **Understanding Silicosis**. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

TAMI L MOSER — Printed

Tami L Moser — Signature

1967 BOBBY

6/15/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. One of the more alarming aspects is from the American Lung Association. It is titled Health effects for the population living near a cement plant: **Understanding Silicosis**. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

Gerry Kerry — Printed

Signature

2149 Country Club Parkway

6/15/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. One of the more alarming aspects is from the American Lung Association. It is titled Health effects for the population living near a cement plant: **Understanding Silicosis**. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

John Gibbs

Printed

Signature

2322 BIRDIE DR

6/15/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. One of the more alarming aspects is from the American Lung Association. It is titled Health effects for the population living near a cement plant: **Understanding Silicosis**. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

RANDY TEACUCK — Printed

Signature

2010 BIRDIE DR

6/15/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. One of the more alarming aspects is from the American Lung Association. It is titled Health effects for the population living near a cement plant: **Understanding Silicosis**. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

Stephanie Reed — Printed

Stephanie Reed — Signature

2472 B. Drive

6/15/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. One of the more alarming aspects is from the American Lung Association. It is titled Health effects for the population living near a cement plant: **Understanding Silicosis**. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

Jeffrey A. Pigue — Jeffrey A. Pigue

Printed — Signature

2202 Birdie Dr
Milliken Co 80543

6/15/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. One of the more alarming aspects is from the American Lung Association. It is titled Health effects for the population living near a cement plant: **Understanding Silicosis**. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

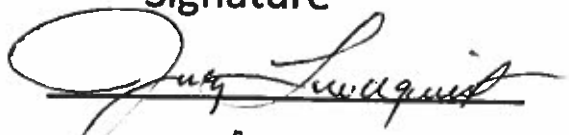
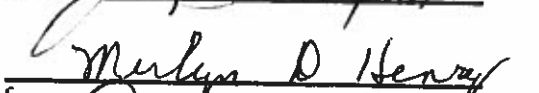
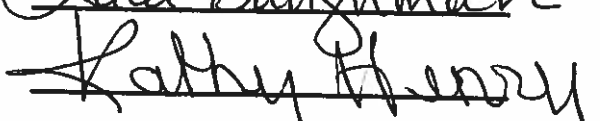
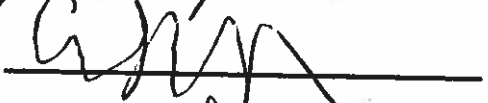
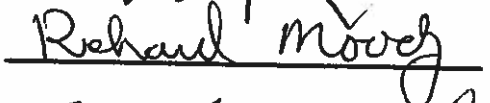
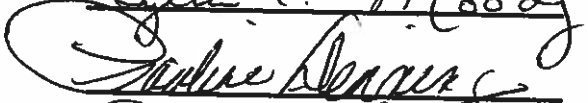
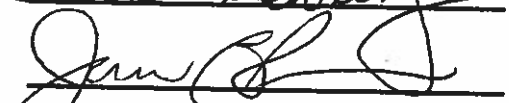
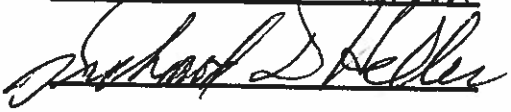
Peter S. Keohane

Printed

Signature

114 B. Drive

PRINTED	SIGNATURES
Rhonda J. Smith	Rhonda J. Smith
Brian E Smith	Brian E Smith
Casey Miller	Casey Miller
Jennifer Miller	
Kathleen Longacre	Kathleen Longacre
JOANNE MILLER	Joanne Miller
LARRY D. MOOMEY	

Name Printed	Signature
Judy Lundquist	
Merlyn Henry	
Lacee Baughman	
Kathy Henry	
Sheri Dalgliesh	
Rich Dalgliesh	
Tyler Roach	
Tarran Roach	
Carrie Yahn	
Richard Moody	
Sylvia Moody	
Pauline Benning	
Jerri Burnham	
Richard D Heller	

6/15/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. One of the more alarming aspects is from the American Lung Association. It is titled Health effects for the population living near a cement plant: **Understanding Silicosis**. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

Nancy Fox-Clement Nancy Fox-Clement

Printed Signature

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

(1)	(2)
Ed Kippes	Willa Kippes
Ed Kippes	Willa Kippes
Printed	Signature

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

Joan H. Eaton

Printed

Signature

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

Printed	Signature
Cindy Vasco	Cindy Vasco
John Vasco	J Vasco
Isabel Ramirez Jr	Isabel Ramirez Jr
Mary Kay Steahly	K Steahly
Lance L. Steahly	Lance L Steahly
Jessica Johnson	Jessica Johnson
Jennifer E Bailey	Jennifer E Bailey
Patton Lundquist	P Lundquist
Liz Lundquist	Liz Lundquist
Peggy Mayeda	Peggy J Mayeda
Mike Mayeda	Mike Mayeda
Gary W Sauder	Gary Sauder
Jordan Perea	Jordan Perea

Print	Sign
Kathi Ramirez	Kathi Ramirez
Steve Kelly	Steve Kelly
Chris Kelly	Chris Kelly
Candy Harmon	Candy Harmon
Steve Harmon	Steve Harmon
Jeremy Boughman	Jeremy Boughman
Jayton Boughman	Jayton Boughman

6/15/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. One of the more alarming aspects is from the American Lung Association. It is titled Health effects for the population living near a cement plant: **Understanding Silicosis**. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The signatures below represent the names of homeowners:

Teresa Miller — Teresa Miller

Printed — Signature

7/4/21

Loveland Ready Mix

Loveland, CO

Dear Stephanie,

This document is to serve as notification that we, the landowners of the Mad Russian Subdivision, are banding together to stop any further development of your Milliken gravel pit proposal.

While we deeply appreciate your visit to our neighborhood and your openness about your plans, we will put together our resources to stop the plan.

We have done some research about gravel pits. No matter the amount of water you use to keep the loose dirt under control, the first part of your dig will be done with no ability to stop the particles from travelling to our home residences. Also, any stored dirt or topsoil will be prone to travel due to the wind.

From the figures we received from you, we would experience:
64 truckloads per day, 3 days per week, for 52 weeks a year, for ten years.

64 x 3 x 52 x 10 = 99,840 truckloads.

Multiply that by approximately the same amount on the south side of the Big Thompson River:

99,840 x 2 = 199,680 truckloads.

There is no way that this will create any benefit to the landowners of the Mad Russian subdivision.

The attached signatures represent the names of homeowners:

KRISSTINE VEIT-WILLIAMS — Krisstine Veit Williams

Printed — Signature

Cathy Palmer — Cathy Palmer

Printed — Signature

8

Printed	Signature
Lyle Schrock	
Printed	Signature
MARK JOYCE	
Printed	Signature
JOSEPH BURGER	
Printed	Signature
Dan Johnson	
Printed	Signature
Jeff Kammerer	
Printed	Signature
DAVID CHAPMAN	
Printed	Signature
Susan Paquette	
Printed	Signature
Michelle Powell	
Printed	Signature
MAX MORGAN	
Printed	Signature
Sharon M. Morgan	
Printed	Signature
Danielle McNeil	
Printed	Signature
SHELTON REGISTER	
Printed	Signature

Printed	Signature
REBECCA WALLIS WEBSTER	Rebecca Wallis
Beth Wishall	Beth Wishall
Mike Wishall	Mike Wishall
Sandra Strait	Sandra Strait
Jerry Strait	Jerry Strait
Jeanne Theede	Jeanne Theede
Ronald Theede	Ronald E Theede
Loni Schlothauer	Loni Schlothauer
SAM SCHLOTHAUER	Sam Schlothauer
Carol R. McIntosh	Carol R. McIntosh
Jon McIntosh	Jon McIntosh
Linda Becker	Linda Becker
Jodi Hagan	Jodi D. Hagan
Mary Wojtkiewicz	Mary Wojtkiewicz

Printed	Signature
JANIE ECHOLS	Janie D Echols
ROY ECHOLS	Roy Echols
Tiffany Gagnon	Tiffany Gagnon
ROBERT K ELLAMS	Robert Kyellams
Bernie Perry	Bernie Perry
GARY PIMPLE	Gary Pimple
Carol Knowski	Carol M Knowski
FRANK J KNOWLSKI	Frank J. Knowski
Hannah Tippett	Hannah Tippett
Linda Wallace	Linda Wallace
LINDA GARVER	Linda Garver
Steve GARVER	Steve Garver
Kathie Records	Kathie Records
Harper Willis-Webster	Harper Willis-Webster

Printed	Signature
KEN MILLER	Ken Miller
Tammy Riegsecker	Tammy Riegsecker
Kellie Matthews	Kellie Matthews
Crystal Kelly	Crystal Kelley
Dallas Williams	
J.R. Schnelzer	
Lisa Schnelzer	Lisa Schnelzer
JoAnne Lawson	JoAnne Lawson
DAVID LAWSON	
ROBERT JOHNSON	
Virginia Mares	Virginia Mares
Sheila Grotzky	Sheila Grotzky
Kenneth Grotzky	Kenneth Grotzky

Printed	Signature
Ted Engelbert	Ted Engelbert
Reed Wagner	Reed Wagner
Morgan Gretzky	Morgan Gretzky
Michael A. Coronel	Michael A. Coronel
Patricia D. Coronel	Patricia D. Coronel
Doug Kehr	Doug Kehr
Corinne Kehr	Corinne R. Kehr
Naomi Warner	Naomi Warner
Sheri Woodson	Sheri Woodson
David Woodson	David Woodson
Don Holtz	Don Holtz
Kathy Sabella	Kathy Sabella
Paula Miller Barrientos	Paula M Barrientos

Printed	Signature
Breanna McNeil	
Racheal Edmonds	
Mark Everett	
Billie Sue Wallis	
Scott Wallace	

Neighbors on county road 48 ½ :

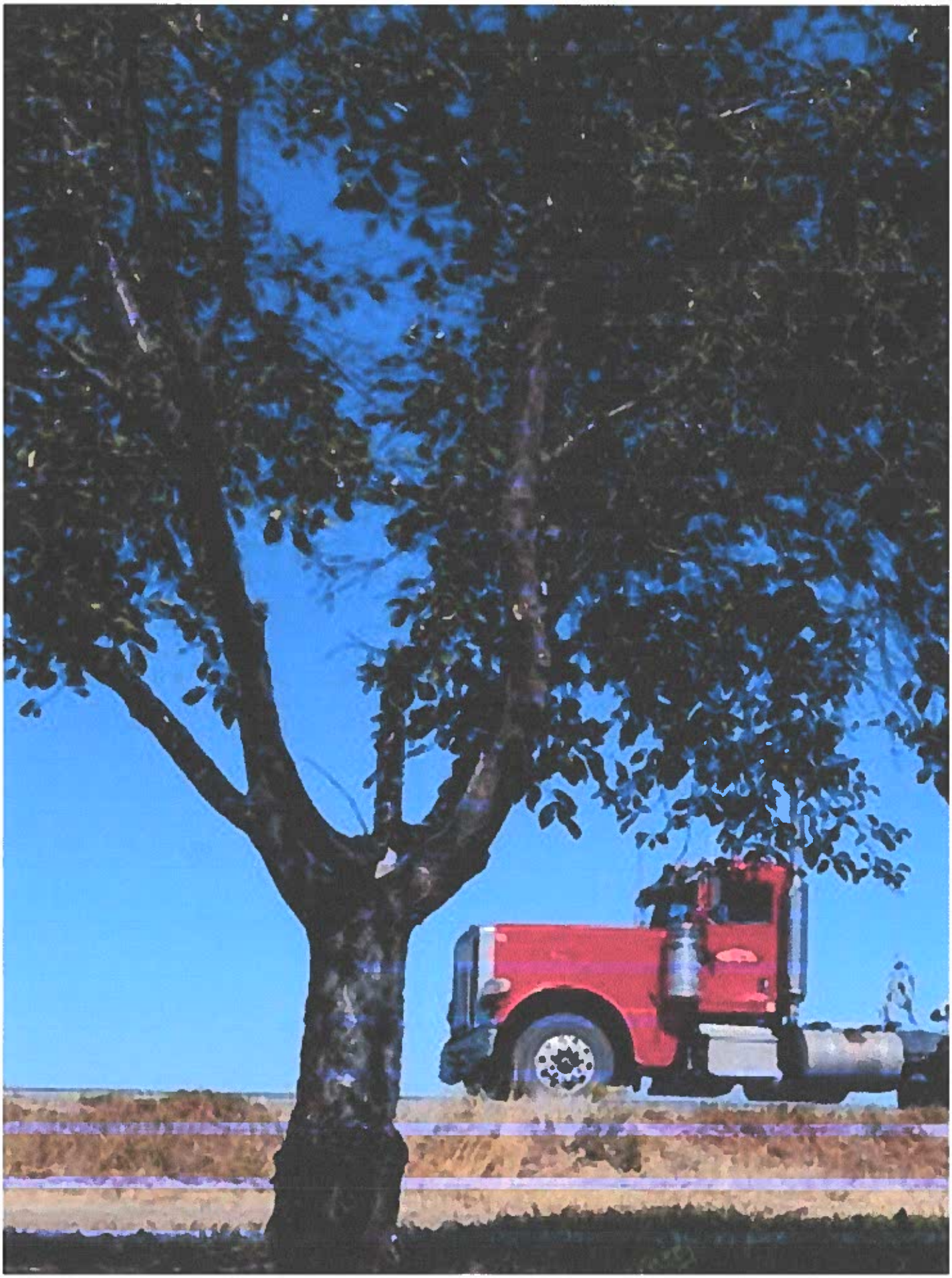
Printed	Signature
Gilbert Spaur	Gilbert Spaur
Tom PRICE	Tom Price
VIRGIL McCLURE	Virgil McClure
Tammy McClure	Tammy McClure
Duncan Todd	

