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Health and Developmental Effects of Lead Exposure

In an article in *Science* magazine Doctor Bruce Lanphear writes that about one child in twenty in the U.S. has toxic levels of lead in their blood¹. High blood lead; that is, greater than 70 micrograms per deciliter of blood, can result in serious disease, such as mental retardation, seizures, cerebral palsy, blindness, or death. Lower levels impair children's development, impair hearing and increase an individual's risk of heart attack and stroke in adulthood.

Sources of lead in the human environment have been gasoline, lead-solder in canned foods and plumbing, batteries, and lead-based paints. Although lead-based paints — paints with greater than 0.06 parts per million of lead — were eliminated in 1978, it is estimated that over 10 million occupied houses in the United States contain non-intact lead paint. Lead-based paints are not a hazard if the paint is kept in good condition, but poorly maintained surfaces shed lead-contaminated chips and dust. Toddlers and young children who live in older, poorly maintained housing or older housing undergoing renovation are at the highest risk of exposure to lead hazards. We spoke with Dr. Lanphear about what should be done to reduce children's exposure to lead.

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ER: Dr. Lanphear, what is your training?

BL: I'm a physician trained in public health and preventive medicine with additional training in general pediatric research. I am a faculty member of the Division of General and Community Pediatrics at Children's Hospital and Medical Center in Cincinnati. I conduct research to prevent specific types of problems like lead poisoning and asthma. Most of my research focuses on understanding, controlling, or preventing common problems among children, mostly those who are under-served

or live in poverty; I am also trying to understand why there has been an increase in ear infections. I direct a fellowship training program to train pediatricians to conduct research on common problems in children.

ER: In your *Science* article you state that one in twenty children in the United States are affected by lead.

BL: A national survey conducted from 1988 to 1994 found that 4.7 percent of children in the United States were estimated to have blood lead levels exceeding 10 micrograms per deciliter. This is remarkably low compared with two decades ago, but since there is no biological purpose for lead in humans, there is no normal level of lead in blood.

We typically measure the amount of lead in our bodies by testing a sample of blood. Lead that accumulates in our bodies is largely due to industrialization: the addition of lead to paint, gasoline, and lead solder used in canned foods and plumbing. Lead can also be found in a variety of other sources such as pottery or folk medicines; occasionally, there are reports of lead in imported items, such as crayons or mini-blinds.

There has been a dramatic decline in blood lead levels in U.S. children. Twenty years ago 88 percent of children had blood lead levels over 10 micrograms per

deciliter of blood, and now it's only 5 percent. Much of that reduction was unintentional; that is, lead was removed from gasoline not to protect children's health, nor to prevent cardiovascular disease in adults, but because it was poisonous to catalytic converters in car exhaust systems. Subsequently there was recognition of the health effects by the EPA and leaded gasoline was completely phased out in the United States on December 31, 1996.

ER: Didn't we know that lead was poison?

BL: It was recognized during the Roman Empire that lead was toxic. In fact historians have speculated that one of the reasons for the decline in the Roman

Empire was widespread lead poisoning. The Romans, particularly the ruling class, used lead-lined goblets for their wine; the Romans would process wine in lead vats because it gave it a sweet flavor. Plumbing was constructed from lead, the aqueducts that transported drinking water were often lined with lead, hence drinking water was frequently contaminated, and the Romans often used lead-cooking vessels. So lead exposure was widespread.

In France and Germany in the 17th and 18th centuries, it was punishable by death to adulterate a stale wine with lead to make it palatable. So we've known for a long time that lead is toxic. But the

distinct form of poisoning in children was not identified until the 1890s in Queensland, Australia, shortly after the introduction of lead in paint. Two pediatricians, J. Lockhart Gibson and A.J. Turner, ultimately showed that lead poisoning was due to lead contaminated paint that was inadvertently picked up by children and ingested by normal mouthing behaviors, such as nail biting. Many of those children died or were paralyzed or became blind.

So we've actually known how to prevent lead poisoning since 1904. There's a quote by A.J. Turner in 1908, "Lead poisoning prevention

In the United States, residential lead hazards are currently the major source of lead intake for children.

is easy. Don't use lead paint where children play." Yet even in Queensland, where there were eventually over 200 children afflicted with lead poisoning — about twenty succumbed each year — it took them another eighteen years to ban it once they recognized that paint was the predominant source. In the United States, it took another fifty years to regulate lead in paint. In the interim, millions of children were affected or died from lead poisoning.

ER: What blood lead levels do you have to have to get that sick?

BL: To develop plumbism, or lead poisoning — which typically presents with abdominal colic, seizures, paralysis, blindness or a coma — a child's blood lead level usually exceeds 70 or 80 micrograms per deciliter. But assays for lead in blood were not available at the turn of the century. Instead, physicians relied on signs such as lead lines — a dark blue, lacy collar surrounding the neck of one or more teeth. They also had to rely on the clinical presentation. Children tend to develop paralysis in the lower extremities rather than the hands, whereas adults' hands were usually affected.

Presumably paralysis affected the fatigue muscles. So for example in the United States there was a report of cabaret dancers who became poisoned from moonshine

and their legs were affected because they were dancers. But most workers who were exposed to lead developed palsy or a wrist drop, which was atypical for children. Children with lead poisoning developed paralysis of the legs, like cabaret dancers.

