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Bred to Suffer: Unregulated Dog Breeding in India and Its Consequences for Companion-Animal Health - A Review, with a Field Case Study of Canine Distemper

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Abstract: ***India has become one of the fastest-growing companion-animal markets in the world, and this expansion has been accompanied by the rapid and largely unregulated growth of a commercial dog-breeding industry. This paper examines the commercial logic of that industry- the repeated breeding of unfit parents in order to preserve a "pure" phenotype, the maintenance of breeding animals in insanitary conditions, and the sale of unvaccinated puppies separated from their dams prematurely- and argues that these practices generate predictable, and frequently fatal, disease in the animals that reach Indian homes. After outlining the economic and legal context, the paper focuses on the two viral diseases most closely associated with the trade, canine parvovirus (CPV2) and canine distemper virus (CDV), reviewing for each the causative agent, its transmission, its pathogenesis, the clinical presentation, and reported survival rates, drawing on both international veterinary literature and Indian epidemiological studies. It then considers the dermatological disease- most commonly demodicosis with secondary pyoderma- that characteristically accompanies the immunosuppressed puppy, and closes with a first-hand field case study of a single dog that died of canine distemper, in which much of the paper's argument is realised in one animal. The central contention is that these deaths are not random misfortunes but the predictable outcome of a systemically flawed model of production. The paper concludes by considering prevention and policy in the light of the WSAVA vaccination guidelines and India's existing, but largely unenforced, animal-welfare legislation.***

Keywords: unregulated breeding; puppy mills; backyard breeding; canine distemper; canine parvovirus; demodicosis; maternally derived antibodies; vaccination; animal welfare; India

1. Introduction

Across Indian cities, the acquisition of a dog frequently follows a similar course. A family chooses a popular breed- a Labrador Retriever, a German Shepherd, or one of the smaller "toy" breeds valued for their appearance and low cost of upkeep- and obtains a puppy through an online listing or an informal contact for a modest sum. The animal is often unusually quiet, underweight, and younger than it ought to be. Within a few days it ceases to eat; vomiting, haemorrhagic diarrhoea, or a neurological sign such as the twitching of a limb follows, and by the time the animal is brought to a veterinarian little can be done, frequently at considerable expense. Repeated many thousands of times each year, this sequence is not an accident. It is the product of an economy of continuous breeding, insanitary housing, premature separation, and absent vaccination that generates diseased animals as reliably as any other process of production generates its output- one whose costs are borne almost entirely out of the buyer's sight.

The economic incentives behind this pattern are straightforward. As disposable incomes have risen, demand has grown for pedigreed breeds whose foreign appearance confers status, and purchasers typically expect such animals to conform closely to type while costing very little. The supply side has organised itself to satisfy these expectations at the animal's expense: breeders inbreed to fix the desired "pure" appearance, and in doing so concentrate recessive genetic disease; they minimise veterinary care and nutrition; they house large numbers of animals in small, unhygienic enclosures; and they remove puppies from their dams early, in order to accelerate turnover, before the neonatal immune system has matured. Each of these practices carries a biological cost, and it is with that biological cost that the present paper is principally concerned.

Two diseases lie at the centre of this account. The first, canine parvovirus, is a highly virulent, environmentally stable, and readily transmitted virus that attacks the rapidly dividing tissues of the young dog and kills the majority of unvaccinated puppies it infects. The second, canine distemper, is a multisystem viral illness that may begin with signs resembling a mild upper-respiratory infection and progress, weeks or months later, to seizures, paralysis, and death, with no curative treatment available at any stage. Neither disease is obscure; both are well characterised and, importantly, preventable by vaccines that are inexpensive relative to the cost of the animal. Their continued prevalence in India therefore reflects not the intractability of the pathogens but the conditions of the trade. A substantial proportion of the puppies sold through this market succumb to parvovirus or distemper within weeks of purchase- an observation that constitutes the central thesis of this paper, which the sections that follow seek to substantiate.

The paper proceeds in stages. Section 2 documents the current state of unregulated breeding in India- its scale, its cost, the conditions under which it operates, and the legislation nominally intended to constrain it. Section 3 examines the biology of harm: the manner in which inbreeding, a compromised transfer of maternal immunity, and an insanitary environment together predispose an animal to infection even before it encounters a pathogen. Sections 4 and

5 address canine parvovirus and canine distemper in detail, and Section 6 considers the dermatological disease that commonly follows immunosuppression. Section 7 presents a field case study, and Sections 8 and 9 turn to prevention, policy, and conclusions. Throughout, the aim is to draw together elements that are seldom examined side by side- the breeding shed, the biology of the individual animal, the clinical presentation, and the outcome.

2. The Landscape of Unregulated Breeding in India

2.1 The scale and economics of the trade

It is difficult to regulate an industry that officially barely exists. Most dog breeding in India happens outside any register, off the books, and in cash. Estimates of its size are therefore rough, but even the rough figures are striking. Animal-welfare advocates and officials have described a trade running to hundreds of crores of rupees a year- figures in the range of five hundred to a thousand crore have been cited- with essentially none of it taxed and no receipts changing hands. A submission by an animal-welfare organisation to the Law Commission of India described a multi-crore pet trade growing at roughly twenty per cent a year. Whatever the exact number, two features are not in dispute: the market is large, and it is growing fast.

The economic incentives all point in the wrong direction for animal welfare. A breeder's revenue is a function of how many saleable puppies can be produced per year for the lowest possible cost. Veterinary care, good nutrition, clean housing, and adequate rest between litters are all costs. Cutting them raises the margin on each puppy. The market does not punish the breeder for producing a sick animal, because the sickness usually appears only after the sale, once the puppy is in the buyer's home and the money has changed hands. The breeder externalises the cost of disease onto the buyer, onto the veterinarian, and above all onto the dog. This is the core structural problem, and it explains why appeals to breeders' conscience have achieved so little: the system rewards precisely the behaviour that harms the animal.

The same logic drives the fixation on foreign, pedigreed breeds. Imported and purebred dogs fetch higher prices than the indigenous Indian dog- the Indie or pariah dog- even though the Indie is by most accounts hardier, better adapted to the local climate, and less prone to the inherited conditions that plague heavily line-bred foreign breeds. Shelters across the country are full of healthy Indies waiting for homes while buyers pay premiums for fragile imported puppies produced in mills. The "Adopt, Don't Shop" slogan is, underneath the hashtag, an argument about exactly this: that the market's preferences are both cruel and, practically speaking, backwards.

2.2 What "unregulated breeding" actually looks like

The term "puppy mill" has a dictionary definition- an establishment that breeds puppies for sale, typically on an intensive basis and in conditions regarded as inhumane- but the reality on the ground is more specific and, frankly, worse than the phrase suggests. In the typical unregulated operation, breeding females live in near-permanent confinement, often in cramped cages or small pens, with little chance to walk, play, or behave like a dog at all. They are bred on almost every heat cycle. Welfare organisations describe females forced to produce three or four litters a year, far beyond what is safe for the mother's body, and sometimes bred before they have even reached physical maturity. The result is a mother who is chronically exhausted, poorly nourished, and physiologically depleted - and an exhausted, depleted mother produces weak puppies.