Click to copy
Related topics

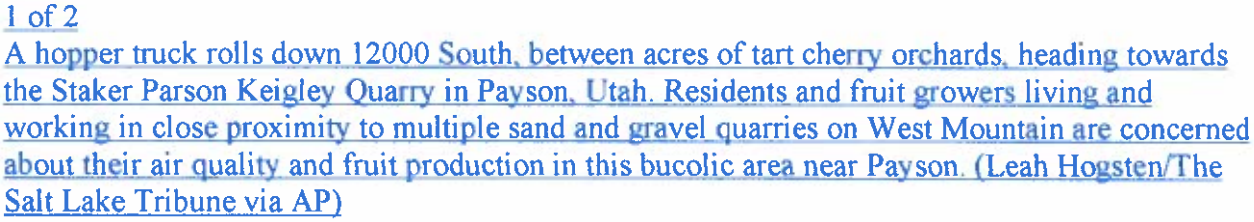
- Environment
- Mountains
- Utah
- Trees

Cherry farmers pitted against gravel quarries over dust

By Brian MafflyOctober 11, 2020



1 of 2



A hopper truck rolls down 12000 South, between acres of tart cherry orchards, heading towards the Staker Parson Keigley Quarry in Payson, Utah. Residents and fruit growers living and working in close proximity to multiple sand and gravel quarries on West Mountain are concerned about their air quality and fruit production in this bucolic area near Payson. (Leah Hogsten/The Salt Lake Tribune via AP)

PAYSON, Utah (AP) — Ryan Rowley's orchards occupy a special piece of Utah where elevation, soils and topography collude to create an ideal environment for growing fruit, especially the tart cherries that have long been associated with the family name.

But trouble lurks over this high-desert Eden, which also supplies Utah builders with a mineral bounty needed for construction.

"You think you would be on a nice country road, but there are trucks and nonstop traffic," Rowley says over the roar of passing gravel haulers on a recent morning as he inspects his orchards near busy quarries on the southern tip of West Mountain. "You can see the dust on the leaves. The trees look sickly. It cuts down on photosynthesis, the absorption of sun the trees are able to capture."

Rowley rubs a thumb across leaves on a recently harvested cherry tree to highlight the damage from mites that proliferate in dusty conditions. Some trees have already shed half their leaves — and it's barely October.

"The leaves are falling a month early. When it does rain, it won't capture as much moisture," he says as he shakes the branch, releasing a cloud of dust as if he were airing out a filthy carpet. "We were wearing masks way before it was cool."

The dust is a testament to a mounting conflict in this still-bucolic corner of ever-sprawling Utah County where sand and gravel quarries extract aggregates to feed a boom in real estate development and road building, pitting important Utah industries against one another. While the farmers complain the state is favoring the construction industry, quarry operators say they are heavily regulated by multiple agencies and their impacts are "minimal."

"We have limited access to these materials to support the growing economy of Utah, so there is a lot of debate over how do we go forward. Those issues are in so many places in the state. A lot of it is driven by demand," Stewart Lamb, director of business development with the Kilgore Cos., said a recent hearing over an expansion of its Benjamin quarry. "If there was no growth in Utah County, there wouldn't be seven or eight mines on West Mountain. How we go forward as a state? There is some really difficult discussions going on and some sincere attempts to make it so we can be sustainable as an industry."

A few miles north of Rowley's orchards, Benjamin residents are fighting Kilgore's expansion, arguing increased truck traffic and fugitive dust emissions are disrupting their lives and threatening their health.

"There is nothing that addresses cumulative effects. When is it too many mines?" asks Benjamin resident Julie Sainsbury. She alleges the pit's dust emissions are unacceptable, made worse by trucks that trail dust even when they are empty.

Seven quarries, covering about 600 acres, operate on West Mountain. Rowley's orchards are near the entrances to the Keigley Quarry operated by Staker Parson and another called the Cherry Pit.

That the latter quarry's name riffs on the Rowley clan's singular product is a source of annoyance for Ryan Rowley, who fears the fugitive dust from the pits and passing trucks could doom cherry growing there.

"There's some mornings that you can see dust coming off this hillside and filling this valley below us," Rowley says. "Sometimes, as I'm out changing water before the sun comes up, there's lines of trucks waiting to get up into that pit. They're overloaded. Their fender flares are covered in gravel. That's (what leads to) all the broken windshields we get. They're covered in dirt. They're not covering their loads, not washing off their trucks. And they string dirt clear down these roads. It turns into dust, and then it's going up in the air, landing on our trees."

While fruit growers and residents acknowledge Utah needs these materials, they complain that the gravel operations are unnecessarily jeopardizing an important agricultural industry and their peaceful quality of life. They contend the quarries and their haulers refuse to take steps that would reduce their impacts and dust levels are inadequately monitored.

The Salt Lake Tribune invited comment from officials at Staker Parson and Kilgore, the operators of the two major pits highlighted in this story, but neither supplied a response.

West Mountain quarries produce sand, gravel, crushed stone and other low-value materials used in construction and the making of concrete and asphalt. Some of this material is processed on-site, resulting in additional industrial emissions.

Across the state, nearly 300 quarries produce between 40 million and 50 million tons of aggregates a year, according to the Utah Geological Survey. The haul in 2018 was worth $286 million, or about $7 a ton.

Because such vast amounts are needed in construction jobs, it is necessary to source sand and gravel close to where it is used to keep down costs, according to Andrew Rupke, an industrial minerals geologist with the survey. In many cases, that means siting quarries near where people live and farm.

"Economically, you can't ship it as far," Rupke says. "They generally have to be closer to where they are used. This is happening in urban areas everywhere. As urbanization occurs, those aggregate operations move farther afield, but they cost more. There is a real tension over how we do this and do it right."

Still, West Mountain growers argue, aggregate producers can tap suitable sand and gravel deposits elsewhere; good luck, they say, finding other places in Utah where tart cherries can be grown economically.

"They say they can't go out anywhere else to get it. To that I say BS. They want to go only three minutes off the freeway. Well, I cannot grow fruit in Nephi (about 25 miles to the south). It's too cold," says Genola fruit grower Cheryl Fowers. "You can do what you want on your property, but if you do something that harms your neighbor, you had better knock it off. It's in their own handbook. We have big issues and they want to keep expanding and expanding."

Aggregates come in two major forms: Sand and gravel are extracted from unconsolidated deposits that are screened and sorted into various grain sizes. Crushed stone is blasted and pulverized from bedrock, usually dolomite and limestone. West Mountain yields both types.

The tensions at West Mountain are hardly unique. Up and down the Wasatch Front, most visibly at the Geneva Rock quarry at Point of the Mountain, gravel operations are stirring up oppositions from neighbors concerned about dust, noise and truck traffic.

State law limits the opacity of quarries' dust plumes to 20%, or 10% off-site, but this standard is difficult to enforce and some critics call it "absurd."

"It's about as unscientific as anything you could apply to this situation," says Brian Moench, president of Utah Physicians for a Healthy Environment. "The whole thing is slanted toward allowing these operators to do whatever they want."

To reduce dust emissions, his group urges operators to use natural gas-powered trucks instead of diesel, to cover their loads, to clean trucks between trips, and to relocate mines to less windy and populous sites. Sand and gravel deposits can be found along 2,000 miles of Lake Bonneville's ancient shorelines rimming the valleys of northern Utah, says John Macfarlane, a neurosurgeon on Moench's board.

"If you go over a valley or two to the west, you can (mine) with much less effect because there is no local population," Macfarlane says. "If they go a little farther away, they can still make their money, just not as much."

The physicians group insists the Utah Department of Environmental Quality should do more to track dust emissions, conduct more frequent quarry inspections, and establish a hotline for residents to report dust incidents.

In addition to visual impacts, the crystalline silica released when rock is pulverized poses a serious threat to human health when inhaled. Macfarlane says these sharply fractured particles can damage lung tissue and cause cancer.

All the quarries are required to submit a fugitive dust mitigation plan with DEQ, which conducts annual inspections and responds to complaints, according to agency spokesman Jared Mendenhall.

Benjamin's plight

Most of West Mountain's quarries are clustered on its southern tip near Payson. On the mountain's east slope to the north, Kilgore Cos. operates the newer Benjamin Pit, now steeped in controversy over its approved expansion from 44 to 97 acres.

Kilgore opened the pit in 2011 to secure materials for reconstructing Interstate 15 through Utah County. Residents were led to believe the pit and its related asphalt plant were temporary, according to Benjamin resident Debi Brozovich, who helped start a grassroots group with Sainsbury called South Utah County Community Voice.

In 2016, Kilgore sought and eventually won approval from the Utah Division of Oil, Gas and Mining and the federal Bureau of Land Management to expand onto adjacent public land. Under pressure from area growers, the Utah County Commission then changed the zoning to confine mines to their existing footprints, but that ordinance does not apply to federal land, which predominates West Mountain.

The grassroots group contested the Kilgore expansion before the Board of Oil, Gas and Mining, but that industry-centric panel rejected the challenge on procedural grounds last month, enabling Kilgore to move forward.

"We have become saturated with mining activity, and it is negatively impacting our community," Sainsbury said at a recent hearing refereed by DOGM Director John Baza. She showed pictures of the quarry releasing dense dust clouds in apparent violation of opacity standards.

"Having a dust-mitigation plan and implementing a plan are two different matters," she said. "There is inadequate dust suppression going on."

While conceding they could do more to monitor emissions, Kilgore officials said the quarry is meeting DEQ's environmental safeguards.

"We follow and are in compliance with the permits we have," Kilgore's lawyer Graden Jackson said at the hearing. "We pledge to you we will continue to be in compliance. We are a large operation. We have no desire to be out of compliance. We live in these communities we serve."

Those assertions mean little to growers and residents who say dust is coating their orchards and neighborhoods and that haulers operate unsafely without consequence.

Gravel quarries could take steps to better control and monitor dust emissions, but operators opt not to, and no agencies are requiring such measures, says Kylara Papenfuss, a field adviser for a fruit-growing cooperative.

"They can treat the loads to keep the dust down. They can put up sensors," she says. "We have examples where they automatically report dust levels and if they are not within the permit, they can get fined. "

While many in Benjamin are unhappy with the mining, some residents of nearby cities support the Kilgore expansion.

"As Utah County continues to grow, having ready access to building materials is crucial to keeping costs manageable," Woodland Hills resident Debra Dimmick writes in an email to DOGM. "The pit provides good jobs for residents of the county and has been a good neighbor to the surrounding area."

West Mountain is not only a rich and convenient source of aggregates for Wasatch Front builders; it is also the geographic feature, rising above Utah Lake's southeast shore, that creates a microclimate ideal for fruit production, resulting in a bounty of apples, peaches and, most famously, tart Montmorency cherries, marketed as "Monties." Utah is the nation's second-largest producer of tart cherries after Michigan, and most of this harvest comes from Payson Fruit Growers, an eight-farm cherry-processing co-op to which Rowley and many of his cousins, uncles and aunts belong.

These growers account for about 40 million pounds of cherries, most of which are dried, put into jams or juiced, then sold around the nation.