ER: What's the physiological mechanism of lead poisoning?

BL: Nobody knows the mechanism for lead's neurotoxic effects. It is a mystery. It may be that lead causes damage by mimicking calcium; calcium is involved in virtually every bodily function. Nonetheless, because we don't know the specific

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mechanism, some scientists find it easy to dismiss the subtle effects of subclinical lead toxicity. And by the way, in virtually every organ system that's been studied, scientists have found adverse effects from lead.

ER: You mentioned cardiovascular disease in adults. Is that one of the diseases that lead causes?

BL: A number of studies have implicated lead with cardiovascular disease. Joel Schwartz, an epidemiologist at Harvard, estimated that if the population mean blood lead level in adults were reduced by 1 microgram per deciliter, there would be 635,000 fewer people with high blood pressure in the U.S. and 3,200 fewer heart attacks, 1,300 fewer strokes, and 3,300 fewer deaths every year.

ER: The immediate effect with children is developmental problems, but what does lead do to children as they grow into adults?

BL: There are four studies that have followed children from birth until later childhood or adolescence. These children were tested anywhere from seven to eleven years of age. Each of these studies has demonstrated that there are persistent effects of lead exposure in early childhood — both in development and behavior. A seminal study by Herbert Needleman and others reported a dramatic increase in rates of school failure and high school dropout for children with higher tooth lead levels. There is another study showing a 70 percent increase

in death from heart disease fifty years later for individuals who had been hospitalized as children for lead poisoning.

ER: Is that where the cardiovascular disease that you mentioned was described?

BL: There are actually quite a few studies using large national data sets or large cohort studies showing the relationship of lead exposure — as measured by blood lead levels — and high blood pressure. There is a dose response: as blood lead levels

Nobody knows the mechanism for lead's neurotoxic effects ... in virtually every organ system that's been studied, scientists have found adverse effects from lead.

increase, there is a corresponding increase in blood pressure. These and other studies have been used to come to the conclusion that lead is an important contributor to cardiovascular disease.

ER: At the end of your article you compare the lead industry to the cigarette industry. Is lead on that scale as a public health problem?

BL: Lead exposure does not currently cause as much disease and disability as does tobacco. But the weight of the evidence suggests that exposure to lead does contribute to an increase in cardiovascular disease, kidney disease, developmental delay, growth delay and behavioral problems, to name only a few. Since the decline in blood lead levels, lead

probably contributes less now that it did twenty years ago. Still, the detrimental effects of lead are more tragic than the diseases caused by tobacco because we knew leaded paint and gasoline were toxic, and yet they were still introduced.

Alice Hamilton, a leading scientist argued against adding lead to gasoline in 1925 saying, "If you can't control lead in the factories, how are you going to control it when you disperse it across the entire country?" The lead industry fought incessantly to keep lead in paint and to add lead to gasoline. In 1922, the State of Massachusetts banned the use of lead paint, but the lead industry was able to repeal that legislation. In 1984 Werner Mayer, the president of the Lead Industry Association, con-

fessed, "Our victories have been in the implementation of legislation to defer regulations." So we have long known that lead was toxic, and yet it was introduced on a wide-scale basis.

In contrast, it wasn't until the release of the U.S. Surgeon General's Report in 1964 that there was convincing data to implicate tobacco as a risk factor for diseases such as lung cancer. The impact of tobacco on health is certainly more profound — it is the major cause of premature death in this country.

ER: How are kids exposed to lead?

BL: In the United States, residential lead hazards are currently the major source of lead intake for children. Children are exposed to these

hazards through a variety of mechanisms. Early on, before children start to crawl, lead in water is a particularly important source of lead intake. Ingestion of formula is often a primary route of exposure to their environment. Subsequently, as children become mobile, but retain their tendency to explore their world by mouthing it, they are exposed to lead by ingestion of paint chips or, more commonly, lead-contaminated dust. Initially dust on floors is a problem, but dust on window sills becomes an important source after children begin to stand and walk. Finally, beginning in the second year of life, playing outdoors on porches and putting soil in their mouths is another source of lead intake.

ER: Is the dust on the windowsills from contaminated soil outside, or is it leaching out from the paint?

BL: Both internal and external sources of lead contribute to dust on window-sills. Most of it however, is probably from the paint. I'll try to answer your question indirectly. We did a study to examine racial disparity in children's blood lead levels. It was recognized thirty or more years ago that black children had higher rates of lead poisoning than did white children. We found that black children, who live in housing with the same amount of leaded paint, water, and soil, had blood lead levels that were twice as high as white children did. Black children, who

were more likely to live in rental housing, were exposed to poorer conditions. The paint was deteriorating and lead contamination of floor dust and window-sill dust was significantly higher compared with white children's houses. Yet the levels of lead in paint and soil around their houses were no different.

The lead is accessible in rental housing because the paint is often in poor condition and it's peeling, cracked, and disintegrating, thus contaminating house dust. From this study, I infer that the lead contaminated dust on window sills, which is significantly higher in black children's homes, was primarily

from leaded paint, since the soil lead levels were similar for both groups of children.

ER: Is it better to remove lead-based paint from your home or to paint over it?