When the female can no longer reproduce, she has no further commercial value to the operation, and at that point she is frequently abandoned, dumped at a shelter, or in the worst cases simply killed. This detail matters for understanding the trade's attitude to the animals in its care: the dog is treated as a piece of production equipment with a service life, not as a living creature. Hygiene follows the same economic logic. Housing is often filthy, waste accumulates, and disinfection is minimal, which- as later sections will show- is precisely the environment in which viruses like parvovirus thrive and spread, since the virus can survive for months in a contaminated space.

Layered on top of the physical cruelty is a quieter, genetic one. Breeders fixated on a particular look, or a "pure" bloodline, routinely mate closely related dogs. This inbreeding concentrates recessive disease-causing genes and produces litters carrying known and unknown disorders, some apparent at birth and many surfacing only later in life. The buyer who brings home a purebred puppy is often, unknowingly, bringing home a genetic liability that will not declare itself for months or years. The mechanics are important enough that Section 3 is devoted to them.

2.3 The law on paper, and the gap on the ground

India is not lacking in the law in this case. The basic law is the Prevention of Cruelty to Animals Act, 1960, which is against cruelty of animals and gives power to seize animals in distress. This Act was followed by the Prevention of Cruelty to Animals (Dog Breeding and Marketing) Rules, 2017, which mandate that every dog breeder gets a licence from the State Animal Welfare Board and basic conditions of housing, hygiene, veterinary, and record keeping including practices like unhygienic environments and inbreeding and banning even selling of unweaned puppies. A set of rules, the Pet Shop Rules 2018, imposed similar registration and welfare requirements on the retail end of the chain, meaning that pet shops would have to keep records of the breeders they buy from and the customers they sell to, and be closed if they don't. In theory, then, everything in the preceding section is already against the law. It is illegal not to have a licence when breeding. Inbreeding is not allowed in a mill. It is not permitted to sell unweaned and unvaccinated puppies. It is an offence to keep animals in dirt. It is not that they don't have rules, it is that they have not rules that are enforced. Welfare groups have noted that, years after the Breeding Rules were notified, many states had not yet determined who was supposed to be the Animal Welfare Board in the Rules that they were considering, and that many boards were either inactive or they did not have any website or even an address to contact. In the gap in enforcement, tens of thousands of

unlicensed breeders and pet stores are still doing their business more or less openly. For the animals, the divide between the written law and the lived law is all the law.

For a member of the public who encounters a suspected mill, the formal remedy is to complain to the State Animal Welfare Board, which is meant to send an inspector; an approach to an established welfare organisation can help push the complaint along, and where cruelty is evident the police have powers under the 1960 Act to seize animals in distress. In practice these routes vary enormously from place to place and depend heavily on the persistence of individual activists. The uncomfortable conclusion is that the main check on the trade is at present not the state but the conscience of private citizens- which is to say, almost no check at all.

2.4 Taken too soon: the premature-separation problem

Of all the shortcuts the trade takes, one deserves to be singled out early, because it does so much damage and because it connects directly to the disease biology in later sections. That shortcut is taking puppies away from their mothers far too soon.

The consensus among veterinarians and welfare experts is that a puppy needs to stay with its mother and littermates for a substantial period- commonly cited as around seventy-five to ninety days, and rarely less than the first two months- in order to develop normally, both behaviourally and immunologically. During those weeks the puppy is still nursing, still receiving protective factors through the mother's milk, and still building the social and behavioural foundations it will need as an adult. Mills routinely ignore this. Welfare officers describe puppies separated from the mother as early as ten to fifteen days after birth, so that "extremely weak puppies are introduced to the market as they have had no time to develop immunity". Regulations in other countries, and India's own rules, treat the sale of such unweaned puppies as a violation precisely because everyone in the field understands the harm.

The reason this is so dangerous becomes clear once the immunology is spelled out, which Section 3.3 does in detail. In a nutshell: a newborn puppy is not immune to disease because of its underdeveloped immune system, but rather because of antibodies that he or she has received from the mother, mainly via his or her first milk. That borrowed protection fades over the following weeks, and a carefully-timed vaccination schedule is meant to take over as it does. A puppy pulled from its mother at two weeks and sold unvaccinated into a contaminated market has, in effect, been pushed off a bridge before the far side was built- and everything in the disease sections that follow is a description of what happens next.

3. The Biology of Harm: How Bad Breeding Produces Sick Dogs

Before turning to the individual diseases, it is worth pausing on a question that is often skipped: why should the manner of a dog's breeding affect whether it later develops a viral infection at all? A virus, after all, is a virus; surely any dog can catch parvovirus or distemper regardless of its parentage. The answer is that breeding practices do not usually cause these infections directly, but they load the dice. Inbreeding, immune impairment, a broken maternal-immunity bridge, and a filthy environment each independently raise the probability that a given puppy will (a) be exposed to a pathogen, (b) be unable to fight it off, and (c) suffer a worse outcome if it does fall ill. Put those together and the "random" tragedy on the examination table starts to look statistically inevitable. This section examines the four mechanisms in turn. Figure 1 sets out the whole chain, from the breeding shed to the diseases that follow.

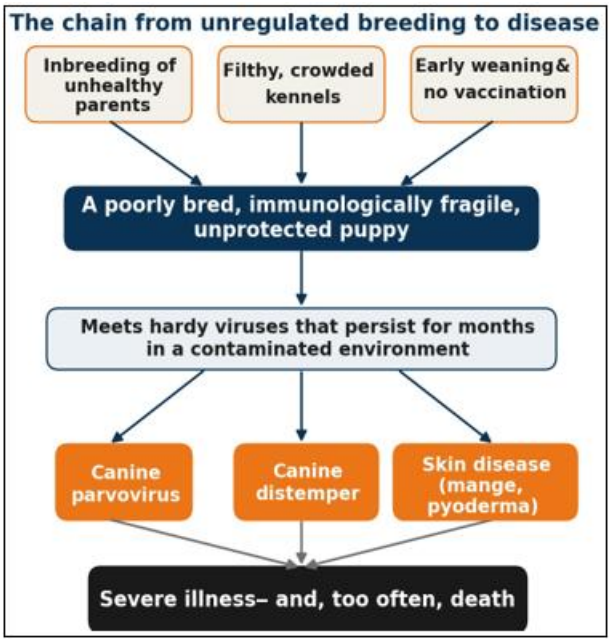

Figure 1: The chain from unregulated breeding to disease: how inbreeding, filthy conditions, and missed vaccination combine to produce a fragile, unprotected puppy that meets- and too often dies from- the diseases examined in this paper.