"We're in a sloping valley, which protects us from frost. As the winds blow, it moves that cold air out of here. And you can't just pick any spot down in the valley and plop a cherry tree because they like to be warm. They can't be froze out," Rowley says. "These trees do like to go dormant. And so we need that snowpack in the winter. They need a break. So tart cherry trees love hot summers and cold winters, but not too cold."

His great-grandparents were fruit farmers who came from England and established orchards in Utah. Don Rowley, Ryan's grandfather, and his brothers grew fruit in Orem until the 1950s, when the arrival of the Geneva Steel plant displaced orchards with subdivisions.

So the Rowley growers established new orchards in Payson, where they discovered ideal fruit-growing conditions under West Mountain. Today, several of these growers' children and grandchildren are still in the fruit business here and hope to remain.

"This is more valuable to me than a stack of town homes and cookie-cutter houses," says Ryan Rowley, gazing over the orchards fanning out from the mountain. "This is a livelihood. This is food on the table. This is the opportunity for us to give back to the community as well as to hire kids and schoolteachers during the summer to help us harvest."

The orchards' survival is under pressure by foreign competition, food-safety regulations, drought, and now gravel quarries and dust emissions pushing up costs and reducing yields.

"It's just a madhouse around here.I just want to see them have some regulation and follow (the rules) that keep everybody safe," Rowley says. "I understand that we need the dirt and the rocks and the gravel to grow, but as we lose this green space around here, we're never going to get it back. And the quality of life around here is going to go down."

Ryan's 160-acre operation, called Rowley's Fruit Farms, yields about 1 million pounds a year and supports three families.

"This has been the most blessed way to live ever, to work the land, be out here. I love my office," he says. "I want to be able to pay that forward to my kids and their future generations. Utah County needs to decide whether or not this industry is important to them. I feel like we've been overlooked. We haven't been heard."

ADVERTISEMENT

EDUCATION/HEALTH /
OPINION/LETTERS

LETTER: Physician Warns of Adverse Health Effects of Proposed Gravel Pit

BY SPECIAL TO THE POST · APRIL 10, 2017

Editor:

As a physician and resident living near the proposed ACA gravel pit at 14110 CR 140, I am writing to express concern for the health effects that it will have on Chaffee County residents who will breathe the dust and diesel exhaust from this operation.

Dust from surface mining operations produces airborne pollution including crystalline silica that can cause lung cancer, silicosis, COPD, kidney and autoimmune diseases; increase susceptibility to infections like TB; and increase hospitalizations for heart disease. The dust from gravel mining may also contain toxins such as heavy metals and radon, both of which cause cancer.

Dust and toxins can travel hundreds of miles, but the proximity to existing and future Chaffee County residents make the proposed gravel pit site an unacceptable health hazard. Fugitive particulate emissions, air pollutant emissions, and visible emissions will be produced, and even if these emissions are within the allowable limits of state regulations, the potential health effects will not be eliminated.

Dust landing on the property and homes of nearby residents will be stirred up during daily activities, thus magnifying the health consequences particularly for children and babies in utero. Because of greater physical activity, higher metabolic rates, and hand-to-mouth actions, young children will be more exposed than adults via both inhalation and ingestion. Exposure of pregnant women living nearby will extend the health consequences to more than one generation because of the damage that increased

pollution and diesel exhaust can do to chromosomes and fetal development. Toxic dust generated by pit operations would continue for years, but the health consequences can last much longer.

Diesel emissions from trucks and equipment will add to the health hazard. Diesel exhaust is a carcinogen and is more toxic than gasoline vehicle exhaust. Long term exposure to even low levels of diesel exhaust raises the risk of dying from lung cancer about 50% for residents who live near industrial operations, and about 300% for the workers.

Based on the scientific evidence and proven health effects of pollutants produced by gravel pit mining, I consider this proposal to be entirely incompatible with what should be Chaffee County officials' first priority – protection of public health, families, children, and pregnant mothers living near this proposed pit and in nearby Poncha Springs and Salida.

I appeal to all area residents to attend the upcoming Planning Commission public hearing on April 25th at 6 pm and the Board of Commissioners public hearing to be held May 9th at 9 am, both scheduled at the County Building in Salida, and urge our county leaders to reject the proposal. The fate of this proposed gravel pit may be decided at those meetings.

For more information or to sign a petition opposing this gravel pit, please send an email to DontCrushSalida@gmail.com.

Thomas E. Syzek, MD, FACEP

Salida, Colorado

Special to the Post

The Salida Daily Post welcomes submissions from the community. Send Press Releases, Letters, News Tips to: editor@salidadailypost.com or call 719-581-9812 for more information.

CATEGORIES

Select Category

PAST ARTICLES

Select Month

COOL LINKS

- PagosaDailyPost
- Salida Arts Council

SALIDA

OSHA FACT Sheet

Crystalline Silica Exposure
Health Hazard Information

What is crystalline silica?

Crystalline silica is a basic component of soil, sand, granite, and many other minerals. Quartz is the most common form of crystalline silica. Cristobalite and tridymite are two other forms of crystalline silica. All three forms may become respirable size particles when workers chip, cut, drill, or grind objects that contain crystalline silica.

What are the hazards of crystalline silica?

Silica exposure remains a serious threat to nearly 2 million U.S. workers, including more than 100,000 workers in high risk jobs such as abrasive blasting, foundry work, stonecutting, rock drilling, quarry work and tunneling. The seriousness of the health hazards associated with silica exposure is demonstrated by the fatalities and disabling illnesses that continue to occur in sandblasters and rockdrillers. Crystalline silica has been classified as a human lung carcinogen. Additionally, breathing crystalline silica dust can cause **silicosis**, which in severe cases can be disabling, or even fatal. The respirable silica dust enters the lungs and causes the formation of scar tissue, thus reducing the lungs' ability to take in oxygen. There is no cure for silicosis. Since silicosis affects lung function, it makes one more susceptible to lung infections like **tuberculosis**. In addition, smoking causes lung damage and adds to the damage caused by breathing silica dust.

What are the symptoms of silicosis?

Silicosis is classified into three types: chronic/classic, accelerated, and acute.

Chronic/classic silicosis, the most common, occurs after 15–20 years of moderate to low exposures to respirable crystalline silica. Symptoms associated with chronic silicosis may or may not be obvious; therefore, workers need to have a chest x-ray to determine if there is lung damage. As the disease progresses, the worker may experience shortness of breath upon exercising and have clinical signs of poor oxygen/carbon dioxide exchange. In the later stages, the worker may experience fatigue, extreme shortness of breath, chest pain, or respiratory failure.

Accelerated silicosis can occur after 5–10 years of high exposures to respirable crystalline silica. Symptoms include severe shortness of breath, weakness, and weight loss. The onset of symptoms takes longer than in acute silicosis.

Acute silicosis occurs after a few months or as long as 2 years following exposures to extremely high concentrations of respirable crystalline silica. Symptoms of acute silicosis include severe disabling shortness of breath, weakness, and weight loss, which often leads to death.

Where are construction workers exposed to crystalline silica?

Exposure occurs during many different construction activities. The most severe exposures generally occur during abrasive blasting with sand to remove paint and rust from bridges, tanks, concrete structures, and other surfaces. Other construction activities that may result in severe exposure include: jack hammering, rock/well drilling, concrete mixing, concrete drilling, brick and concrete block cutting and sawing, tuck pointing, tunneling operations.

Where are general industry employees exposed to crystalline silica dust?

The most severe exposures to crystalline silica result from abrasive blasting, which is done to clean and smooth irregularities from molds, jewelry, and foundry castings, finish tombstones, etch or frost glass, or remove paint, oils, rust, or dirt form objects needing to be repainted or treated. Other exposures to silica dust occur in cement and brick manufacturing, asphalt pavement manufacturing, china and ceramic manufacturing and the tool and die, steel and foundry industries. Crystalline silica is used in manufacturing, household abrasives, adhesives, paints, soaps, and glass. Additionally, crystalline silica exposures occur in the maintenance, repair and replacement of refractory brick furnace linings.

In the maritime industry, shipyard employees are exposed to silica primarily in abrasive blasting operations to remove paint and clean and prepare steel hulls, bulkheads, decks, and tanks for paints and coatings.

How is OSHA addressing exposure to crystalline silica?

OSHA has an established Permissible Exposure Limit, or PEL, which is the maximum amount of crystalline silica to which workers may be exposed during an 8-hour work shift (29 *CFR* 1926.55, 1910.1000). OSHA also requires hazard

Crystalline Silica Exposure
Health Hazard Information

communication training for workers exposed to crystalline silica, and requires a repirator protection program until engineering controls are implemented. Additionally, OSHA has a National Emphasis Program (NEP) for Crystalline Silica exposure to identify, reduce, and eliminate health hazards associated with occupational exposures.

What can employers/employees do to protect against exposures to crystalline silica?

- Replace crystalline silica materials with safer substitutes, whenever possible.
- Provide engineering or administrative controls, where feasible, such as local exhaust ventilation, and blasting cabinets. Where necessary to reduce exposures below the PEL, use protective equipment or other protective measures.
- Use all available work practices to control dust exposures, such as water sprays.
- Wear only a N95 NIOSH certified respirator, if respirator protection is required. Do not alter the respirator. Do not wear a tight-fitting respirator with a beard or mustache that prevents a good seal between the respirator and the face.
- Wear only a Type CE abrasive-blast supplied-air respirator for abrasive blasting.
- Wear disposable or washable work clothes and shower if facilities are available. Vacuum the dust from your clothes or change into clean clothing before leaving the work site.
- Participate in training, exposure monitoring, and health screening and surveillance programs to monitor any adverse health effects caused by crystalline silica exposures.
- Be aware of the operations and job tasks creating crystalline silica exposures in your workplace environment and know how to protect yourself.
- Be aware of the health hazards related to exposures to crystalline silica. Smoking adds to the lung damage caused by silica exposures.
- Do not eat, drink, smoke, or apply cosmetics in areas where crystalline silica dust is present. Wash your hands and face outside of dusty areas before performing any of these activities.
- Remember: If it's silica, it's not just dust.

How can I get more information on safety and health?

OSHA has various publications, standards, technical assistance, and compliance tools to help you, and offers extensive assistance through workplace consultation, voluntary protection programs, strategic partnerships, alliances, state plans, grants, training, and education. OSHA's *Safety and Health Program Management Guidelines* (*Federal Register* 54:3904-3916, January 26, 1989) detail elements critical to the development of a successful safety and health management system. This and other information are available on OSHA's website.

- For one free copy of OSHA publications, send a self-addressed mailing label to OSHA Publications Office, 200 Constitution Avenue N.W., N-3101, Washington, DC 20210; or send a request to our fax at (202) 693-2498, or call us toll-free at (800) 321-OSHA.
- To order OSHA publications online at **www.osha.gov**, go to **Publications** and follow the instructions for ordering.
- To file a complaint by phone, report an emergency, or get OSHA advice, assistance, or products, contact your nearest OSHA office under the U.S. Department of Labor listing in your phone book, or call toll-free at **(800) 321-OSHA (6742)**. The teletypewriter (TTY) number is (877) 889-5627.
- To file a complaint online or obtain more information on OSHA federal and state programs, visit OSHA's website.

This is one in a series of informational fact sheets highlighting OSHA programs, policies, or standards. It does not impose any new compliance requirements. For a comprehensive list of compliance requirements of OSHA standards or regulations, refer to *Title 29 of the Code of Federal Regulations*. This information will be made available to sensory-impaired individuals upon request. The voice phone is (202) 693-1999. See also OSHA's website at **www.osha.gov**.

This is one in a series of informational fact sheets highlighting OSHA programs, policies, or standards. It does not impose any new compliance requirements. For a comprehensive list of compliance requirements of OSHA standards or regulations, refer to *Title 29 of the Code of Federal Regulations*. This information will be made available to sensory-impaired individuals upon request. The voice phone is (202) 693-1999. See also OSHA's website at **www.osha.gov**.

OSHA
Occupational Safety and Health Administration
U.S. Department of Labor
2002

April 7, 2017

Open Letter to the Editor and Residents of Chaffee County

RE: **Adverse Health Effects of Proposed Gravel Pit, 14110 CR 140 Chaffee County**

As a physician and resident living near the proposed ACA gravel pit at 14110 CR 140, I am writing to express concern for the health effects that it will have on Chaffee County residents who will breathe the dust and diesel exhaust from this surface mining operation.