BL: It is not entirely clear how to safely and effectively remove or contain leaded paint. There has been some progress in understanding how

to remove leaded paint, but there are no studies that demonstrate if lead hazard controls — types of lead abatement — are beneficial or safe for children with blood lead levels less than 25 micrograms per deciliter of blood.

For houses in good condition, it is probably safer and less costly to maintain the condition of painted surfaces. Extensive cleaning and dust sampling should follow any renovation or painting in older housing. For housing that is in poor condition and contains a lead hazard, repairing deteriorated surfaces and doing extensive clean up is essential. What is controversial, however, is how clean is adequate. I am convinced

that after any renovation or lead abatement, it is critical to clean floors to attain dust lead levels below 10 micrograms per square foot.

I live in a house built in 1911 that contains

leaded paint and I have three young children, the youngest is eleven months old. But I don't worry about lead because the paint is in great shape. I haven't even tested the lead in dust, because the paint is in good shape. It has never gone through a major renovation. If I undertook any renovation or extensive painting that requires any scraping, I would make sure that the work area is meticulously cleaned and tested for lead

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levels in dust. I would also keep my children away from such work.

On the other hand, rental housing built in the same era, with the same amount of leaded paint, tends not to be as well maintained. And when you have poorly maintained housing, one needs to make sure that lead is not accessible. This can best be accomplished by dust sampling and evaluating the condition of painted surfaces. A visual inspection is not always adequate. If a major painting and renovation job was recently conducted, the paint condition might appear excellent, but the levels of lead in the dust may be exceedingly high.

ER: Some kids have been found to have higher blood lead levels after abatement.

BL: Right. Throughout the 1980s there were reports that lead levels in some children's blood increased after their homes were abated. It was thought that the increase was due to certain types of lead removal, such as dry sanding. Sanding probably did disseminate lead in house dust more efficiently than other methods, but we tend to overlook the fact that anything that disrupts the integrity of leaded paint can result in lead contamination.

So post-abatement clearance tests or standards were needed to make sure the house was safe for occupancy. Subsequently, in 1988, the Department of Housing and Urban Development promulgated post-abatement lead standards. These standards, however, were based on limited scientific data. HUD adopted the State of Maryland's standards,

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which were based upon what was thought to be feasible to achieve. Furthermore, these standards were developed when blood lead levels of 25 micrograms per deciliter of blood or higher were considered elevated, not 10 micrograms per deciliter as it is today. The downside of these post-abatement standards is that they created this illusion that any level of lead below those standards — they were originally set at 800 micrograms per square foot for window sills, 500 for entry window sills, and 200 for floors — were safe.

Now the EPA has proposed a health-based standard for floors of 50 micrograms per square foot, and some people are saying, "That's awfully low — it cannot be achieved". Yet data from a 1989 national survey indicates that the median level of floor lead loading is about 5 micrograms per square foot; not 50 micrograms per square foot.

The EPA has said that if 1 to 5 percent of children have a blood lead level exceeding 10 micrograms per deciliter, that is consistent with posing a threat. Yet EPA has proposed a standard for dust on floors of 50 micrograms per square foot.

We found in a pooled analysis of almost 1,300 children from twelve different lead exposed cohorts, that at 5 micrograms per square foot, 20 percent of the children are estimated

to have a blood lead level exceeding 10 micrograms per deciliter. Thus, there is a clear mismatch between the proposed standards and the estimated proportion of children with an elevated blood lead level. In short, the EPA has relied on a number of assumptions that are not supported by scientific data.

ER: Is there any place in the world that is pristine that you can use as a background to find out what is a normal level of lead in people?

BL: We know that the background virgin soil — virgin meaning non-industrial — ranges from about 10 to 80 parts per million (ppm). The median soil lead levels around the houses of 205 children in Rochester, N.Y. was 1,000 ppm, which is as high or higher than many lead smelter communities or Superfund sites in the U.S.

ER: Is 1,000 ppm what the average urban dweller is being exposed to?

BL: Well, that level is in an older city in the Northeastern United States. If you sampled soil around housing in western communities that are newer, for example, the lead levels will be quite a bit lower. The Northeast contains children with the highest prevalence of subclinical lead

toxicity or elevated blood lead levels. The prevalence of children with undue lead exposure that live in older cities in the Midwest and the Southeast are also elevated compared with other regions of the U.S.

The question that I think you're alluding to is, Could you then compare the developmental status of children with low levels of lead exposure from certain parts of the country or world, such as Nepal, and children in the Northeastern U.S.?

ER: That's right.

BL: You really can't because there are so many differences besides just lead exposure. For example, children in Nepal probably are relatively undernourished compared to many of the children in the United States. Iron deficiency, maternal intelligence, socioeconomic status, and a whole host of other factors contribute to developmental status. And that's been one of the major problems with trying to make a convincing argument that lead has detrimental effects. Children who have higher levels of blood lead tend to have other strikes against them: they tend to live in the inner city and have poorer nutrition than children who don't have lead exposure. And yet a study from Boston actually showed that the detrimental effects of lead

were greater among a middle class white population than among impoverished African American children in another study.

It is estimated from an analysis of all childhood lead studies that for an increase in blood lead levels from 10 micrograms per deciliter to 20 micrograms per deciliter, there is a 2.5 percent decline in IQ. The effect was lower — 1.3 percent — in the African American population compared with the more affluent population 5.8 percent decline.