3.1 Inbreeding and the loss of genetic diversity

All dogs have two sets of each gene, one from the mother and one from the father. Some problemcausing mutations are recessive, which means that a dog has to inherit two copies to be diseased, while a dog with one faulty and one normal copy will be a healthy 'carrier' that will not show signs of the problem but may pass it on. In a genetically diverse population, two carriers of the same rare fault are unlikely to meet and mate together so the disease remains rare. This safety margin is lost when inbreeding occurs. Closely related individuals tend to have the same secret weaknesses, so that their children would have a much greater chance of having two copies of the weaker gene and therefore manifesting the disease. Geneticists quantify this by the coefficient of inbreeding (COI), which is a number that estimates the probability that the two copies of a gene in an individual are identical due to sharing a common ancestor. The essence of this line of thinking was captured in a memorable quip by the founder of this line of thought, Sewall Wright: dominant harmful mutations are eliminated from a population fairly rapidly as they are revealed, and can be selected against, while recessive ones accumulate quietly and inbreeding brings them to the fore. Inbreeding, in other words, is a machine for bringing to the surface just the genes that natural selection had swept under the carpet. It makes two copies of

the same gene, homozygosity, and thus raises the risk of expression of recessive disease. The real limits are important to appreciate the extent to which mill breeding is reckless. At less than about five per cent COI, a population typically maintains good diversity and immune function and general vigour. At about 5-10 per cent COI, the effects are increasingly found, such as smaller litters, weaker puppies, and an increased risk of recessive diseases becoming present. At more than 10 per cent COI, it becomes truly dangerous. To put that in perspective, a 1st cousin marriage would result in roughly a 6.25 per cent COI. To give perspective on that, a 1st cousin marriage would be about 6.25 per cent COI- which is not accepted by many human societies. Yet genotype-based studies of purebred dogs have found average inbreeding levels across breeds that are extraordinarily high by the standards of any wild species, with one large study reporting a mean adjusted inbreeding coefficient of around 0.25 across 227 breeds- a level at which inbreeding depression is firmly expected to occur. Mill breeding, which pairs relatives casually and repeatedly with no pedigree analysis at all, sits at the worst end of this spectrum.

The health consequences are well documented. Higher inbreeding is associated with a measurable increase in morbidity: a large analysis using pet-insurance data found significantly more nonroutine veterinary events in more inbred breeds than in less inbred ones, and close inbreeding specifically harms litter size and the survival of newborns. Beyond raising the odds of any single named disorder, high inbreeding tends to reduce a dog's overall biological robustness- its resilience, its fertility, and, critically for this paper, the competence of its immune system. The next subsection follows that immune thread.

A final, sobering point from population genetics is the concept of the "extinction vortex." When inbreeding drives up puppy mortality and genetic disease, the pool of viable breeding animals shrinks, which forces still more inbreeding, which worsens the problems further- a downward spiral in which a breed, or a closed breeding line, progressively destroys its own health. Mills, which prize a narrow "look" and breed within a small set of related animals, are perfectly designed to fall into this vortex, and to drag the animals in their care down with them.

Table 1: Coefficient of inbreeding (COI): approximate thresholds and expected effects in dogs.

Approx. COI	Genetic situation	Expected effects on the dog
Below ~5%	Ample genetic diversity retained	Strong immune function and general vigour; the target range for responsible breeding.
~5–10%	Diversity narrowing	Harmful effects begin to appear: smaller litters, less robust puppies, rising expression of recessive faults.
~6.25%	Equivalent to a first-cousin mating	A pairing treated as taboo in many human societies, given here for scale.
Above ~10%	High homozygosity	Genuinely dangerous: higher puppy mortality, more genetic disease, risk of an "extinction vortex".
~25% (breed averages seen)	Extremely high by any species' standard	Level at which inbreeding depression is firmly expected; documented as an average across many purebred breeds.

3.2 Inbreeding depression and the compromised immune system

The blanket term for the loss of fitness that follows from inbreeding is "inbreeding depression," and it is not a vague notion but a recognised complex of reproductive and physical problems observed across species. In dogs, research in genetics, immunology, and veterinary medicine has increasingly converged on a specific and worrying finding: high inbreeding is associated with a rise in immune-mediated disease and cancer, and with reduced immune function generally. A dog can be genetically predisposed, in other words, to have an immune system that either underperforms against infection or malfunctions against its own tissues.

Some of the most direct evidence comes from studies of the genome itself. When researchers examine "runs of homozygosity" - long stretches of chromosome where a dog carries identical copies inherited from a common ancestor, the genomic fingerprint of inbreeding- they find that certain biological processes are consistently under-represented in the genetic diversity of heavily inbred dogs. In at least one breed study, "immune response" was among the most strongly depleted categories, a pattern the authors linked to that breed's high prevalence of allergic skin disease. The interpretation is intuitive: the immune system depends on carrying a wide variety of genes, particularly those coding for the molecules that recognise foreign invaders, and inbreeding narrows exactly that variety.

For the purposes of this paper, the significance is direct. A mill-bred puppy is disproportionately likely to be inbred, and an inbred puppy is disproportionately likely to have an immune system that is weaker, narrower, or more prone to malfunction than that of a well-bred dog. When such a puppy meets canine parvovirus or canine distemper, it is meeting them with second-rate defences. The same weakness reappears in Section 6, where it becomes the permissive condition for demodectic mange to run out of control. Genetics is not destiny, but in the mill context it stacks the deck heavily against the animal before any virus is even in the picture.

3.3 The broken bridge: maternal antibodies, colostrum, and the window of susceptibility

If inbreeding is the slow, structural way that bad breeding harms a dog, the destruction of maternal immunity is the fast, immediate way. Understanding it requires a short detour into how a newborn puppy is actually protected, because the whole logic of vaccination- and the whole tragedy of the mill puppy- turns on this mechanism.

A puppy is born with an immune system that works but is inexperienced; it has not yet met the pathogens of the world and has not yet learned to make its own antibodies against them. Nature bridges this gap with borrowed immunity. In the first hours after birth, the mother's initial milk- the colostrum- is rich in antibodies, and the newborn gut is briefly able to absorb these antibodies whole into the bloodstream. These are the "maternally derived antibodies," or MDA, and for the first weeks of life they are the puppy's main shield against diseases

like parvovirus and distemper. It is, quite literally, the mother lending her immune memory to the child.

This borrowed protection has a built-in expiry. Maternally derived antibody declines steadily after birth, falling with a half-life of roughly nine to ten days. This creates a critical and rather cruel window. There comes a period when the level of maternal antibody has dropped too low to actually protect the puppy from infection, yet is still high enough to interfere with, and neutralise, a vaccine given at that moment. During this "window of susceptibility," the puppy is caught in a gap: unprotected by its mother, and not yet protected by vaccination. This gap is not a rare accident; it is a normal feature of puppy immunology, and it is the single most important reason that puppies must be vaccinated on a schedule of several doses rather than one. Figure 2 shows why: as maternal antibody fades, it passes through a level that no longer protects the puppy but still blocks a vaccine, leaving a window in which the animal is defended by neither.