Dust from mining operations is a serious health hazard, producing airborne particulate matter including crystalline silica. Breathing crystalline silica dust can cause lung cancer, silicosis, COPD, kidney and autoimmune diseases. Studies show that silica presents a health danger not only to workers but also to residents exposed to silica dust. Exposed individuals are more susceptible to lung infections like tuberculosis, and even short term inhalation of the type of particles typical of gravel pit dust are associated with increased hospitalizations for heart disease. For the first time in 45 years, the Occupational Safety & Health Administration (OSHA) acknowledged recent scientific evidence showing that low-level exposures to silica causes serious health effects, and changed their rules of occupational exposure to protect workers. The fugitive dust from gravel pit mining may also contain toxins such as heavy metals and radionuclides such as radon, both of which cause cancer.

The dust from a newly dug gravel pit is likely even more toxic than long-standing dust, because excavation produces "freshly fractured" silica particles, which are even more biologically damaging than "aged" particles. Particulate matter from mining operations can travel hundreds of miles, but the proximity to existing and future Chaffee County residents make the proposed gravel pit site an unacceptable health hazard. Fugitive particulate emissions, air pollutant emissions, and visible emissions will be produced, and even if these emissions are within the allowable limits of state regulations, the potential health effects will not be eliminated.

Residents nearby the proposed gravel pit may have greater exposure than will gravel pit employees because the dust that lands on their yards, driveways, and inside their homes will be re-suspended during their daily activity, rather than merely during the hours of gravel pit operations. For nearby children and babies in utero, the public health consequences will be greatly magnified. Because of greater physical activity, higher metabolic rates, and hand-to-mouth actions, young children will be more exposed than adults via both inhalation and ingestion. Exposure of any pregnant women living nearby will extend the public health consequences to more than one generation because of the damage that industrial pollution and increased diesel exhaust can do to chromosomes and fetal development. Toxic dust generated by pit operations would continue for years, but the health consequences can last much longer.

Diesel emissions from trucks and other heavy equipment used at the pit will add to the health hazard. Diesel exhaust is a proven carcinogen and is more toxic than exhaust from gasoline vehicles. A recent landmark study indicates that long term exposure to even low levels of diesel exhaust raises the risk of dying from lung cancer about 50% for residents who live near industrial operations, and about 300% for the workers.

Based on the scientific evidence and proven health effects of pollutants produced by gravel pit mining, I consider the gravel pit proposal to be entirely incompatible with what should be Chaffee County officials' first priority – namely protection of public health, of families, of children, and of pregnant mothers living near this proposed pit and in nearby Poncha Springs and Salida. I appeal to all area residents to attend the upcoming Planning Commission meeting on April 25th at 6 pm and the Board of Commissioners meeting to be held May 9th at 9 am, both scheduled at the County Building in Salida, and urge our county leaders to reject the proposal. The fate of this proposed gravel pit may be decided at those meetings.

Sincerely,

Thomas Syzek MD

Thomas E. Syzek, MD, FACEP
Salida, Colorado

J. Med. Toxicol. (2012) 8:166–175
DOI 10.1007/s13181-011-0203-1

REVIEW ARTICLE

Clearing the Air: A Review of the Effects of Particulate Matter Air Pollution on Human Health

Jonathan O. Anderson · Josef G. Thundiyil · Andrew Stolbach

Published online: 23 December 2011

Abstract The World Health Organization estimates that particulate matter (PM) air pollution contributes to approximately 800,000 premature deaths each year, ranking it the 13th leading cause of mortality worldwide. However, many studies show that the relationship is deeper and far more complicated than originally thought. PM is a portion of air pollution that is made up of extremely small particles and liquid droplets containing acids, organic chemicals, metals, and soil or dust particles. PM is categorized by size and continues to be the fraction of air pollution that is most reliably associated with human disease. PM is thought to contribute to cardiovascular and cerebrovascular disease by the mechanisms of systemic inflammation, direct and indirect coagulation activation, and direct translocation into systemic circulation. The data demonstrating PM's effect on the cardiovascular system are strong. Populations subjected to long-term exposure to PM have a significantly higher cardiovascular incident and mortality rate. Short-term acute exposures subtly increase the rate of cardiovascular events within days of a pollution spike. The data are not as strong for PM's effects on cerebrovascular disease, though some data and similar mechanisms suggest a lesser result with smaller amplitude. Respiratory diseases are also exacerbated by exposure to PM. PM causes respiratory morbidity and mortality by creating oxidative stress and inflammation that leads to pulmonary anatomic and physiologic remodeling. The literature shows PM causes worsening respiratory symptoms, more frequent medication use, decreased lung function, recurrent health care utilization, and increased mortality. PM exposure has been shown to have a small but significant adverse effect on cardiovascular, respiratory, and to a lesser extent, cerebrovascular disease. These consistent results are shown by multiple studies with varying populations, protocols, and regions. The data demonstrate a dose-dependent relationship between PM and human disease, and that removal from a PM-rich environment decreases the prevalence of these diseases. While further study is needed to elucidate the effects of composition, chemistry, and the PM effect on susceptible populations, the preponderance of data shows that PM exposure causes a small but significant increase in human morbidity and mortality. Most sources agree on certain "common sense" recommendations, although there are lonely limited data to support them. Indoor PM exposure can be reduced by the usage of air conditioning and particulate filters, decreasing indoor combustion for heating and cooking, and smoking cessation. Susceptible populations, such as the elderly or asthmatics, may benefit from limiting their outdoor activity during peak traffic periods or poor air quality days. These simple changes may benefit individual patients in both short-term symptomatic control and long-term cardiovascular and respiratory complications.

Keywords Particulate matter · Air pollution · Cardiovascular · Respiratory · Public policy

J. O. Anderson (✉) · J. G. Thundiyil
Department of Medical Education,
Orlando Health Emergency Medicine,
86 W. Underwood, Suite 200,
Orlando, FL 32806, USA
e-mail: janders120@gmail.com

A. Stolbach
Johns Hopkins University Emergency Medicine,
1830 E. Monument St. Suite 6-100,
Baltimore, MD 21287, USA

Introduction

While some correlation between poor air quality and human disease has been recognized since antiquity, the health effects of air pollution entered the world's consciousness in the twentieth century. In 1930, sulfur dioxide from local

factory emissions mixed with a dense fog over the Meuse Valley in Belgium. Over 3 days, several thousand people were stricken with acute pulmonary symptoms, and 60 people died of respiratory causes [1]. In December 1952, a dense smog containing sulfur dioxide and smoke particulate descended upon London, resulting in more than 3,000 excess deaths over 3 weeks and as many as 12,000 through February 1953 [2]. The lethality of air pollution was immediately recognized but not well understood. To this day, because the effects of air pollution on illness occur at a population level, many clinicians fail to appreciate the relationship between air pollution and health.

The 1970 Clean Air Act (CAA) was the first major American regulatory effort aimed at both studying and setting limits on emissions and air pollution. The 1970 CAA defined the National Ambient Air Quality Standards (NAAQS [3]). These standards set limits on six primary pollutants found in air: carbon monoxide, lead, nitrogen dioxide, ozone, sulfur dioxide, and particulate matter (PM) [4].

PM is a complex mixture of extremely small particles and liquid droplets made up of acids, organic chemicals, metals, and soil or dust particles [5]. Sources of PM are both natural and anthropogenic. Manmade sources of PM include combustion in mechanical and industrial processes, vehicle emissions, and tobacco smoke. Natural sources include volcanoes, fires, dust storms, and aerosolized sea salt.

PM can be described by its "aerodynamic equivalent diameter" (AED). Particles of the same AED will tend to have the same settling velocity. Researchers traditionally subdivide particles into AED fractions based on how the particles are generated and where they deposit in human airways: <10, <2.5, and <0.1 μm (PM_{10}, $PM_{2.5}$, and $PM_{0.1}$, respectively). Particles with a diameter greater than 10 μm have a relatively small suspension half-life and are largely filtered out by the nose and upper airway. Researchers define a diameter between 2.5 and 10 μm ($PM_{2.5-10}$) as "coarse," less than 2.5 μm as "fine," and less than 0.1 μm as "ultrafine" particles. When interpreting PM research, it is important to appreciate that PM_{10} contains ultrafine ($PM_{0.1}$), fine ($PM_{0.1-2.5}$), and coarse ($PM_{2.5-10}$) fractions. In a mixed environmental sample, the total number and total surface area of these particles *increases* exponentially as the diameter of the particle decreases. However, the total particulate mass of a substance generally *decreases* exponentially with decreasing particle diameter. For example, in a sample of PM_{10}, the numerical majority of particles would be ultrafine, but these particles would make up a negligible portion of the sample's total particulate mass (Fig. 1).

Studies show an increase in morbidity and mortality related to PM exposure. While the increased daily risks from PM exposure are modest for any individual, the costs of the worldwide healthcare burden are staggering when applied to populations. The World Health Organization estimates that

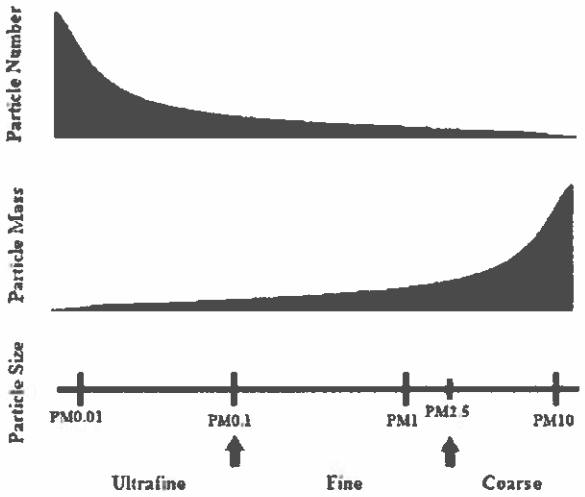


Fig. 1 A hypothetical mixed particle distribution

$PM_{2.5}$ concentration contributes to approximately 800,000 premature deaths per year, ranking it the 13th leading cause of mortality worldwide [6].

This paper provides a review of the effect of ambient airborne PM on human morbidity and mortality. We review the current understanding of the mechanisms that underlie the observed clinical findings. Emphasis is placed primarily on research concerning the cardiovascular, respiratory, and cerebrovascular systems. This review concludes with public health recommendations based on a summary of the reported literature's findings.

Methods

The authors conducted a scientific review of all available literature published over the last 30 years. Our primary objective was to determine the association or lack of association between PM and human health. Our secondary objective was to summarize the proposed mechanisms for any purported associations based on existing human, animal, and in vitro studies. We initiated a PubMed database search using the MESH terms "PM," "particulate matter," "air pollution," "ultrafine particles," "fine particles," "coarse particles," "PM10," "PM2.5," and "PM0.1." Articles were selected and agreed upon by the authors based on relevance and impact. Effort was made to provide both positive and negative studies where appropriate. Emphasis was placed on well-conducted trials and epidemiological investigations. Studies were only excluded for redundancy. After analysis of the available data, this paper concludes with individual and public health recommendations based on the existing scientific evidence.

PM and Cardiovascular Health Effects

Several large studies suggest that PM exerts significant effects on the cardiovascular system [7–9]. Research on this topic has focused on both the long-term effects of chronic PM exposure and the acute effects of increases in ambient PM on cardiovascular mortality. In a previous analysis [10], it was shown that for any increase in mortality caused by PM, two thirds of the effect was accounted for by the cardiovascular diseases.

Cardiovascular Mechanisms

Animal studies demonstrate a link between chronic PM exposure and the development of atherosclerosis via systemic inflammation [11, 12]. Human studies show that the effects appear to be mediated by the inflammatory cytokines IL-6, TNF-ά, and C-reactive protein (CRP). Increases in both IL-6 [13] and CRP [14] have been associated with the development of acute myocardial infarction. Ruckerl et al. [15] described transient IL-6 and TNF-ά elevations in diabetic patients for 2 days following PM10 exposure. In a prospective cohort study of German patients, Hoffman et al. [16] associated exposure to PM2.5 with elevations in CRP. Other researchers demonstrated similar increases in CRP from PM10 exposure from both combustion [17] and organic matter [18]. In contrast, some studies have found only a weak or absent link between PM and markers of inflammation [19–22]. Discrepancies among studies appear related to differences in composition of PM, variable exposure to anti-inflammatory medications, and differences in obtaining PM exposure data [10].