ER: IQ measurements tend to be not very precise.

BL: The image I used in the article to illustrate our current approach to the prevention of childhood lead poisoning was the canary in the mine. Miners would send a canary in a cage down the mineshaft, if it stopped whistling or died, the miners would know there were toxic gases present. That is currently the way we deal with residential lead hazards. We let children occupy a house or apartment and then test their blood. If we find that they have an elevated blood lead level, then we know there is a high likelihood that lead hazards are present. That's considered secondary prevention.

Let me digress to define the three levels of prevention. In primary prevention — vaccination is a classic example — you vaccinate somebody before they develop measles or before they are even exposed to somebody that has measles. In the case of lead poisoning, we

could screen houses to identify lead hazards prior to occupancy or after renovation and lead abatement. Dust testing is now done after abatement, but if the dust clearance tests are not set low enough to protect children, they are of limited benefit. Indeed, as I already mentioned, we will cause some children's blood lead levels to increase.

A classic example of secondary prevention is screening an individual for high blood pressure. Once identified, you take certain measures

Miners would send a canary in a cage down a mineshaft and if it stopped whistling or died the miners would know there were toxic gases there. That is currently the way we deal with residential lead hazards. We let children occupy a house or apartment and then test their blood. If we find they have an elevated blood lead level, then we know there is a high likelihood that lead hazards are present.

BL: Measuring IQ is fairly precise. But it is not entirely clear what we're measuring because IQ is a rather global or crude measure of cognition. Still, because IQ is so well standardized, it's actually a fairly useful measure.

ER: Your *Science* article argued for a more preemptive approach rather than measuring kids' blood after they've been exposed.

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or medications to try to delay the onset of a heart attack or other kinds of cardiovascular disease. For lead poisoning, secondary prevention is when you screen children to identify those with a high blood lead level and attempt to reduce their environmental exposures to lead. Unfortunately, lead hazard controls for children with elevated blood levels, including types of abatement, dust control and calcium supplementation may not be safe or effective. Moreover, there is evidence that the detrimental effects of lead are irreversible or persistent. So identifying children, who are already exposed, is too late.

A classic example of tertiary prevention is after somebody has had a stroke and you provide him or her with physical therapy and assist him or her with their daily activities to try to minimize the impact of the stroke.

Ninety-five percent of our health care dollars goes towards tertiary and secondary prevention; 5 percent or less goes to primary prevention and public health. In the case of lead poisoning, tertiary prevention includes environmental interventions and chelation therapy to reduce the body's burden of lead.

ER: But primary prevention is the most efficient as well as the most humane approach.

BL: In the short term it is easier and less expensive to screen children — to use them as canaries in the coal

mine. It doesn't require costly screening of housing, for example. But in the long term, it is cost effective to prevent lead poisoning by screening housing because undue lead exposure has such a dramatic impact on children's development. The major savings are in increased productivity in adulthood and fewer children needing special education. These estimates, however, underestimate the benefits, because they neglect to include the impact on cardiovascular disease, kidney

General published a report on the adverse effects of tobacco in 1964; it probably took another ten or twenty years for people to recognize the impact of that report. The tobacco industry and the lead industry fought the notion that smoking and lead were harmful.

Lead poisoning was largely confined to inner-city black children, children who have no voice, and so there was little public outcry. Moreover, one cannot stop breathing — inhalation of air contaminated from

gasoline emissions was one source of lead exposure — and you cannot stop children from mouthing things or putting their hands in their mouths. So exposure to lead was involuntary.

ER: We have a short attention span.

The Northeast contains children with the highest prevalence of subclinical lead toxicity or elevated blood lead levels. There are cities and towns that don't have a lead problem... On the other hand, there are cities like Rochester, N.Y. where 35 percent or more of children have an elevated blood lead level. So it's still epidemic in some cities.

disease and behavioral problems, to name only a few.

ER: Prevention of exposure to lead would be the most effective treatment.

BL: Right. And I think the point is worth repeating: if we only discovered that lead was a problem five or ten years ago, it really wouldn't be so criminal. It is the long history of cover-up by the industry that makes this so tragic.

ER: Was it really a cover-up when the effects of lead were so widely known?

BL: You could make the same argument for tobacco. The Surgeon

BL: Yes. How do we eliminate childhood lead exposure and sub-clinical lead toxicity, when we've moved on to asthma and attention deficit disorder as problems? Not to downplay either of those, those are both really important problems, but lead exposure and subclinical lead toxicity have shifted back to being an inner-city problem. It was only during the 1980s when more affluent families perceived that their children also were at risk that there was a loud and sustained outcry about lead poisoning. If we become complacent, we will continue to use impoverished inner-city children, who are predominantly black, as canaries.

I think lead poisoning is one of the most well described environmen-

tal justice problems. Yet the environmental justice community is not actively advocating for these children. It is addressing other, sexier chemicals that may or may not cause problems. We know how bad of a player lead is, it is the quintessential environmental toxin. It limits children's opportunities and it perpetuates racial inequality.

ER: As opposed to using children, specifically poor children, as canaries, what would be the alternative?

BL: Instead of screening children to identify whether they have an elevated blood lead level as our primary strategy, we should conduct targeted screening of houses. Targeted because there are cities and towns in the United States that don't have a lead problem.