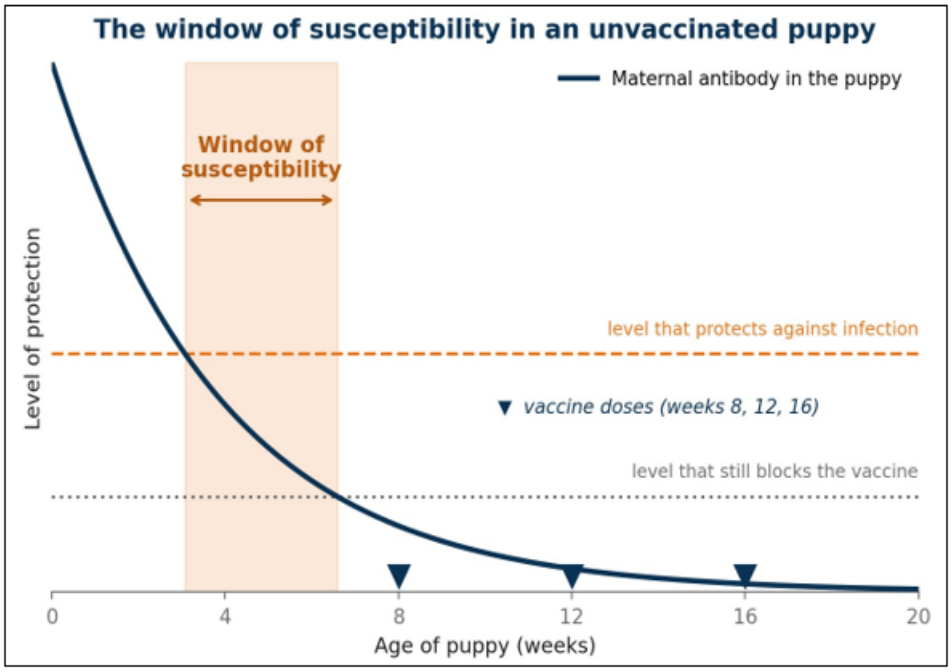

Figure 2: The window of susceptibility. Maternal antibody protects the newborn but fades over the first weeks of life; there is a period in which it is too low to prevent infection yet still high enough to neutralise a vaccine. A series of doses to at least sixteen weeks of age is what closes the gap- and the schedule the mill puppy never receives.

The WSAVA (World Small Animal Veterinary Association) guidelines address this by recommending a series of core-vaccine doses given every two to four weeks through early life, with the crucial final dose delivered at sixteen weeks of age or older- because only by then can veterinarians be confident that any interfering maternal antibody has faded and the vaccine will actually "take". The whole schedule is, in essence, a series of attempts to catch the puppy in the moment after maternal antibody has waned but before a wild virus finds it. The details of that schedule are set out in Section 8.

Now place the mill puppy into this picture. It is separated from its mother at, say, two weeks- cutting short the nursing relationship and everything that comes with it. It is sold unvaccinated, so the carefully-timed replacement schedule never begins. And it is moved through, and sold into, environments heavily contaminated with virus. The puppy therefore enters its window of susceptibility with no vaccination on board and heavy pathogen exposure all around it. The bridge of maternal immunity has been dismantled from the puppy's side, and nothing has been built to replace it. In Indian epidemiological studies of parvovirus, the fingerprints of exactly this failure are visible: infection concentrates overwhelmingly in unvaccinated puppies under six months of age. The window of susceptibility, in the mill puppy, is not a window at all. It is a wide-open door.

3.4 The environment: crowding, filth, stress, and the durability of viruses

The final mechanism is the simplest to grasp and, in some ways, the most damning. Even a genetically sound, well-timed, fully-vaccinated puppy would be endangered by the physical conditions of a typical mill; a weak, unvaccinated one has almost no chance. Overcrowding places many animals in close contact, so any pathogen that enters spreads rapidly from dog to dog. Poor sanitation means that infectious material- faeces above all- accumulates in the environment. And chronic stress, from confinement, noise, and the absence of normal social life, is itself immunosuppressive, further blunting the animals' already-compromised defences.

The durability of the viruses turns these conditions from merely bad into catastrophic. Canine parvovirus is famously hardy: it is a non-enveloped virus, which makes it resistant to many ordinary disinfectants, and it can survive and remain infectious in the environment for something in the region of five to seven months. An infected dog sheds enormous quantities of virus in its faeces- figures on the order of a billion virus particles per gram of stool are cited- and this

contamination lingers, so that fomites (contaminated objects, surfaces, hands, and equipment) and the environment itself become long-lived sources of infection. A single infected puppy passing through a dirty pen can seed that pen for months, ready to infect the next litter that arrives. In a facility that does not disinfect properly and cycles litter after litter through the same spaces, parvovirus can become effectively endemic.

The four mechanisms of this section do not act in isolation; they compound. An inbred puppy (3.1) with a genetically weakened immune system (3.2), stripped of maternal protection and never vaccinated (3.3), living in a crowded, filthy, stressful space saturated with hardy virus (3.4), is not a healthy animal that might get unlucky. It is a sick outcome waiting to be realised. With that framework in place, the next two sections examine what the two headline viruses actually do once they find such a dog.

4. Canine Parvovirus (CPV-2): The First Killer

Canine parvovirus is, for young dogs, one of the most dangerous infectious diseases in the world, and it is the disease that most often claims the mill puppy first. It is worth examining in some detail, both because of its severity and because its story- a virus that emerged, spread globally, mutated, and settled into endemic circulation- illustrates how quickly a pathogen can establish itself in a dog population.

4.1 The virus and its origins

Canine parvovirus type 2 (CPV-2) is the cause of the classic disease. A small, non-enveloped virus that has only one strand of DNA inside an icosahedral protein shell (capsid); the major capsid protein VP2 dictates host range and immune system recognition of the virus. CPV-1 is another, unrelated, minute virus of canines, but is far less important- that is the type of parvovirus that people refer to when they talk about "parvo".

CPV-2 comes with a fascinating history. It didn't exist, as far as anybody can tell, prior to the late 1970s. It is believed to have evolved around 1978 from a feline panleukopenia virus (fPV) (or one closely related to fPV), with a few amino-acid changes in the capsid protein VP2 conferring the ability to infect dogs. Quickly and from a small start, it spread around the whole world in only a few years, leading to outbreaks and high dog mortality rates worldwide. Since then, it has had a long life. Almost all instances of the original CPV-2 have been replaced by a series of antigenic variants of CPV-2 (CPV-2a, CPV-2b, and CPV-2c) with additional VP2 mutations and different geographic distribution. CPV-2a is now the more prevalent type of the virus in India, with CPV-2c being the next most common and the original CPV-2 being rare, as seen in the molecular studies from 2010 to 2023. It is a very persistent disease, partly due to this continuous mutability.