Acute exposure to PM causes changes in coagulation and platelet activation providing a more proximal link between PM and coronary artery disease. Many experts consider fibrinogen to be an important risk factor for cardiovascular disease [10]. Ruckerl et al. [15] associated a 5-day cumulative exposure to PM_{10} with increased fibrinogen levels in survivors of myocardial infarction. Other pro-coagulant factors, such as plasminogen activator fibrinogen inhibitor-1 (PAI-1), were also associated with PM elevations [17]. Intratracheal instillation of diesel exhaust particles led to increased platelet activation in hamsters and rapid thrombosis formation [23]. Further hamster studies also suggested that small particles translocate into the blood stream and exert prothrombotic effects [24]. Schicker et al. [18] showed that transient increases in PM10 exposure caused during hay-stacking increased platelet aggregation within 2 h of the activity. This activity also increased Von Willebrand factor and Factor VIII, markers of vascular endothelial activation.

Long-Term Effects

The "Harvard Six Cities study [7]," a cohort study published in 1993, followed 8,111 patients for 16–18 years and showed a 29% (95% CI, 8–47%) increase in the adjusted mortality rate for the most polluted of the cities compared to the least polluted. Particulate air pollution was positively associated with death from lung cancer and cardiopulmonary disease (Table 1).

Pope et al. [8] followed this in 1995 with another prospective cohort study of 552,000 patients in 151 metropolitan areas using the American Cancer Society's Cancer Prevention 2 database (ACS CPS 2). These data showed a 17% (95% CI, 9–26%) increase in all-cause mortality and a 31% (95% CI, 17–46%) increase in cardiopulmonary mortality when comparing the most and least polluted cities. In 2002 [25] and 2004 [26], Pope et al. re-reviewed the expanding ACS CPS 2 database, now with 1.2 million participants, and extended the follow up. Their research demonstrated an average increase in cardiopulmonary mortality of 9% (95% CI, 3–16%) for each 10-μg/m^3 increase in $PM_{2.5}$. Subsequently, they determined that a 10-μg/m^3 increase in PM increased ischemic cardiovascular disease mortality by 18% (95% CI, 14–23%) and mortality from arrhythmia, congestive heart failure, and cardiac arrest by 13% (95% CI, 5–21%).

In 2007, the Women's Health Initiative Study [27] followed a cohort of over 65,000 postmenopausal women with no previous

Table 1 Long-term effects of PM on the cardiovascular system

Author	Year	PM	ΔPM (in μg/m^3)	Outcome measure	Effect (95% CI)
Dockery et al. [17]	1993	PM_{10}	18.6	All-cause mortality	26% (8–47)
Pope et al. [18]	1995	PM_{10}	24.5	All-cause mortality	17% (9–26)
		PM_{10}	24.5	Cardiopulmonary mortality	31% (17–46)
Hoek et al. [28]	2002	BS	10.3	Cardiopulmonary mortality	71% (10–167)
Pope et al. [25]	2002	$PM_{2.5}$	10	Cardiopulmonary mortality	9% (3–16)
Pope et al. [26]	2004	$PM_{2.5}$	10	Ischemic CVD mortality	18% (14–23)
		$PM_{2.5}$	10	CHF, arrhythmia, CP arrest	13% (5–21)
Miller et al. [27]	2007	$PM_{2.5}$	10	Cardiovascular event	24% (9–41)
		$PM_{2.5}$	10	Cardiovascular mortality	76% (25–147)
Toren et al. [29]	2007	PM	Not measured	Cardiovascular mortality	12% (7–19)

PM particulate matter, *ΔPM* increase in ambient PM, *BS* black smoke

heart disease over approximately 6 years. The investigators revealed that long-term PM exposure in this population resulted in a 24% (95% CI, 9–41%) increase in cardiovascular events and an astonishing 76% (95% CI, 25–147%) increase in cardiovascular mortality per 10-μg/m^3 increase in $PM_{2.5}$. While these results had fairly wide confidence intervals, these data suggest that this cohort of patients may be particularly susceptible to ambient PM exposure.

The findings of cardiovascular effects from PM exposure are not unique to the USA. In the Netherlands, long-term exposure to traffic-related air pollution increased cardiopulmonary mortality by 71% (95% CI, 10–167%) [28]. A 2007 cohort study [29] of 250,000 Swedish construction workers from 1972 to 2002 found that workers with occupational PM exposure had a 12% (95% CI, 7–19%) increase in ischemic cardiovascular disease mortality.

While increases in PM have been consistently shown to increase cardiovascular morbidity and mortality, the effects of PM reduction have also been studied. In the 72 months following the ban of bituminous coal sales in Ireland in 1990, black smoke concentration decreased by 35.6 μg/m^3 over this time, and standardized respiratory and cardiovascular mortality decreased by 15.5% (95% CI, 12–19%) and 10.3% (95% CI, 8–13%), respectively [30]. An 8-year extension of the Harvard Six Cities data studied the population subset that moved from areas of higher to lower PM concentration [31], finding that a 10-μg/m^3 decrease in $PM_{2.5}$ resulted in a 27% (95% CI, 5–43%) decrease in overall mortality.

Short-Term Effects

A 2001 review [32] of 12 prior studies concluded that a 10-μg/m^3 increase in PM_{10} increased hospital admissions for congestive heart failure and ischemic heart disease by 0.8% (95% CI, 0.5–1.2%) and 0.7% (95% CI, 0.4–1.0%), respectively. Similarly, a 2006 review [33] showed a 0.44% (95% CI, 0.02–0.86%) and 1.28% (95% CI, 0.78–1.78%) increase in admissions for ischemic heart disease and heart failure for a 10-μg/m^3 increase in $PM_{2.5}$, respectively. In a smaller trial, Pope et al. [34] used a case-crossover of 12,000 patients in Utah to show that a 10-μg/m^3 increase in $PM_{2.5}$ led to a 4.5% (95% CI, 1.1–8.0%) increase in acute ischemic coronary events. In an analysis of PM concentrations from 20 major cities in the USA using the National Morbidity Mortality Air Pollution Study (NMMAPS) data, Samet et al. [9] showed a 10-μg/m^3 increase in PM_{10} caused an increase in all-cause and cardiopulmonary mortality by 0.5% (95% CI, 0.1–0.9%) and 0.7% (95% CI, 0.2–1.2%), respectively (Table 2).

Similar results have been found in Japan [35], Australia, and New Zealand [36]. In 2008, Samoli et al. [37] re-analyzed the data of the APHEA 2, NMMAPS, and several Canadian studies in order to assess the coherence of findings using the same methods for all three sets of data. They were able to show an increase in daily all-cause mortality for Canadian, European, and US cities. Interestingly, the short-term mortality resulting from acute increases in PM are not limited to the critically ill or dying. In fact, much of the mortality occurred among active individuals with one or more risk factors.

PM and Respiratory Health Effects

While much of the interest in PM has focused on the cardiovascular system [7, 8], many studies evaluated the association between PM exposure and respiratory illness. Researchers have evaluated endpoints including respiratory symptoms, medication use, lung function, health-care utilization, and mortality.

Table 2 Short-term effects of PM on the cardiovascular system

Author	Year	PM	ΔPM (in μg/m^3)	Outcome measure	Effect
Morris [32]	2001	PM_{10}	10	Hospital admission, IHD	0.7% (95% CI, 0.4–1.0)
		PM_{10}	10	Hospital admission, CHF	0.8% (95% CI, 0.5–1.2)
Domicini et al. [33]	2006	$PM_{2.5}$	10	Hospital admission, IHD	0.44% (95% CI, 0.02–0.86)
		$PM_{2.5}$	10	Hospital admission, CHF	1.28% (95% CI, 0.78–1.78)
Barnett et al. [36]	2006	$PM_{2.5}$	10	Hospital admission, IHD	1.6% (95% CI, 0.7–2.4)
		$PM_{2.5}$	10	Hospital admission, CHF	3.6% (95% CI, 1.8–5.4)
		$PM_{2.5}$	10	Hospital admission, AMI	2.7% (95% CI, 1.3–4.2)
Pope et al. [34]	2006	$PM_{2.5}$	10	Ischemic cardiac event	4.5% (95% CI, 1.1–8.0)
Samet et al. [9]	2000	PM_{10}	10	All-cause mortality	0.5% (95% CI, 0.1–0.9)
		PM_{10}	10	Cardiopulmonary mortality	0.7% (95% CI, 0.2–1.2)
Omori et al. [35]	2003	TSP	20	All-cause mortality	1.0% (95% CI, 0.8–1.3)
		TSP	20	Cardiopulmonary mortality	1.1% (95% CI, 0.7–1.5)

PM particulate matter, *ΔPM* increase in ambient PM, *TSP* total suspended particles, *IHD* ischemic heart disease, *CHF* congestive heart failure, *AMI* acute myocardial infarction

Respiratory Mechanisms

PM triggers pulmonary oxidative stress and inflammation. Human airway epithelial cells exposed to PM express inflammatory cytokines [38, 39]. Alveolar macrophages exhibit respiratory burst activity, producing reactive oxygen species, nitrogen species, and release TNF-α and IL-1 after exposure [40]. In addition to oxidative stress generated from activation of inflammatory cells, reactive oxygen species may be directly generated from the surface of particles [41]. These responses can be potent and were shown to cause measurable pulmonary damage after only a single exposure in mice [42]. This oxidative damage is associated with the primary development of asthma and chronic obstructive pulmonary disease (COPD). Long-term exposure to PM results in airway remodeling and chronic inflammation [43]. PM may also contribute to asthma development by enhancing atopy and IgE responses [44, 45]. Several controlled human experiments have demonstrated adverse affects on the pulmonary system. PM exposure has been shown to increase airway responsiveness to methacholine [46], increase neutrophil numbers in bronchial lavage [47], decrease CO diffusion capacity, and decrease maximum mid-expiratory flow [48].

Respiratory Symptoms and Medication Usage

As part of the Children's Health Study, McConnell et al. [49] found that asthmatic children had a 40% (95% CI, 10–80%) increased risk of bronchitic symptoms for a 19-μg/m^3 increase in PM_{10}. Similarly, a 10-μg/m^3 increase in PM_{10} led to a 12% (95% CI, 4–22%) increase in severe asthma symptoms in Seattle children [50]. A study of inner-city asthmatic children revealed an association between $PM_{2.5}$ increases and missed school days for asthma [51]. A study of adult Parisians [52] showed a 41% (95% CI, 16–71%) increase in acute asthma exacerbations per 10-μg/m^3 increase in PM_{10}. Interestingly, nearly all PM levels in these studies were below levels set out in the NAAQS.

Respiratory medication use also increased in times of peak PM concentration. Use of rescue bronchodilators increased as ambient $PM_{2.5}$ rose in Denver [53] and the Northeast USA [54]. A review of 80,000 Alaskan Medicaid enrollees found prescription rates for bronchodilators increased by 18.1% and 28.8% when PM_{10} exceeded 34 and 61 μg/m^3, respectively [55]. Together, these data suggest that increases in ambient PM worsen asthma symptoms.

PM and Pulmonary Function

Several recent studies suggest that PM levels may affect lung function and lung development. The Children's Health Study [56] followed 1,759 patients over 8 years, finding that children who lived in communities with the highest PM concentrations were five times more likely to have low FEV1 than those in communities with the lowest PM concentrations. Moreover, children that moved from areas of higher to lower PM_{10} concentration had increased growth in lung function, and those that moved from areas of lower to higher PM_{10} concentration had decreased growth in lung function [57]. Even children with better lung function were susceptible to new onset asthma when exposed to higher levels of $PM_{2.5}$ [58]. Lower lung function has also been shown for children with cystic fibrosis exposed to higher levels of PM_{10} and $PM_{2.5}$ [59].

Similar inverse correlations between PM exposure and individual PEFR and FEV1 measurements have been reproduced internationally [60]. In the developing world, where indoor biomass burning can lead to PM levels exceeding 200 μg/m^3, researchers demonstrated that chronic exposure in children can lead to adult COPD, increased rates of lung infection, and impaired lung function [61].

In adults, effects of PM on lung function have been found primarily in susceptible populations. Investigators showed that asthmatic Londoners taking walks in areas of high PM had significantly higher reduction in FEV1, FVC, and increases in sputum biomarkers of inflammation [62]. In elderly patients, PM_{10} and $PM_{2.5}$ increases were associated with decreases in PEFR [63]. In COPD patients, decrements in lung function were associated with increases in $PM_{2.5}$ concentration [64]. Downs et al. [65] demonstrated that declines in PM_{10} concentration may actually lead to an attenuated decline in lung function in adult patients. However, research on healthy adults has not as consistently shown an association between PM and respiratory compromise [66].