On the other hand, there are cities like Rochester, N.Y. where 35 percent or more of children have an elevated blood lead level. So it's still

epidemic in some cities. It would be very cost effective to start screening houses, identify those that have lead hazards, and then begin to test lead hazard controls to make sure that we are truly protecting children.

The good news is there is general consensus that lead is toxic at low levels. All of the major scientific and public health organizations — the American Public Health Association, and the American Academy of Pediatrics — have all said that lead is toxic. And compared to 20 years ago, there is actually quite a bit of attention being given to

lead as a public health issue.

The bad news is we're dealing with lead in a simplistic fashion. Since lead is toxic, it's a good thing to remove it from houses. Unfortunately, we're encountering the same problem with lead that we encountered with asbestos. Namely, sometimes you make the problem worse by disturbing the hazard and contaminating a house or a school.

We must ensure that lead is inaccessible and that we adequately clean up after we've disrupted leaded paint. Unfortunately, there are no rigorous controlled trials showing that paint abatement or some of these other existing types of lead hazard controls are either safe or effective for primary prevention efforts.

We're encountering the same problem with lead that we encountered with asbestos. Namely, sometimes you make the problem worse by disturbing the hazard and contaminating a house or school.

ER: HUD standards aren't strict enough?

BL: HUD's proposed post-abatement standards and EPA's proposed health-based standards are not strict enough to protect the majority of children. If we used existing lead hazard controls, as prescribed by HUD, and required that lead was no longer accessible, as measured by dust clearance testing to achieve levels below 5 or 10 micrograms per square foot on floors, for example, they would probably be effective. But we haven't proven that. As long

as the dust lead levels and other screening tests that we use to identify lead hazards are not protective, many children will develop an elevated blood lead *because* of the lead hazard controls.

We don't know how many houses would fail a standard of 10 micrograms per square foot on floors for example. We have some idea based upon inference from studies, but we really don't know. If it's 5 percent of the houses, we could probably develop a strategy to address housing containing lead hazards. We could tear down the one percent that are just falling apart and find ways to put money towards those 4 percent of houses. But if it's 20 percent, we're going to have some problems because in the

process of trying to address those hazards, we may make housing unaffordable or less available for families.

A key missing ingredient to develop a scientific strategy is to estimate the proportion of houses that would fail a specific standard. HUD is actually in the midst of doing such a survey, so probably by next summer we'll have some information on that. HUD has been doing some stellar work. One wishes they would have tackled residential lead hazards ten or fifteen years ago, but they are doing some excellent work now: they're putting money into national surveys, they are asking some hard questions.

ER: Why did it take so long for HUD to act on lead?

BL: I think part of the problem goes back to the 1970s when there were the two factions within HUD: one group wanted to revitalize cities and the second group wanted to make housing inexpensive and available. Declaring lead poisoning a residential problem suited neither of those two factions. In fact, they both viewed lead as an obstacle to achieving their goals.

Thus, HUD claimed that lead wasn't a housing problem, it was an environmental problem. Meanwhile the EPA was saying lead isn't an environmental problem, it was a housing problem. There was no federal agency willing to take responsibility for lead poisoning caused by residential lead hazards.

ER: What is stopping us from addressing lead poisoning?

BL: There are several hurdles to overcome; cost, identification of proven methods to control residential lead hazards, and promulgation of effective health-based lead standards for house dust and residential soil.

A few years ago California instituted a fee on all current and past uses of lead products like paint, gasoline and batteries. Now there are millions of dollars available every year to address lead poisoning. A similar proposal was introduced in the state of Ohio, but it has languished in someone's office. The disadvantage of this type of fee, like the recent tobacco settlement, is that it shifts the cost to the consumer. The companies that prospered by the negligent use of lead-contaminated products do not share in the economic blame. Similarly, there was a resolution from the American Public

Health Association — proposed by Rick Rabin of Massachusetts — to demand that the industries help cover some of the cost of abatement and other lead hazard control work. That hasn't gone anywhere yet.

One obstacle, similar to the early problems with tobacco litigation, was that you couldn't point to one paint company and say, It was Sherwin Williams that made the paint that poisoned my child. Just as you cannot say it was Marlboro or Kool cigarettes that caused your lung cancer when you smoked three or four brands over a lifetime. But you can bring a suit against the entire industry. If Sherwin Williams manufactured 30 percent of all leaded paint and this company 28 percent, blame them all in proportion to their contribution to lead poisoning. Similarly, one can spread the blame for leaded gasoline across various petroleum companies.

A second part of the strategy is to define the extent of the problem on both a local and national level. For example, we don't really have a sense of what proportion of housing would fail any particular lead standard in Cincinnati, or the United States. How many of our houses contain lead hazards? The first thing to do is conduct a survey and estimate the extent of the problem.

ER: And that takes money.

BL: That takes money. What we can do however, to make this type of survey more interesting to people who do not live in the inner city, is to comprehensively address residential hazards. People who live in the suburbs do care about asthma, many of their children have asthma. The

prevalence of asthma is higher in the inner city, but it's by no means limited to the city. We can also look at other kinds of hazards such as unprotected windows, absence of smoke detectors, accessible drugs or poisons that might be ingested. Residential hazards are responsible for a large proportion of the injuries and illness in young children and many of these hazards have lifelong implications. Asthma, for example, which is often a chronic disease, appears to originate from exposures to settled allergens in early childhood. So you can make the cost of these surveys palatable by demonstrating the impact beyond lead poisoning.