4.2 Transmission and environmental persistence

Parvovirus spreads by the faecal-oral route: a dog is infected by ingesting virus shed in the faeces of an infected animal, directly or- very commonly- through contaminated environments and objects. Two facts magnify this route enormously. Infected dogs excrete huge amounts of virus, around a billion per gram of faeces, starting about 4-5 days after exposure (before they are sick), and for about 10 days after recovery, although this can be longer depending on duration of infection. And the virus is very resilient, remaining active for months and not being killed by many common disinfectants, due to the absence of a fragile lipid envelope that makes killing viruses by disinfectants so easy. It is difficult to keep parvovirus contained in a crowded, poorly-sanitised environment because of heavy shedding, pre-symptomatic transmission and persistence in the environment.

4.3 Pathogenesis: an attack on the fastest-dividing cells

To understand why parvovirus is so lethal to puppies specifically, one has to understand what kind of cells it attacks. Parvoviruses can only replicate in cells that are themselves actively dividing, because they depend on machinery that dividing cells make available. This means the virus homes in on the body's most rapidly proliferating tissues. In a young dog, three tissues fit that description and bear the brunt of the disease.

The first and most important is the lining of the small intestine. The intestinal wall renews itself constantly from proliferating cells housed in structures called crypts, and about four to seven days after exposure the virus localises in these mitotically active crypt cells and destroys them. The consequence is that the intestinal lining collapses: the surface that normally absorbs nutrients and holds back gut bacteria is stripped away. This produces the hallmark of the disease- severe, often bloody (haemorrhagic) diarrhoea- and opens the door for bacteria from the gut to invade the bloodstream.

The second target is the bone marrow and lymphoid tissue, where immune cells are produced. Parvovirus infection of these proliferating tissues causes a sharp fall in white blood cells- leukopenia, and specifically lymphopenia- which cripples the very defences the dog needs to fight the infection off, and worsens its vulnerability to secondary bacterial invasion. The third target, seen mainly in very young puppies whose mothers had no antibodies to pass on, is the heart muscle: the virus can attack the actively dividing cells of the developing myocardium, causing an often-fatal inflammation of the heart (myocarditis). This cardiac form is now uncommon, because most breeding females carry enough parvovirus antibody- from vaccination or past exposure- to protect their newborns in those first weeks, which is itself a quiet argument for the maternalimmunity mechanisms of Section 3.3.

The clinical crisis flows directly from this pathogenesis. The destroyed gut lining causes catastrophic fluid loss and dehydration; the breached gut barrier and collapsed white-cell count let gut bacteria invade the blood, producing sepsis and a body-wide inflammatory response; and death, when it comes, usually results from that combination of dehydration, shock, and overwhelming infection, often within the first two to three days after hospitalisation.

4.4 Clinical signs

The clinical picture of parvoviral enteritis is grimly consistent. Affected dogs- most often puppies between about six weeks and six months of age- show anorexia (refusal to eat), lethargy, and depression, followed by vomiting and the characteristic profuse, foul-smelling, frequently bloody diarrhoea. Fever is common. As fluid pours out and cannot be replaced, the puppy becomes rapidly and severely dehydrated; the animals that die of the disease are described as extremely dehydrated at death. In the background, unseen without a blood test, the white-cell count is collapsing. In the rare cardiac form, very young puppies may instead show signs of sudden heart failure and die with little warning. The overall morbidity of CPV-2 in susceptible populations approaches 100 per cent- nearly every exposed, unprotected dog becomes infected- which is part of why the disease is so feared.

4.5 Diagnosis

In practice, parvovirus is often suspected on the clinical picture alone — a young, unvaccinated puppy with vomiting and bloody diarrhoea is a parvovirus case until proven otherwise. Confirmation is usually achieved with a rapid in-clinic antigen test performed on a faecal sample (the widely-used SNAP-type ELISA), which detects viral antigen in the stool and gives an answer within minutes. For research, surveillance, and confirmation of variants, more sensitive and specific molecular methods are used- chiefly the polymerase chain reaction (PCR) and its variants, which detect the virus's genetic material and can distinguish CPV-2a, 2b, and 2c. Most of the Indian prevalence studies discussed below relied on PCR or rapid antigen kits, or both.

4.6 Treatment and prognosis: the value of supportive care

There is no antiviral drug that reliably cures canine parvovirus. Treatment is supportive- that is, it does not kill the virus but keeps the dog alive and stable while the dog's own body clears the infection and the gut lining regenerates. The cornerstone is aggressive intravenous fluid therapy to correct the massive dehydration, alongside control of vomiting, correction of electrolyte and blood-sugar disturbances, antibiotics to counter the bacterial invasion that follows the gut-barrier breach, and careful nursing. Various adjuncts- hyperimmune plasma, agents to stimulate whitecell production, and antiviral drugs such as oseltamivir- have been tried, but the evidence for most of them in genuinely ill puppies remains limited or unproven, and none replaces good supportive care.

The prognosis is a study in contrasts, and the contrast is precisely the argument of this paper. Left untreated, parvovirus kills the great majority of the puppies it infects- mortality figures of more than seventy per cent, and in some series up to around ninety per cent, are reported in unvaccinated pups, against less than one per cent in adult dogs. With prompt, aggressive supportive care, that mortality can be cut dramatically- to somewhere in the range of twenty to forty per cent in various accounts. The difference between a puppy that lives and one that dies is therefore often not the virus but the response: whether the disease is caught early and whether the family can afford and access days of intensive hospital care. The mill puppy is doubly disadvantaged here, because it is both more likely to be infected and, frequently, sold to buyers who did not budget for a five figure hospital bill in the puppy's first month.

4.7 The Indian picture

Indian epidemiological studies confirm that parvovirus is common, that it concentrates in the young and unvaccinated, and that it shows the demographic pattern one would expect. A study in and around Bhubaneswar, Odisha, testing faecal samples from dogs with diarrhoea by PCR, found roughly forty-one per cent positive for CPV, with infection concentrated in dogs under six months of age and higher in males. A larger screening study in the Junagadh district of Gujarat found an overall prevalence of about 4.8 per cent across all dogs presented, but within the positive cases the pattern was stark: infection was highest in puppies under three months, in males, and- most tellingly- in non-vaccinated dogs, which made up over ninety per cent of cases. A study across Ahmedabad and Gandhinagar reported a combined case prevalence of parvovirus and distemper of about 8.7 per cent, with parvovirus specifically at around 5.7 per cent. And a survey of freeranging dogs in central India, measuring antibodies rather than active infection, found that more than eighty-eight per cent had been exposed to parvovirus at some point- evidence of just how thoroughly the virus saturates the general dog population from which so much of this disease ultimately flows.

Read together, these studies tell a coherent story. Parvovirus is endemic in India; it strikes puppies far more than adults and the unvaccinated far more than the vaccinated; and the reservoir of infection in the wider dog population is enormous. Every one of those facts points back to Sections 2 and 3. A trade that sells young, unvaccinated puppies into an environment where the virus is everywhere is not gambling- it is dealing from a marked deck.