PM and Respiratory-Related Healthcare Utilization

In a large case–control study [67], 10 μg/m^3 increases in $PM_{2.5}$ were associated with a 9% (95% CI, 4–14%) increase in bronchiolitis hospitalizations for infants. Large pediatric studies demonstrate increased asthma ED visits for increases in PM [68] and that PM_{10} increases of 6.5 μg/m^3 are associated with a 15% (95% CI, 2–30%) increase in respiratory-related hospital admissions [69] (Table 3).

For adults, several large studies have demonstrated an association between respiratory hospitalization and ambient PM_{10} [70] and $PM_{2.5}$ [71] concentrations. This includes admissions for asthma, COPD, and pneumonia. The effects appear to be stronger for elderly patients with even short-term exposures [72]. A study [73] of 12 million Medicare enrollees in 108 counties demonstrated significant increases in respiratory hospitalizations for increases in $PM_{2.5}$ in the Eastern USA. Because the same effects were not consistently observed in the Western USA, the authors suggested that morbidity may be related to the specific chemical constituents of PM which differs across the country. Several recent

Table 3 The effects of PM on respiratory admissions

Author	Year	PM	ΔPM (in μg/m^3)	Outcome measure	Effect (95% CI)
Karr et al. [67]	2006	$PM_{2.5}$	10	Infant bronchiolitis admissions	9% (4–14)
Lin et al. [68]	2005	$PM_{10-2.5}$	6.5	Pediatric respiratory admissions	17% (6–29)
Samoli et al. [92]	2011	PM_{10}	10	Pediatric asthma admissions	2.54% (0.06–5.08)
Peng et al. [93]	2008	$PM_{10-2.5}$	10	Respiratory admissions	0.33% (−0.21–0.86)
Zanobetti et al. [70]	2009	$PM_{2.5}$	10	Respiratory admissions	2.07% (1.2–2.95)
		$PM_{2.5}$	17	Pneumonia admissions	6.5% (1.1–11.4)
Medina-Ramon et al. [71]	2006	PM_{10}	10	COPD admissions	1.47% (0.93–2.01)
		PM_{10}	10	Pneumonia admissions	0.84% (0.5–1.19)
Dominici et al. [33]	2006	$PM_{2.5}$	10	COPD admissions	1.61% (0.56–2.66)
McGowan et al. [77]	2001	PM_{10}	14.8	Respiratory admissions	3.37% (2.34–4.40)
Ostro et al. [94]	2009	$PM_{2.5}$	14.6	Pediatric respiratory admissions	4.1% (1.8–6.4)

PM particulate matter, *ΔPM* increase in ambient PM

large studies have provided further evidence that the strength of PM effect may depend on the composition [74]. Investigations in European cities [75], Asian cities [76], and Oceania cities [77] have demonstrated a consistent and small though significant association between PM concentrations and emergency visits for respiratory diseases.

PM and Respiratory Mortality

The Six Cities study [7], 20 cities study [9], and ACS CPS 2 [8] cohort revealed an association between PM exposure and cardiopulmonary mortality. These studies did not, however, separate the impact on respiratory mortality versus cardiovascular mortality. A follow-up investigation using data from the 20 Cities Study revealed a 0.87% (95% CI, 0.38–1.36%) increased respiratory mortality for short-term increases in PM_{10} by 10 μg/m^3 [78]. This was subsequently expanded into a larger cohort of 112 US cities, where researchers found a 1.68% (95% CI, 1.04–2.33%) increase in respiratory mortality for every 10-μg/m^3 increase in $PM_{2.5}$ [79]. A study of California counties similarly revealed an increased respiratory mortality with increases in PM_{10} [80].

These results have been reproduced in countries around the world. A Norwegian study [81] demonstrated a 17% (95% CI, 9–25%) increase in mortality risk from COPD for every quartile increase in $PM_{2.5}$. In a study of 275,000 adults in ten Italian cities [82], short-term PM_{10} increases led to a 2.29% (95% CI, 1.03–3.58%) increase in respiratory mortality. Similar results for increased respiratory mortality have been found in Asian cities where researchers have demonstrated excess respiratory mortality risk for increases in PM_{10} [83]. Nearly identical effect sizes for respiratory mortality were found in the APHEA2 trial which studied this relationship across 29 European cities [84]. One study even demonstrated an association between PM_{10} and respiratory mortality in children under age five [85] (Table 4).

PM and Cerebrovascular Health Effects

Ischemic cerebrovascular and cardiovascular disease share many risk factors, features, and pathophysiological mechanisms. As an example, CRP, similar to cardiovascular disease, has also been implicated in the genesis of stroke [86].

Table 4 The effects of PM on respiratory mortality

Author	Year	PM	ΔPM (in μg/m^3)	Outcome measure	Effect (95% CI)
Zeka et al. [78]	2005	PM_{10}	10	Respiratory mortality	0.87% (0.38–1.36)
Zanobetti et al. [79]	2009	$PM_{2.5}$	10	Respiratory mortality	1.68% (1.04–2.33)
Wong et al. [83]	2008	PM_{10}	10	Respiratory mortality	0.62% (0.22–1.02)
Analitis et al. [84]	2006	PM_{10}	10	Respiratory mortality	0.58% (0.21–0.95)
Hales et al. [91]	2010	PM_{10}	10	Respiratory mortality	1.3% (0.5–2.1)
Pope et al. [25]	2002	$PM_{2.5}$	10	Lung cancer mortality	8% (1–16)
Ostro et al. [80]	2006	$PM_{2.5}$	10	Respiratory mortality	2.2% (0.6–3.9)

PM particulate matter, *ΔPM* increase in ambient PM

However, the evidence linking PM and stroke is more sporadic and the mechanisms less well understood.

Dominici et al. [33] reviewed an air quality data for 204 US urban counties and showed that a 10-μg/m^3 increase in ambient $PM_{2.5}$ increased the risk of hospitalization for cerebrovascular events by 0.8% (95% CI, 0.3–1.3%). A separate review [87] of Medicare patients found an increase of 1.03% (95% CI, 0.04–2.04%) for hospital admission for ischemic stroke for each 10-μg/m^3 increase in PM_{10}. Still other investigators found a previous day $PM_{2.5}$ increase of 5.2 μg/m^3 led to a 3% (95% CI, 0–7%) increase in risk of TIA and ischemic stroke.

In contrast, a recent large prospective multi-center stroke registry found no increase in the general population for ischemic stroke from exposure to $PM_{2.5}$. There was, however, an 11% (95% CI, 1–22%) increase in stroke risk in exposed patients with diabetes [88]. A large case-crossover study found an association between other components of air pollution (NO2 and CO) and cerebrovascular disease, but no correlation was noted with changing PM levels [89]. Similarly, a large registry of first-ever strokes found no association with PM_{10} for ischemic or hemorrhagic stroke [90].

There are several reasons why studies of PM and cerebrovascular disease have produced conflicting results. Some studies do not completely adjust for all confounding variables. There is further heterogeneity due to differences in the definition of cerebrovascular disease, or whether pollution is measured on the day of admission or symptom onset [88]. Further, it is possible that exposure to PM may not contribute to an overall increase in cerebrovascular disease, but only trigger events in vulnerable populations.

Recommendations and Conclusions

In evaluating the literature, there appears to be a small, but consistent and significant, effect of PM on human health. Overall, the small individual effects result in a large global public health burden. Notably, the effects are most pronounced for cardiovascular disease. Several studies have demonstrated an increase in cardiovascular mortality and hospitalizations. There are similar effects, of smaller amplitude, in respiratory disease. More study is needed to clarify the relationship between PM and cerebrovascular disease.

There are limitations to much of the available PM research. Most studies do not use individual exposure data. Rather, air monitors in population centers are used as surrogates for individual exposure. Even after adjusting these data for time spent in traffic, exposure to second-hand smoke, etc., estimates may not be accurate. Despite these limitations, different types of studies conducted in different locations find similar results. A dose–response relationship between PM exposure and adverse effects has been identified, and improvement in health endpoints is observed when the PM exposures are reduced. Overall, the available evidence suggests a causal association between long- and short-term PM exposure and cardiovascular and respiratory morbidity and mortality.

Further research is still needed to fully understand how PM affects human health. While studies show increased PM concentration has adverse health affects, the actual composition of particulates that is harmful has not yet been elucidated. Further studies are also needed to clarify the time course of PM-induced effects. In limited studies, some effects seem to appear within hours, while other reach their zenith within several days peak PM exposure. The data on this "lag time" effect can

Table 5 Air quality index and recommendations

AQI level	AQI value	$PM_{2.5}$	PM_{10}	Actions to protect your health from particle pollution
Good	0–50	0–15	0–50	None
Moderate	51–100	16–35	51–154	Unusually sensitive people should consider reducing prolonged or heavy exertion
Unhealthy for sensitive groups	101–150	36–65	155–254	Susceptible groups[a] should reduce prolonged or heavy exertion
				Everyone else should limit prolonged or heavy exertion
Unhealthy for sensitive groups	151–200	66–150	255–354	Susceptible groups[a] should avoid all physical activity outdoors
				Everyone else should avoid prolonged or heavy exertion
Very unhealthy	201–300	>150	>354	Susceptible groups[a] should remain indoors and keep activity levels low
				Everyone else should avoid all physical activity outdoors

EPA-456/F-09-002

Air quality index: a guide to air quality and your health. EPA, August 2009

AQI air quality index

[a]People with heart or lung disease, children, or older adults

be contradictory, and this phenomenon remains incompletely understood. The true biological mechanisms leading to PM-induced pathology continue to be investigated. Also, while regional exposure data has become standard for PM epidemiology, studies with true individual exposure have yet to be fully realized. Finally, studies defining susceptible populations will help to shape further population-based recommendations.

Clinical Recommendations

When a patient presents with an acute illness, the clinician will not be able to determine the degree to which PM contributed. In illnesses where PM is known to contribute to risk, that percentage risk increase is usually measured in the single digits. Therefore, it is unlikely that there will ever be specific therapies for PM-related illness. Rather, health care providers should be familiar with prevention strategies for PM-related illness. Indoor PM exposure can be minimized by using air conditioning, particulate air filters, avoiding use of indoor combustion for cooking and heating, and smoking cessation [95]. Susceptible groups may benefit from limiting their outdoor exercise during peak traffic periods or poor air quality days [96]. The Air Quality Index (AQI) (http://airnow.gov) provides up-to-date information regarding local concentrations of PM and other pollutants. While government agencies have put out recommendations for minimizing PM exposure, peer-reviewed controlled data are limited for the implementation of these recommendations (Table 5).

Though PM exposure is ubiquitous, there is no defined and studied "safe" level. Patient education and behavioral modification strategies may contribute to better overall health. Additionally, these data can enable policy makers, after weighing the economic impact, to enforce or strengthen existing legislation that limits PM exposure. Volcanoes, forest fires, and other natural PM sources are part of our world and are unavoidable. However, by reducing modifiable PM exposure, we will likely see reductions in morbidity and mortality.