Finally, we have to make sure that the standards to identify hazards are protective, and we need to use proven methods to eliminate lead hazards once they are identified. We still have a long way to go, but all the pieces are in place to prevent childhood lead poisoning.

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The HUD lead website is:
www.hud.gov/lea/leagrant.html

The EPA lead website is:
www.epa.gov/lead

The Danger of Being a Wide-Ranging Predator

Introduction:

In the last three decades wild dogs, *Lycaon pictus*, have vanished from twenty-five of the thirty-nine African countries in which they once lived. Today six countries support populations greater than one hundred animals, and few of those populations are stable or increasing. The total number of wild dogs left in all of Africa is approximately 3,000 individuals, and their rapid decline makes this species one of the most endangered. The major reasons for the decline of wild dogs are loss of habitat and prey, and human persecution.

Wild dogs live in packs of eight to ten adults with their offspring, and hunt savannah animals such as impalas, kudu, wildebeests, and warthogs. Wild dogs weigh about 50 pounds, while their competitors, hyenas and lions, weigh in at 100 and 300 pounds respectively.

Rosie Woodroffe and Joshua Ginsberg observed that small populations of African wild dogs in protected areas were vulnerable to extinction because although the protected areas set aside for them were quite large, whenever the dogs crossed the border, they were almost always killed.

Conservation biologists know that small populations of species are more vulnerable to extinction than larger populations. Random events like a hard winter, or disease might not kill a big population but they can wipe out a small one. However, Woodroffe and Ginsberg in the course of their field work observed that wild dogs were suffering significant mortality when

they got close to or crossed the boundaries of their refuges. They call this increased mortality near the border an edge effect.

To see if this is important to the survival of other endangered species, they compiled extinction data on ten different wide ranging carnivores: African wild dogs; gray wolves in Canada; dhole in India(a kind of wild dog); lions in Africa; tigers in India; snow leopard in India, Nepal, Pakistan; jaguar in Central America; spotted hyenas in East Africa; the California black bear; and grizzly bears in Canada and the Northwestern United States. The collected data show that animals that range widely, large predatory animals at the top of the food web, are most exposed to threats

As we worked on the IUCN action plan it became apparent that wild dogs were fine as long as you could constrain them to a protected area.

on reserve boarders, regardless of their population size. The grizzly bear and the African wild dog both require over 3,500 square kilometers (1,400 square miles) of habitat to support a population, while the California black bear has a critical reserve size of only 36 square kilometers (14 square miles).

Conservation biologists spend a lot of time and money managing small populations for inbreeding and disease, and other internal threats, but if animals are killed whenever they cross the border of their reserves, they are not going to survive long enough for internal threats to be a problem. We spoke with the Joshua Ginsberg and Rosie Woodroffe about their work and its implications for conservation biology¹.

ER: Doctors Ginsberg and Woodroffe, what are your jobs and training?

JG: I received my Ph.D. at Princeton studying the ecology and behavior of Grezy's zebra. My present title is director of the Asia program at the Wildlife Conservation Society based at the Bronx Zoo in New York. I am now managing 114 projects in fifteen countries in Asia for the Wildlife Conservation Society. Previous to that I worked in Thailand on coral reefs and sea turtle conservation, I then spent three and one half years at Oxford and four and one half years at the Institute of Zoology in London where I did postdoctoral research on African wild dogs. I recently published the African wild dogs status survey and conservation action plan with the IUCN. Rosie was the senior author on that and David Macdonald and I were the third and second, respectively.

RW: I'm currently a Research Fellow at the University of Cambridge. I did both of my degrees at the University of Oxford, my doctorate on the ecology and behavior of the European badger. I did a postdoc with Josh on the behavioral ecology of the banded mongooses at the Institute of Zoology in London. Since coauthoring the action plan for wild dogs with Josh I have been doing conservation-oriented work on large carnivores. I also advise the British government on the control of tuberculosis in British badgers, which is a serious local problem.

ER: What is the background for your paper?

RW: I think probably one of the greatest insights of conservation

biology over the last one or two decades has been to understand why small populations are vulnerable to extinction. Conservation biologists got terribly excited about processes like random variation, and who breeds and who doesn't, or what sex ratio of offspring they produce. Theoretical conservation biologists were able to show that random processes like that can make it more

likely that a small population goes extinct. But there's not very good evidence for the vulnerability of small populations as a result of demographic effects, so it's often thought that it's not external factors that are making the animal go extinct, rather it's something that is internal to themselves that makes it likely for them to go extinct.

JG: And conservation has focused on this issue of small populations.

RW: Research money has often been directed towards things like in-vitro fertilization for tigers so that we can maintain the genetic diversity of tiger populations. Losing genetic diversity is considered to be a serious threat to the persistence of tiger populations.

JG: California condors, black-footed ferrets, Florida panthers, North Carolina red wolves, the dusky seaside sparrow, all of these animals were down to a handful of individuals. Several of them have gone extinct in the wild and then been reintroduced. All of the technologies and all of the theoretical focus has been when you get down to that small a population, if you have a bad year for breeding, the species can go under.