Table 2: Selected Indian studies of canine parvovirus and distemper prevalence

Location (study)	What was measured	Key finding
Bhubaneswar, Odisha	CPV in diarrhoeic dogs, by PCR	~41% positive; concentrated in dogs under 6 months and in males.
Junagadh, Gujarat	CPV screening, kit + PCR	~4.8% overall; within cases, highest in pups under 3 months, males, and non-vaccinated dogs (>90% of cases).
Ahmedabad & Gandhinagar	CPV and CDV case prevalence	~8.7% for both combined; CPV ~5.7% and CDV ~11.6%.
Central India, free-ranging dogs	Antibody exposure (seroprevalence)	>88% exposed to CPV and >72% to CDV — a vast reservoir in the general population.

5. Canine Distemper (CDV): The Disease Without a Cure

If parvovirus is the fast killer, canine distemper is the slow and merciless one, and it is the disease at the heart of the case

study in Section 7. It deserves particular attention for a reason easy to state and hard to overstate: it cannot be cured. Every other disease in this paper can, with money and effort and luck, be treated. For distemper there is no treatment that removes the virus; once the disease has taken hold- especially once it reaches the nervous system- the veterinarian can offer support and comfort, but not a cure. Prevention is, quite literally, the only real defence, which makes the sale of unvaccinated puppies something close to a death sentence issued in advance.

5.1 The virus

The canine distemper virus is a single stranded RNA virus, which belongs to the family Paramyxoviridae, genus Morbillivirus and is an enveloped virus that causes distemper in dogs. It is related to the viruses of human measles and cattle rinderpest and the similarities are instructive: distemper is very contagious, is a profound immunosuppressor and may leave lasting damage to the nervous system like measles. It's also not just a dog disease, as it's been spread to a variety of carnivores, and some other mammals, which is part of the reason it can't be eradicated, and why it poses a threat to wildlife as well as pets. In the case of the individual dog, the important points are that he is highly susceptible, that it affects several different organ systems simultaneously and that it has a special and particularly fatal affinity for the brain.

5.2 Transmission

Distemper spreads chiefly through the air, by aerosol and respiratory secretions: an infected dog sheds virus in the droplets it breathes, coughs, and sneezes out, and a susceptible dog inhales them. This makes close contact between dogs- exactly the condition of a crowded mill or shelter- ideal for transmission. Because the virus also suppresses the immune system and is shed by animals that may not yet look obviously ill, an outbreak can move through a group of dogs before anyone realises distemper is present. As with parvovirus, the epidemiology is complicated by the large number of susceptible species that can maintain the virus in circulation.

5.3 Pathogenesis: from the lymph nodes to the brain

The course of distemper inside the body is a staged advance, and understanding the stages is essential to understanding both the clinical signs and the grim prognosis. After a dog inhales the virus, CDV first replicates in the lymphatic tissue near the point of entry- the tonsils and local lymph nodes. From there it spreads through the blood to the rest of the lymphoid system, and this early assault on the immune tissues produces the profound immunosuppression that is a signature of the disease and that opens the door to secondary infections.

What happens next depends heavily on the strength of the dog's immune response, and this is where the disease reveals its two faces. If the dog mounts a strong and rapid immune response, it may clear the virus and recover. If the response is weak or intermediate- as it will tend to be in a young, inbred, immunosuppressed mill puppy- the virus spreads onward from the lymphoid tissue to colonise the epithelial cells of many organs: the skin, the respiratory tract, the gastrointestinal tract, and, most fatefully, the central nervous system. Distemper is described as an epitheliotropic virus, meaning it targets epithelial (surface-lining) tissues, which is why its signs are so varied and body-wide.

The invasion of the central nervous system is the crux of the disease. There the virus establishes what is, in effect, a persistent infection, "hiding" within the nervous tissue from the immune system. This provokes a low-grade but relentless immune response that damages the tissue over time, and the characteristic lesion is a demyelinating encephalitis- an inflammation of the brain in which the myelin insulation around nerve fibres is progressively destroyed. Because this can smoulder for weeks, neurological signs may appear long after the initial illness seems to have passed: the systemic phase may run its course in around ten days, yet neurological signs can be delayed by several weeks or months as demyelination advances. A dog that appeared to "recover" from a bout of respiratory and gut illness can therefore begin, weeks later, to twitch, stumble, and convulse- a delayed betrayal that is one of the cruellest features of the disease and, as Section 7 shows, a central element of the case study. Figure 3 traces this progression through the body.

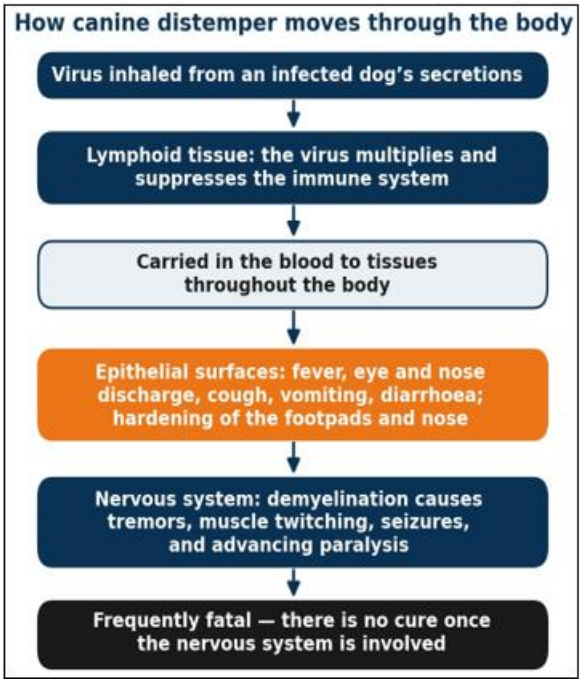

Figure 3: How canine distemper moves through the body: from inhalation, through the lymphoid tissue- where it multiplies and suppresses the immune system- and the epithelial surfaces, to the nervous system, where progressive demyelination produces the tremors, seizures, and paralysis that so often prove fatal.

5.4 Clinical signs: a disease of the whole body

Because distemper attacks so many systems, its clinical picture is broad, and it typically unfolds in a rough sequence from systemic and epithelial signs toward neurological ones. It is easiest to group them.

Systemic and general signs: The illness often begins with non-specific signs of systemic infection: fever, marked lethargy, and loss of appetite. These are easy to mistake for

any minor illness, which is part of why early distemper is so often missed.

Respiratory signs. As the virus colonises the respiratory epithelium, dogs commonly develop nasal and ocular discharge, coughing, and frequently, because of the accompanying immunosuppression and secondary bacterial infection- pneumonia. Ocular discharge and inflammation (conjunctivitis) are common, and distemper can affect the eyes more deeply as well.

Gastrointestinal signs: Involvement of the gut epithelium produces vomiting and diarrhoea, adding to the dog's debilitation and fluid loss.

Cutaneous signs: Two skin manifestations are worth highlighting because they feature in the case study. The first is a pustular dermatitis, particularly on the abdomen. The second, and more distinctive, is "hard pad disease": an abnormal thickening and hardening of the keratin on the footpads and the nose (nasal and digital hyperkeratosis), which is a classic, if not universal, sign of distemper and is suggestive of the diagnosis when it appears alongside other systemic signs. Distemper can also disrupt the development of tooth enamel in puppies infected before their adult teeth have formed, leaving permanent defects. The point for this paper is that distemper is, among other things, a skin disease, and a dog with deteriorating, crusting, hardening skin in the context of systemic and neurological illness fits the distemper picture closely.