References

1. Nemery B, Hoek P, Nemmar A (2001) The Meuse Valley fog of 1930: an air pollution disaster. Lancet 357:704–708
2. Bell ML, Davis DD (2001) Reassessment of the lethal London fog of 1952: novel indicators of acute and chronic consequences of acute exposure to air pollution. Environ Health Perspect 109(3): 389–394
3. Anonymous. "History | Clean Air Act | US EPA". US Environmental Protection Agency, 16 Nov 2010. http://www.epa.gov/air/caa/caa_history.html. Accessed on 22 July 2011
4. Anonymous. "National Ambient Air Quality Standards (NAAQS) | Air and Radiation | US EPA". US Environmental Protection Agency, 19 July 2011. http://www.epa.gov/air/criteria.html. Accessed on 22 July 2011
5. Anonymous. "Particulate Matter | Air & Radiation | US EPA". US Environmental Protection Agency, 6 July 2011. http://www.epa.gov/pm/. Accessed on 22 July 2011
6. World Health Organization (2002) World health report 2002. World Health Organization, Geneva
7. Dockery D, Pope C, Xu X, Spengler J, Ware J et al (1993) An association between air pollution and mortality in six U.S. cities. NEJM 329(24):1753–1759
8. Pope C, Thun M, Namboodiri M, Dockery D, Evans J et al (1995) Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med 151:669–674
9. Samet J, Dominici F, Curriero F, Coursac I, Zeger S (2000) Fine particulate air pollution and mortality in 20 U.S. cities, 1987–1994. NEJM 343(24):1742–1749
10. Brook R, Rajagopalan S, Pope A, Brook J, Bhatnagar A, Diez-Roux A et al (2010) Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. Circulation 121:2331–2378
11. Chen L, Nadziejko C (2005) Effects of subchronic exposures to concentrated ambient particles (CAPs) in mice. V. CAPs exacerbate aortic plaque development in hyperlipidemic mice. Inhal Toxicol 17(4–5):217–224
12. Sun Q, Wang A, Jin X, Natanzon A, Duquaine D et al (2005) Long-term air pollution exposure and acceleration of atherosclerosis and vascular inflammation in an animal model. JAMA 294 (23):3003–3010
13. Ridker P, Rifai N, Stampfer M, Hennekens C (2000) Plasma concentration of interleukin-6 and the risk of future myocardial infarction among apparently healthy men. Circulation 101(15): 1767–1772
14. Wennberg P, Wensley F, Di Angelantonio E, Johansson L, Boman K et al (2011) Haemostatic and inflammatory markers are independently associated with myocardial infarction in men and women. Thromb Res (in press)
15. Rückerl R, Ibald-Mulli A, Koenig W, Schneider A, Woelke G et al (2006) Air pollution and markers of inflammation and coagulation in patients with coronary heart disease. Am J Respir Crit Care Med 173(4):432–441
16. Hoffmann B, Moebus S, Dragano N, Stang A, Möhlenkamp S et al (2009) Chronic residential exposure to particulate matter air pollution and systemic inflammatory markers. Environ Health Perspect 117(8):1302–1308
17. Chuang K, Chan C, Su T, Lee C, Tang C (2007) The effect of urban air pollution on inflammation, oxidative stress, coagulation, and autonomic dysfunction in young adults. Am J Respir Crit Care Med 176(2):370–376
18. Schicker B, Kuhn M, Fehr R, Asmis L, Karagiannidis C et al (2009) Particulate matter inhalation during hay storing activity induces systemic inflammation and platelet aggregation. Eur J Appl Physiol 105(5):771–778
19. Sullivan J, Hubbard R, Liu S, Shepherd K, Trenga C et al (2007) A community study of the effect of particulate matter on blood measures of inflammation and thrombosis in an elderly population. Environ Health 6:3
20. Steinvil A, Kordova-Biezuner L, Shapira I, Berliner S, Rogowski O (2008) Short-term exposure to air pollution and inflammation-sensitive biomarkers. Environ Res 106(1):51–61
21. Forbes L, Patel M, Rudnicka A, Cook D, Bush T et al (2009) Chronic exposure to outdoor air pollution and markers of systemic inflammation. Epidemiology 20(2):245–253
22. Diez Roux A, Auchincloss A, Astor B, Barr R, Cushman M et al (2006) Recent exposure to particulate matter and C-reactive

protein concentration in the multi-ethnic study of atherosclerosis. Am J Epidemiol 164(5):437–448
23. Nemmar A, Hoet P, Dinsdale D, Vermylen J, Hoylaerts M et al (2003) Diesel exhaust particles in lung acutely enhance experimental peripheral thrombosis. Circulation 107(8):1202–1208
24. Nemmar A, Hoylaerts M, Hoet P, Dinsdale D, Smith T et al (2002) Ultrafine particles affect experimental thrombosis in an in vivo hamster model. Am J Respir Crit Care Med 166:998–1004
25. Pope C, Burnett R, Thun M, Calle E, Krewski D et al (2002) Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 287:1132–1141
26. Pope C, Burnett R, Thurston G, Thun M, Calle E et al (2004) Cardiovascular mortality and long-term exposure to particulate air pollution. Circulation 109:71–77
27. Miller K, Siscovick D, Sheppard L, Shepherd K, Sullivan J et al (2007) Long-term exposure to air pollution and incidence of cardiovascular events in women. NEJM 356:447–458
28. Hoek G, Brunekreef B, Goldbohm S, Fischer P, van den Brandt P (2002) Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study. Lancet 360:1203–1209
29. Toren K, Bergdahl I, Nilsson T, Jarvholm B (2007) Occupational exposure to particulate air pollution and mortality due to ischaemic heart disease and cerebrovascular disease. Occup Environ Med 64:515–519
30. Clancy L, Goodman P, Sinclair H, Dockery D (2002) Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study. Lancet 360:1210–1214
31. Laden F, Schwartz J, Speizer FE, Dockery DW (2006) Reduction in fine particulate air pollution and mortality: extended follow-up of the Harvard Six Cities study. Am J Respir Crit Care Med 173:667–672
32. Morris R (2001) Airborne particulates and hospital admissions for cardiovascular disease: a quantitative review of the evidence. Environ Health Perspect 109(4):495–500
33. Dominici F, Peng R, Bell M, Pham L, McDermott A et al (2006) Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA 295(10):1127–1134
34. Pope C, Muhlestein J, May H, Renlund D, Anderson J et al (2006) Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution. Circulation 114:2443–2448
35. Omori T, Fujimoto G, Yoshimura I, Nitta H, Ono M (2003) Effects of particulate matter on daily mortality in 13 Japanese cities. J Epidemiol 13:314–322
36. Barnett A, Williams G, Schwartz J, Best T, Neller A et al (2006) The effects of air pollution on hospitalizations for cardiovascular disease in elderly people in Australian and New Zealand cities. Environ Health Perspect 114(7):1018–1023
37. Samoli E, Peng R, Ramsay T, Pipikou M, Touloumi G et al (2008) Acute effects of ambient particulate matter on mortality in Europe and North America: results from the APHENA study. Environ Health Perpect 116:1480–1486
38. Silbajoris R, Osornio-Vargas A, Simmons S, Reed W, Bromberg P et al (2011) Ambient particulate matter induces IL-8 expression through an alternative NF-κB mechanism in human airway epithelial cells. Environ Health Perspect 119:1379–1383
39. Quay J, Reed W, Samet J, Devlin R (1998) Air pollution particles induce IL-6 gene expression in human airway epithelial cells via NF-kappaB activation. Am J Respir Cell Mol Biol 19(1):98–106
40. Driscoll K, Lindenschmidt R, Maurer J, Higgins J, Ridder G (1990) Pulmonary response to silica or titanium dioxide: inflammatory cells, alveolar macrophage-derived cytokines, and histopathology. Am J Respir Cell Mol Biol 2(4):381–390
41. Risom L, Moller P, Loft S (2005) Oxidative stress-induced DNA damage by particulate air pollution. Mutat Res 592(1–2):119–137
42. Riva D, Magalhães C, Lopes A, Lanças T, Mauad T et al (2011) Low dose of fine particulate matter (PM2.5) can induce acute oxidative stress, inflammation and pulmonary impairment in healthy mice. Inhal Toxicol 23(5):257–267
43. Hogg J, Chu F, Utokaparch S, Woods R, Elliott W et al (2004) The nature of small-airway obstruction in chronic obstructive pulmonary disease. NEJM 350(26):2645–2653
44. Diaz-Sanchez D, Tsien A, Casillas A, Dotson A, Saxon A (1996) Enhanced nasal cytokine production in human beings after in vivo challenge with diesel exhaust particles. J Allergy Clin Immunol 98(1):114–123
45. Diaz-Sanchez D, Tsien A, Fleming J, Saxon A (1997) Combined diesel exhaust particulate and ragweed allergen challenge markedly enhances human in vivo nasal ragweed-specific IgE and skews cytokine production to a T helper cell 2-type pattern. J Immunol 158(5):2406–2413
46. Nordenhall C, Pourazar J, Ledin M, Levin J, Sandstrom T et al (2001) Diesel exhaust enhances airway responsiveness in asthmatic subjects. Eur Respir J 17:909–915
47. Behndig A, Mudway I, Brown J, Stenfors N, Helleday R et al (2006) Airway antioxidant and inflammatory responses to diesel exhaust exposure in healthy humans. Eur Resp J 27:359–365
48. Pietropaoli A, Frampton M, Hyde R, Morrow P, Oberdorster G et al (2004) Pulmonary function, diffusing capacity, and inflammation in healthy and asthmatic subjects exposed to ultrafine particles. Inhal Toxicol 16(1):59–72
49. McConnell R, Berhane K, Gilliland F, London S, Vora H et al (1999) Air pollution and bronchitic symptoms in southern California children with asthma. Environ Health Perspect 107(9):757–760
50. Slaughter J, Lumley T, Sheppard L, Koenig J, Shapiro G et al (2003) Effects of ambient air pollution on symptom severity and medication use in children with asthma. Ann Allergy Asthma Immunol 91(4):346–353
51. O'Connor G, Neas L, Vaughn B, Kattan M, Mitchell H et al (2008) Acute respiratory health effects of air pollution on children with asthma in US inner cities. J Aller Clin Immunol 121:1133–1139
52. Desqueyroux H, Pujet J, Prosper M, Squinzai F, Momas I (2002) Short-term effects of low-level air pollution on respiratory health of adults suffering form moderate to severe asthma. Environ Res 89:29–37
53. Rabinovitch N, Strand M, Gelfand E (2006) Particulate levels are associated with early asthma worsening in children with persistent disease. Am J Resp Crit Care Med 173:1098–1105
54. Gent J, Koutrakis P, Belanger B, Triche E, Holford T et al (2009) Symptoms and medication use in children with asthma and traffic-related sources of fine particle pollution. Environ Health Perspect 117(7):1168–1174
55. Chimonas M, Gessner B (2007) Airborne particle matter from primarily geologic, non-industrial sources at levels below NAAQS is associated with outpatient visits for asthma and quick-relief medication prescriptions among children less than 20 years old enrolled in Medicaid in Anchorage, Alaska. Environ Res 103:387–404
56. Gauderman J, Avol E, Gilliland F, Vora H, Thomas D et al (2004) The effect of air pollution on lung development from 10 to 18 years of age. NEJM 351:1057–1067
57. Avol E, Gauderman J, Tan S, London S, Peters J (2001) Respiratory effects of relocating to areas of differing air pollution levels. Am J Resp Crit Care Med 164:2067–2072
58. Islam T, Gauderman J, Berhane K, McConnell R, Avol E et al (2007) Relationship between air pollution, lung function and asthma in adolescents. Thorax 62:957–963
59. Goss C, Newsom S, Schildcrout J, Sheppard L, Kaufman J (2004) Effect of ambient air pollution on pulmonary exacerbations and lung function in cystic fibrosis. Am J Resp Crit Care Med 169:816–821
60. Barraza-Villarreal A, Sunyer J, Hernandez-Cadena L, Escamilla-Nunez M, Sienra-Monge J et al (2008) Air pollution, airway