RW: That's the theoretical background against which our work was carried out. That was the accepted wisdom if you like. In contrast with that, we were looking at a situation with wild dogs, where it didn't seem as though things like randomly bad years were important in driving wild dogs to extinction. The fact was they were putting their noses over the

population that's headed towards a smaller population and you don't do something about the factors that are causing that decline, you'll never see the small population effects because the population will die out or the species will die out before you can start measuring them.

So if you focus on maintaining these small populations, you'll be looking inside the protected area and you'll be looking for inbreeding problems and you'll be looking at rates of reproduction, and you may well miss the fact that outside the protected area they're getting hammered.

If you want to conserve large carnivores, make a bigger reserve. It works pretty well as a rule of thumb. The question is not whether making a bigger reserve works, but why it works.

borders of protected areas and getting shot. It was because a small population of wild dogs would be living in a small reserve and was therefore spending quite a lot of time outside the reserve that they were more likely to get killed.

JG: We found that processes outside protected areas influence whether a species will persist inside a protected area. You can have wonderful parks with great resources and lots of food, but if they're getting snared and run over and shot at and poisoned outside the protected area, they can't breed fast enough to keep up with that loss.

Inbreeding is not something that happens overnight, it's a process. Random variation in the number of kids you have or even the number of boys and girls you have is a generational process. If you get a declining

ER: Edge effects are extensively studied especially for birds in North America. Why is it a new idea for mammals?

RW: I think that there are two differences, the first of which is the scale of what we are talking about. In the Hwange reserve in Zimbabwe where Josh was working, there's a part which measures 18,000 square kilometers, it's almost as big as Israel, and yet you're seeing a substantial edge effect. So we're talking about edge effect on large tracts of land, where we couldn't have expected

it at that scale. For another thing, we're not talking about unprotected landscape with bits of forest in it, we're talking about edge effects in protected areas where wildlife is meant to be safe.



JG: Right. People have been studying edge effects for twenty years. Usually people had studied reserves and the impact of invasive species moving in on it. Traditionally an edge effect was something like cats coming in from an agricultural landscape and killing ground-nesting birds in the Smokies; or cowbirds moving into forests as roads provided avenues into the forests. In a sense our paper's showing that edge effects can happen outside reserves as well as in them.

RW: The accepted wisdom was that if you were a small population, the primary threat to you was being small. And therefore you have to manage for the random processes which make small populations intrinsically more likely to go extinct, even in the absence of external pressures. We were contrasting that view with what we were seeing with wild dogs where it seemed as though external pressures were much more important in causing local extinctions. So we tried to compare those two competing ideas. Is it external pressures which are causing the extinction of these populations, or was it an intrinsic property of a small population?

For this paper we tried to tease apart which of those two things which could be causing extinction of populations inhabiting small areas. So we collected the existing data on ten species of large carnivore, each in an area where it was primarily restricted to protected areas. And sure enough, all of them disappeared from small reserves and persist in large reserves.

ER: The data for this paper was collected all over the world, but your insight came from your own field work?

JG: I had studied wild dogs for five years in the field and previous to that spent probably another five years just thinking about them. Wild dogs have huge home range areas and the wild dogs I was studying would do fine inside protected areas and then they'd go outside the protected areas and they'd get snared and they'd get run over by cars; in other places they'd contract diseases, or they'd get shot by people. As we worked on the IUCN action plan it became apparent that wild dogs were fine as long as you could constrain them to a protected area. In Kruger National Park in South Africa where they have put a fence around it, they don't find evidence of disease in the wild dog populations in the way they do in many other places.

It was our field-based observation, leading to the insight that external forces were important for controlling the population. After we finished



Photo: Chicago Zool. Soc.

the action plan we got into a discussion about whether this insight is generally true because wild dogs are weird animals: they live in large

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packs, all the males are related, all the females are related, they move over huge areas. Is this just an oddball evolutionary dead end that wild dogs have gotten themselves into, or was there a more general principle to be gleaned from this observation?

RW: The good thing about studying carnivores for this kind of analysis is that the data on where they are and where they used to be are often very good. People tend to remember and write it down if they've seen a tiger or a snow leopard. So we were able to find sets of protected areas where we knew these species had been

present in the past, and then find out in which reserves those species have persisted and where they have disap-

peared. Then we did an analysis which told us how big a reserve you needed to have a certain probability of a population persisting. That turns out to differ enormously between species. It goes from about thirty-six square kilometers for the black bear, up to 4,000 square kilometers for the grizzly bear. So while all of these species are more likely to disappear from small reserves, they differ enormously between species in how small a reserve they can persist in. What's small depends upon what species you are.

JG: Right. Those two examples are good because you've got a bear at either end of the spectrum. The two most closely related species of the ten we selected have very different minimum reserve areas. Why is it that some animals are particularly susceptible to fragmentation and others aren't, even when they are closely related like the grizzly and the black bear? If we were right that mortality that was happening outside the reserve was important in causing extinction of the population, then we would expect that home range size should be important. That is, if an animal ranges widely, then it's more likely to enter that danger zone on the border of the reserve and cross reserve borders. We indeed found that home range size was a better predictor of critical reserve size than population density. That implies that edge effects are more important in causing the extinction of these protected populations than were random effects due to simply being a small population.

ER: I don't see how that follows.