Neurological signs: These are the most feared and, for prognosis, the most decisive. Distemper can produce a wide array of neurological abnormalities depending on where in the nervous system the demyelination falls: muscle twitching, focal or generalised seizures (including the classic rhythmic "chewing-gum" fits), cerebellar and vestibular signs causing loss of balance and coordination (ataxia), involuntary rhythmic muscle jerking (myoclonus), tremors, and weakness or partial paralysis of the limbs- paraparesis or tetraparesis- sometimes progressing toward more complete paralysis. In one hospital series of dogs showing neurological distemper, motor dysfunction was the single most common sign, and a large share of the affected dogs were nonambulatory- unable to walk- with tetraparesis. Crucially, these neurological signs, whether they come on acutely or chronically, tend to be progressive, and progression carries a poor prognosis. A dog that begins to show tremors and hind-limb weakness from distemper is, more often than not, a dog whose condition will worsen.

5.5 Diagnosis

The clinical picture (systemic, respiratory, gastrointestinal, ocular, cutaneous and especially neurologic signs in an unvaccinated or incompletely vaccinated dog) is often the only basis for diagnosis for distemper. There are several ways to get a confirmation, such as the finding of characteristic inclusion bodies (sometimes called Lentz corpuscles) in infected cells under the microscope, antibody assays, immunochromatographic (rapid) tests which test for the presence of the antigen in the stool, and laboratory tests which test for the presence of the virus's genetic material by reverse-transcriptase PCR. In real-world clinical settings, however, which are often encountered with patients in India, clinical judgment is the primary means of diagnosis, and rapid antigen testing is important, especially when neurological symptoms make the diagnosis clear.

5.6 Why there is no cure, and what "treatment" means

There is no specific therapy that cures canine distemper- no drug that reliably eliminates the virus from an infected dog. Treatment is entirely supportive: fluids for dehydration, antibiotics for the secondary infections the virus's immunosuppression invites, anticonvulsants for seizures, and nutritional and nursing care. Experimental adjuncts continue to be investigated- one randomised trial examined silver nanoparticles alongside supportive care- but such approaches remain investigational, not established cures. Everything the veterinarian does is aimed at keeping the dog alive and comfortable in the hope that its own immune system will prevail; nothing directly attacks the virus in a way that reliably works.

This is why the framing that opened this section matters so much. For a disease you cannot cure, the entire game is prevention. A vaccinated dog is very unlikely to develop distemper at all; an unvaccinated one that becomes infected and progresses to neurological disease is very likely to die or to be euthanised. The distance between those two outcomes is a single, inexpensive vaccine given on schedule- the very thing the mill puppy is denied.

5.7 Prognosis

The figures are disturbing. The prognosis is poor with high morbidity and high mortality with the onset of neurological signs. Nearly half of the dogs in the earlier neurological distemper study, some forty-seven per cent, died from the disease and the authors reported that as many as eightynine per cent of the dogs that became severely affected, especially neurologically, died or were euthanised. The demyelination does not resolve completely and the survivors usually have permanent deficits, such as persistent myoclonus, residual weakness, and/or permanent neurological damage. To sum up: distemper is a disease that frequently causes death, frequently causes maiming, and frequently maims and kills those that it does in the exact animals that the breeding industry produces – the young that have not been vaccinated. This process is followed by one of these animals in Section 7.

5.8 The Indian picture

Although the amount of data available for distemper is less than for parvovirus it does indicate a live threat, especially in India. Around 11.6 per cent of the animals were observed to be affected by distemper in the Ahmedabad and Gandhinagar study, which was higher than the number of those affected by parvovirus in the same study. The seroprevalence study in the central part of India revealed over 72 per cent of the free-ranging population of dogs had antibodies against the virus, suggesting a high prevalence of the virus in the general population with high antibody levels. Distemper has been reported to be a common cause of sickness and death throughout the country and a few Indian studies have

focussed on demyelinating neurological distemper and its association with age, sex and breed. The message is similar to that of parvovirus – the disease is endemic, the reservoir is vast and the animals at greatest risk and at least protected are young and unvaccinated animals.

Table 3: Canine parvovirus and canine distemper compared at a glance

Feature	Canine parvovirus (CPV-2)	Canine distemper (CDV)
Type of virus	Small, non-enveloped, single-stranded DNA (Parvoviridae)	Enveloped, single-stranded RNA; Morbillivirus (Paramyxoviridae)
Main route of spread	Faecal–oral; extremely hardy in the environment (months)	Airborne / respiratory secretions
Primary targets	Rapidly dividing cells: gut crypts, bone marrow, (heart in neonates)	Lymphoid tissue, then epithelia of many organs, then the CNS
Hallmark signs	Vomiting, severe bloody diarrhoea, dehydration, leukopenia	Fever, respiratory + GI signs, hard-pad skin change, then seizures, tremors, paralysis
Cure available?	No specific cure; supportive care only	No specific cure; supportive care only
Mortality (untreated pups)	Very high: >70%, up to ~90%	High, especially with neurological signs (~47% lethality in one series)
Chief defence	Vaccination + prompt intensive supportive care	Vaccination (prevention is essentially the only defence)

6. The Skin Dimension: Demodicosis, Pyoderma, and the Immunosuppressed Puppy

The two viral diseases above are the headline killers, but the mill puppy's suffering is rarely confined to a single condition. A deteriorating coat- patchy hair loss, crusting, redness, sores- is one of the most visible and common signs that something is wrong with such a dog, and it is a sign that features directly in the case study. It is worth understanding, because the most important skin disease in this context is not a random infestation but a direct readout of the puppy's compromised immune status, which ties the skin story back to the genetics and immunology of Section 3.

6.1 Demodicosis: when the immune system loses control of a normal resident

Demodectic mange (demodicosis) is a condition triggered by small mites (mainly Demodex canis) that reside in the follicles of the skin and glands of the fur. The key and frequently overlooked fact is that these mites can be a normal part of the skin of most healthy dogs, and are harmless in low numbers. The disease is not due to picking up the mite, but due to failing to control it: when an immune system of a dog fails to control the mites, they multiply to high numbers and clinical disease occurs. In essence Demodicosis is an infestation from within, which occurs because the immune system is unable to monitor or protect against the infestation.

Demodicosis is so revealing in the mill context because it is a highly contagious disease. The severe generalized form is very linked with a hereditary susceptibility and with immunosuppression (sensitiveness) congenital or acquired. Juvenile onset generalised demodicosis is usually detected around the age of three months and is probably due to some defect in the immune system that normally keeps these mites in check; the predisposition to develop the disease is inherited and dermatologists recommend that affected dogs should not be bred. Now read the logic in the text on Section 3: If an inbred puppy has a genetically weakened immune system, it's the same kind of animal in which a normally harmless mite is running amuck — breeding such an animal will propagate the defect that produces the disease.