inflammation, and lung function in a cohort study of Mexico city schoolchildren. Environ Health Perspect 116:832–838
61. Grigg J (2009) Particulate matter exposure in children. Proc Am Thorac Soc 6:564–569
62. McCreanor J, Cullinan P, Nieuwenhuijsen M, Stewart-Evans J, Malliarou E et al (2007) Respiratory effects of exposure to diesel traffic in persons with asthma. NEJM 357:2348–2358
63. Lee J, Son J, Cho Y (2007) The adverse effects of fine particle air pollution on respiratory function in the elderly. Sci Total Environ 385:28–36
64. Lagorio S, Forastier F, Pistelli R, Iabarone I, Michelozzi P et al (2006) Air pollution and lung function among susceptible adult subjects: a panel study. Environ Health 5:11
65. Downs S, Schindler C, Liu J, Keidel D, Bayer-Oblesby L et al (2007) Reduced exposure to PM10 and attenuated age-related decline in lung function. NEJM 357:2338–2347
66. Gent J, Triche E, Holford T, Belanger K, Bracken M et al (2003) Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. JAMA 290(14):1859–1867
67. Karr C, Lumley T, Achreuder A, Davis R, Larson T et al (2006) Effects of subchronic and chronic exposure to ambient air pollutants on infant bronchiolitis. Am J Epidemiol 165(5):553–560
68. Strickland M, Darrow L, Klein M, Flanders W, Sarnat J et al (2010) Short-term associations between ambient air pollutants and pediatric asthma emergency department visits. Am J Respir Crit Care Med 182:307–316
69. Lin M, Stieb D, Chen Y (2005) Coarse particulate matter and hospitalization for respiratory infections in children younger than 15 years in Toronto: a case crossover analysis. Pediatrics 116: e235–e240
70. Zanobetti A, Franklin M, Koutrakis P, Schwartz J (2009) Fine particulate air pollution and its components in association with cause-specific emergency admissions. Environ Health 8:58
71. Medina-Ramon M, Zanobetti A, Schwartz J (2006) The effect of ozone and PM10 on hospital admissions for pneumonia and chronic obstructive pulmonary disease: a national multicity study. Am J Epidemiol 163(6):579–588
72. Arena V, Mazumdar S, Zborowski J, Talbott E, He S et al (2006) A retrospective investigation of PM10 in ambient air and cardiopulmonary hospital admissions in Allegheny County, Pennsylvania: 1995–2000. J Occup Environ Med 48(1):38–47
73. Peng R, Bell M, Geyh A, McDermott A, Zeger S et al (2009) Emergency admissions for cardiovascular and respiratory diseases and the chemical composition of fine particle air pollution. Environ Health Perspect 117:957–963
74. Bell M, Ebisu K, Peng R, Samet J, Dominici F (2009) Hospital admissions and chemical composition of fine particle air pollution. Am J Respir Crit Care Med 179:1115–1120
75. Colais P, Serinelli M, Faustini A, Stafoggia M, Randi G et al (2009) Air pollution and urgent hospital admissions in nine Italian cities. Results of the EpiAir Project. Epidemiol Prev 33(6 Sup 1):77–94
76. Yi O, Hong Y, Kim H (2010) Seasonal effect of PM10 concentrations on mortality and morbidity in Seoul, Korea: a temperature-matched case-crossover analysis. Environ Res 110:89–95
77. McGowan J, Hider P, Chacko E, Town G (2002) Particulate air pollution and hospital admissions in Christchurch, New Zealand. Aust N Z J Public Health 26:23–29
78. Zeka A, Zanobetti A, Schwartz J (2005) Short term effects of particulate matter on cause specific mortality: effects of lags and modification by city characteristics. Occup Environ Med 62:718–725
79. Zanobetti A, Schwartz J (2009) The effect of fine and coarse particulate air pollution on mortality: a national analysis. Environ Health Perspect 117:898–903
80. Ostro B, Broadwin R, Green S, Feng W, Lipsett M (2006) Fine particulate air pollution and mortality in nine California counties: results from CALFINE. Environ Health Perspect 114:29–33
81. Naess O, Nafstad P, Aamodt G, Claussen B, Rosland P (2007) Relation between concentration of air pollution and cause-specific mortality: four-year exposures to nitrogen dioxide and particulate matter pollutants in 470 neighborhoods in Oslo, Norway. Am J Epidemiol 165(4):435–443
82. Stafoggia M, Faustini A, Rognoni M, Tessari R, Cadum E et al (2009) Air pollution and mortality in ten Italian cities. Epidemiol Prev 33(6):65–76
83. Wong C, Vichit-Vadakan N, Kan H, Qian Z (2008) Public health and air pollution in Asia (PAPA): a multicity study of short-term effects of air pollution on mortality. Environ Health Perspect 116:1195–1202
84. Analitis A, Katsouyanni K, Dimakopoulou K, Samoli E, Nikoloulopoulos A (2006) Short-term effects of ambient particles on cardiovascular and respiratory mortality. Epidemiology 17 (2):230–233
85. Conceicao G, Miraglia S, Kishi H, Saldiva P, Singer J (2001) Air pollution and child mortality: a time-series study in Sao Paulo, Brazil. Environ Health Perspect 109(3):347–350
86. Calabrò P, Golia E, Yeh E (2011) Role of C-reactive protein in acute myocardial infarction and stroke: possible therapeutic approaches. Curr Pharm Biotechnol (in press)
87. Wellenius G, Schwartz J, Mittleman M (2005) Air pollution and hospital admissions for ischemic and hemorrhagic stroke among Medicare beneficiaries. Stroke 36(12):2549–2553
88. O'Donnel M, Fang J, Mittleman M, Kapral M, Wellenius G et al (2011) Fine particulate air pollution (PM2.5) and the risk of acute ischemic stroke. Epidemiology 22(3):422–431
89. Villeneuve P, Chen L, Stieb D, Rowe B (2006) Associations between outdoor air pollution and emergency department visits for stroke in Edmonton, Canada. Eur J Epidemiology 21(9):689–700
90. Henrotin J, Besancenot J, Bejot Y, Giroud M (2007) Short-term effects of ozone air pollution on ischaemic stroke occurrence: a case-crossover analysis from a 10-year population-based study in Dijon, France. Occup Environ Med 64(7):439–445
91. Hales S, Blakely T, Woodward A (2010) Air pollution and mortality in New Zealand: cohort study. J Epidemiol Community Health. doi:10.1136/jech.2010.112490
92. Samoli E, Nastos PT, Paliatsos AG, Katsouyanni K, Priftis KN (2011) Acute effects of air pollution on pediatric asthma exacerbation: evidence of association and effect modification. Environ Res 111(3):418–424
93. Peng RD, Chang HH, Bell ML, McDermott A, Zeger SL et al (2008) Coarse particulate matter air pollution and hospital admissions for cardiovascular and respiratory diseases among Medicare patients. JAMA 299(18):2172–2179
94. Ostro B, Roth L, Malig B, Marty M (2009) The effects of fine particle components on respiratory hospital admission in children. Environ Health Perspect 117(3):475–480
95. Anonymous. Indoor air home. United States Environmental Protection Agency. 21 Nov 2011. http://www.epa.gov/iaq/. Accessed on 25 Nov 2011
96. Anonymous. Air quality guide for particle pollution. United States Environmental Protection Agency. August 2003. http://www.epa.gov/airnow/airqualityguideparticles.pdf. Accessed on 25 Nov 2011

Sent: Sunday, August 8, 2021 11:19 AM To: tsyzek@thesullivangroup.com <tsyzek@thesullivangroup.com> Subject: gravel pits Dear Dr. Syzek, I recently volunteered with a neighborhood group in Milliken, Co to do some research on the health effects caused by gravel mines. I found the article published in the Salida Daily Post published in April of 2017 which you wrote. Yesterday we had an informational meeting attended by neighbors opposed to this gravel mine for various reasons. It is directly across a stream (to the south) from a golf course, and a good sized subdivision. there are beautiful wetlands on this property, and it is a path used daily by blue herons, sand cranes, deer and even a moose. The Sand and gravel company claims they will use back roads and "only" run 60 to 90 trips a week on a narrow dirt road that backs up directly on the west to the subdivision and golf course. They also claimed they would be here for 10 years and then told us there would be another 10 on the other side of the Big Thompson River which runs through. After reading your article I am more alarmed than ever. One of the co-owners of this gravel company attended the meeting and the red flags are now overwhelming me. There is a school close to this property, the river runs through it and a large subdivision is within 1/4 mile of the proposed dig. I am looking for any information I can find about what silica dust in particular (not to mention diesel fuel and road dust) could do to my neighborhood. We are already overwhelmed by oil and gas trucks jamming the main road running north to Highway 34 and the north part of the county. It looks as though we will have a fight on our hands since Weld County almost routinely approves these requests. We have children, old people and veterans with health problems related to their service, and many others living here who would be adversely affected. I would so appreciate any information you could give me related to this issue, and what the outcome was of your fight, as it may be our only hope. Thank you in advance for any suggestions you make. Sheila Grotzky, resident of the Mad Russian Subdivision in Milliken Colorado. You can also reach me by phone at 970-587-6408 or at 115 Birdie Dr, Milliken, CO 80543

Why put a Gravel Pit / mining operation that creates a known carcinogen (cancer-causing) right next to homes and a school?

You won't find the answer to that here, or anywhere else for that matter. It's a bad and lethal idea.

What's in the dust created by a Gravel Pit operation?

It is not the same as the dust created by farming or other periodic natural events. The killer is the fine particles of dust you cannot see. The mining and crushing of gravel creates and releases fine particulate matter called Crystalline Silica into the air which will be carried by the wind towards homes and schools. These dangerous particles will permeate homes, neighborhood parks, schools, and playgrounds.

Adults and vulnerable children and seniors will be exposed to this harmful carcinogen every day, all day. Why the City and County would **CHOOSE** to allow the creation of a toxic environment for our neighborhoods and these neighborhood schools when they do, *in fact,* have state and local government statutory and federal regulatory authority, and Texas Attorney General and Supreme Court ruling precedence to use their authority to deny the permit in order to protect public health, safety, economic development, and quality of life is inexcusable, incomprehensible, and UNACCEPTABLE.

So what's wrong with Crystalline Silica?

Crystalline Silica, a known carcinogen (cancer causing agent) which has been found to cause lung cancer, silicosis, and other health hazards!

SOME FACTS:

- Some of the Crystalline Silica can be of the most dangerous variety with a designation as a PM2.5 particle. T hose are particles that measure less than 2.5 micro meters in size
- Once these tiny particles enter the lung *they stay there*. The body's natural defense encapsulates them *causing permanent lung damage or cancer*.
- Winds can carry these fine particles over great distances.
- The closer you are to the source, the higher the concentration and danger
- Health effects can range from Silicosis, lung cancer, tuberculosis increased lung irritation
- There is no cure for silicosis
- Once these fine particles enter the lungs, the body has no means to expel them
- Crystalline Silica clings to inanimate objects like homes, outdoor and playground equipment, trees, plants, and grass and vehicles / cars, so you and your families will come into contact with it.
- Crystalline Silica will infiltrate home and schools' heating and cooling system and there is no viable way to stop it or mitigate it.
- The dust is cumulative; each day over the 20 or more years the pit is in operation more and more of this hazardous dust will accumulate inside and around homes and the schools.
- Our neighborhood homes and the new middle school is adjacent to and/or sits downwind of the proposed pit and its loading and hauling facilities
- They don't call this "Wind Country" for nothing. Most days of the year the wind speed exceeds 15 mph and is often much, much higher in our neighborhoods.

Why put a Gravel Pit / mining operation that creates a known carcinogen (cancer-causing) right next to homes and a school?

Below are some links and excerpts from articles that address this serious hazard.

http://www.osha.gov/OshDoc/data_General_Facts/crystalline-factsheet.pdf

What is crystalline silica?

Crystalline silica is a basic component of soil, sand, granite, and many other minerals.

Quartz is the most common form of crystalline silica. And we are NOT talking countertop grade. Cristobalite and tridymite are two other forms of crystalline silica. **All three forms may become respirable size fine particles when workers chip, cut, drill, or grind objects that contain crystalline silica.**

What are the hazards of crystalline silica?

Silica exposure remains a serious threat to nearly 2 million U.S. workers, including more than 100,000 workers in high risk jobs such as abrasive blasting, foundry work, stonecutting, rock drilling, **quarry work** and tunneling. The seriousness of the health hazards associated with silica exposure is demonstrated by the fatalities and disabling illnesses that continue to occur in sandblasters and rockdrillers. **Crystalline silica has been classified as a human lung carcinogen. Additionally, breathing crystalline silica dust can cause silicosis, which in severe cases can be disabling, or even fatal. The respirable silica dust enters the lungs and causes the formation of scar tissue, thus reducing the lungs' ability to take in oxygen. There is no cure for silicosis. Since silicosis affects lung function, it makes one more susceptible to lung infections like tuberculosis.**

http://www.airinfonow.com/html/ed_particulate.html

Particles can come in almost any shape or size, and can be solid particles or liquid droplets. We divide particles into two major groups. These groups differ in many ways. One of the differences is size, we call the bigger particles PM10 and we call the smaller particles PM2.5.

BIG. The big particles are between 2.5 and 10 micrometers (from about 25 to 100 times thinner than a human hair). These particles are called PM10 (we say "P M ten", which stands for Particulate Matter up to 10 micrometers in size). These particles cause less severe health effects.

SMALL. The small particles are smaller than 2.5 micrometers (100 times thinner than a human hair). These particles are called PM2.5 (we say "P M two point five", as in Particulate Matter up to 2.5 micrometers in size).

The smaller particles are lighter and they stay in the air longer and travel farther. PM10 (big) particles can stay in the air for minutes or hours while PM2.5 (small) particles can stay in the air for days or weeks.

And travel?

- **PM10 particles can travel *as little as a hundred yards* or *as much as 30 miles.***
- **PM2.5 particles *go even farther; many hundreds of miles.***


HWY 257
RAIL ROAD
RIVER
SITE 1
SITE 2
SITE 3
SITE 4
SITE 5