JG: We knew that edge effects were happening. We knew that extinction was occurring. Most people, given the data sets that we had, would say, smaller reserves have fewer carnivores, therefore, the cause of extinctions must be small populations. And our thought was it may be that edge effects are more important than a small population. It's not rocket science. If you want to conserve large carnivores, make a bigger reserve. It works pretty well as a rule of thumb. The question is not whether making a bigger reserve works, but why it works.

If the protected animals are in a small reserve, one way to increase their population size would be to

With the Florida panther for instance, you hear about managing disease, and it being inbred, and all this complicated management, but the single most important cause of mortality for Florida panthers is road accidents.

supplement their food, thereby increasing density, thereby solving your problem. But just increasing your prey density alone isn't going to protect the population. With the Florida panther for instance, you hear about managing disease, and it being inbred and all this complicated management, but the single most important cause of mortality for Florida panthers is road accidents.

I think conservation biologists sometimes get excited about ideas that are biologically interesting. It's more intellectually stimulating to think about how to deal with inbreeding depression than it is to think about road underpasses or getting local

people out to go and pick up snares. Another good way to do conservation is to increase the ability for carnivores to move outside of protected areas. And in fact, that's what we've been doing in much of North America for the recovery of the lynx, for the recovery of black bears. The recovery has happened because large areas are moving out of agriculture, they're reverting to forests and the edge is disappearing. Over the last eighty years New England has gone from being 80 or 90 percent agricultural to 80 or 90 percent forest cover, and black bears are back in spades. That's because they have less edge and they've got fewer threats and they've got more habitat. Another way to say that is, to reduce the probability of

extinction, reduce edge; you can do that by linking up habitat and recovering habitat. In most of the world, unfortunately, we're creating more and more edge.

RW: I think that the spatial scale at which edge effects are occurring

is a surprising fact. There is a recent report about a wild dog reintroduction in Tsavo in Southeastern Kenya, which is a 25,000 square kilometer-reserve. Those dogs were released in the middle of the reserve, they wandered out of it and got killed. They had radio collars on; they traveled many miles the first day and then the biologists lost contact with them. Eventually all the radio collars turned up buried in villages around the reserve because the dogs had wandered out of it.

ER: Could it be that the dogs were put in the wrong place rather than too small a place?

JG: Tsavo does not have a lot of wild dogs in it, they've got more lions and hyenas, and that may have kept them moving. But it's just that when you've got a home range area of 1,000 to 1,500 square kilometers, even with a 25,000 square kilometer block, odds are that somewhere in your wandering you're going to move out of it, and if you do and you're an animal that inspires antipathy, you've got troubles.

One of the funny things in life is people generally don't like dogs, but they do like cats. People write about how regal lions are and what vermin wild dogs are. People I think don't like the fact that canids disembowel their prey. There's also the issue of rabies, and so many European and Neo-Europeans are inclined to shoot the dogs or shoot the foxes or shoot the jackals because they carry rabies.

RW: But in this case the local Masai cattle herders said wild dogs aren't a problem for them. They have developed traditional herding practices which have been developed over millennia to avoid losing their livestock to carnivores. They're very good at coexisting with predators because they've been doing it for such a long time. Most areas where the primary stock are small, sheep and goats rather than cattle, wild dogs have been decimated or even extirpated.

ER: What are the implications of this paper for conservation policy?

RW: I think this work forces us to reevaluate our ideas about what causes extinction. I think if scientists have a

biologically interesting bit of theory they may think they understand what causes extinction of small populations. Then when they see small populations going extinct they may think the theory explains what they're seeing. But I think this bit of an empirical study forces us to go back and rethink what's causing extinctions in other countries as well.

I have been involved recently



Photo: Rob Sclarafo

with some people who work on butterflies, and when they think about which butterfly species disappear more rapidly, they realize the more mobile species need bigger patches of habitat to survive. It has always been assumed that there was just some random small population size which was causing local extinctions of butterflies.

JG: I think the first policy implication is that for the last ten or fifteen years people have been talking about win-win solutions in conservation: you can have your cake and eat it too. Those proposed solutions have frequently involved minimizing the value of protected areas. Many people have said we don't really need protected areas, they're neocolonial, they're

imperialistic, and they're horrible — I say that tongue in cheek. And many people in the conservation world have focused their efforts on community-based conservation, or on extractive use of secondary forest products, or on multiple use of wildlife and wildland resources; they've assumed that you can have it all. However, there have been an increasing number of studies of hunting in tropical forests for instance, that show that if you get more than one or two people per square kilometer, hunting no longer is a stable subsistence use of the land.

Outside protected areas we still need to do buffer management. In fact, our work says quite clearly that we need to do more of that, because if we don't manage buffer zones, if we don't find solutions that allow people to use resources outside protected areas, then we're not going to save predators anyway.

RW: Right, the reason that you have edge problems in the first place is because of there being people outside the reserve. At Tsavo where there are snares immediately outside the reserve border these wild dogs are getting accidentally caught and dying. If you've got poor parks management and what should be a buffer zone outside it full of snares and full of domestic dogs carrying rabies, it doesn't really function as a protected area any more. So it's not enough to set habitat on the side, you have to actively protect it, as well as doing extra management outside the reserve.

JG: Right. And unless you can do that, you'll lose your predators in the reserves. That's our take-home

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