6.2 Secondary pyoderma: the infection on top of the infestation

Demodicosis rarely stays a pure mite problem. The damaged, mite-riddled skin is readily invaded by bacteria, producing a secondary bacterial skin infection called pyoderma- and when this rides on top of demodicosis it is sometimes termed pyodemodicosis. The clinical picture then becomes markedly worse: erythema (redness), papules and pustules, hair loss (alopecia), oily seborrhoea, hyperpigmentation, crusting, and, as the infection deepens, furunculosis and the discharge of pus. The primary bacterial culprit in canine pyoderma is Staphylococcus pseudintermedius, an organism that is normally a harmless part of the skin's microbiota but that overgrows once the skin barrier and immune defences are compromised.

The severity of this is easy to underestimate. In its generalised, deep form, secondary pyoderma is not a cosmetic problem: deep and extensive pyoderma can become genuinely debilitating and even life-threatening, and dogs with severe disease can show systemic illness- generalised lymphnode enlargement, lethargy, and fever- when the deep infection takes hold. There is, moreover, a vicious circularity: the deep pyoderma itself appears to contribute to immunosuppression, so that the infection and the immune failure feed on each other. A mill puppy that arrives with a weakened immune system, develops demodicosis because of it, and then develops deep pyoderma on top of that, can find itself in a self-perpetuating spiral of skin destruction and further immune collapse- all of it, ultimately, traceable to how it was bred and kept.

The skin dimension therefore does two jobs. It broadens the picture beyond the two famous viruses to the wider burden the mill puppy carries; and it shows the same underlying mechanism from a different angle- a compromised immune system, produced by inbreeding and poor rearing, expressed this time as loss of control over the dog's own skin. In the case study that follows, deteriorating skin appears alongside the neurological collapse of distemper, a reminder that in the real, sick animal these processes do not occur in tidy isolation but pile onto one another at once.

7. Field Case Study: A Dog's Decline from Canine Distemper

Everything up to this point has drawn on published research. This closing section is different in kind: it is a first-hand field record of a single dog that the author followed directly, whose

short life brought together, in one body, much of what the earlier sections describe. The dog came from the unregulated trade, was never properly protected, passed through more than one of the diseases discussed in this paper, and did not survive. The author documented its decline in photographs and video, which accompany this account as a visual record of a course that textbooks usually describe only in words.

7.1 The dog and its history

The subject of this case was a Labrador Retriever, left unnamed here to preserve anonymity, that died at approximately thirteen months of age. It had come from an unregulated source and had no record of complete vaccination- the two circumstances that, as the earlier sections have argued, matter most. Although still young, by the time the events described here began it had already sustained a heavier medical burden than its age would suggest. It had survived an earlier illness whose clinical signs were consistent with canine parvovirus: the severe vomiting and diarrhoea that kill most unvaccinated puppies and leave the survivors weakened. It also carried a chronic skin disease- progressive hair loss accompanied by pus and crusting of the skin, the picture of a secondary bacterial infection (pyoderma) established upon demodicosis in an animal whose immune system was failing to contain it. In the terms of Section 3, an unvaccinated dog already weakened by one viral infection and burdened by chronic, infected skin disease was as vulnerable to a third and more dangerous infection as an animal can be.

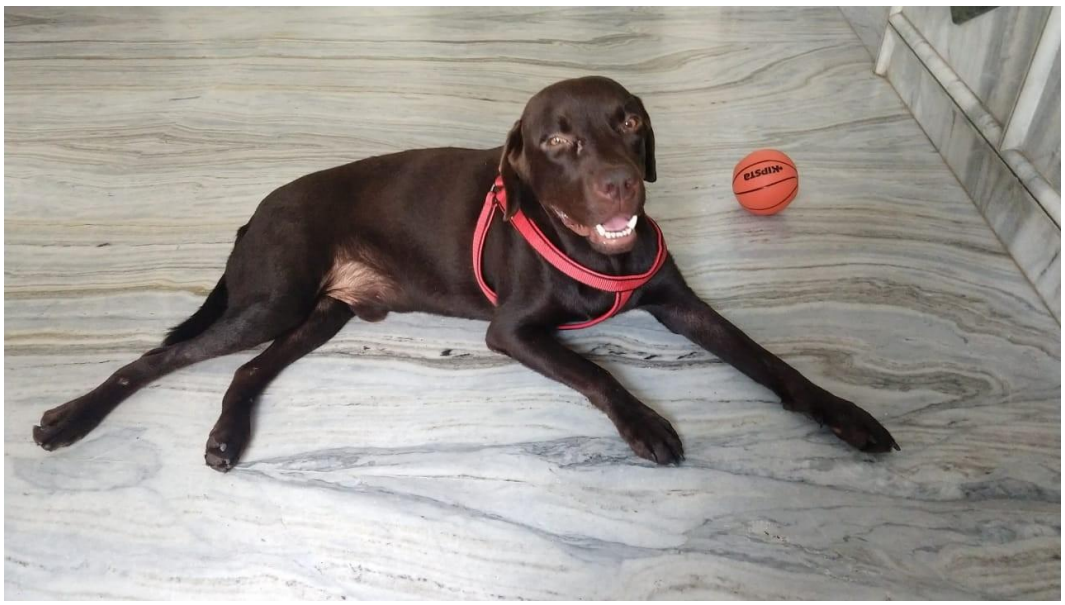

Figure 4: The subject of the case study: the Labrador Retriever whose decline is described in this section

7.2 The course of the illness

That third infection was canine distemper, and its course in this dog followed the natural history set out in Section 5 almost exactly. It began with the systemic signs of a general viral illness, and the skin, already compromised, worsened. Then, after the interval that is characteristic of distemper- a lull in which the animal can seem to be holding its own- the neurological phase began. There were tremors and involuntary, rhythmic twitching of the muscles; a loss of coordination and balance; and a creeping weakness of the limbs that advanced toward paralysis. This is the point at which distemper becomes, in practice, untreatable: fluids and nursing care can support a sick dog and ease its distress, but nothing available can repair the damage the virus does to the nervous system once it has taken hold. The decline continued through paralysis to its end, and the dog died. The author's photographs record the deterioration of the skin across this period, and the video records the tremors and failing coordination of the neurological phase.

7.3 What the case shows

A decline of this shape- from early systemic and skin signs, through a delayed onset of tremors and neurological deterioration, to paralysis and death - fits the established natural history of canine distemper closely, and Section 5 accounts for nearly every element of it. The gap between the first illness and the neurological signs is not evidence of two separate diseases; it is the expected consequence of the way distemper reaches the brain, spreading from the lymphoid tissue to the nervous system and there causing the slow, progressive demyelination that produces tremors, involuntary movement, and advancing paralysis weeks after the systemic phase seems to have passed. The progressive motor signs and the loss of the ability to walk are precisely what the clinical literature ties to a poor outcome, and to the majority of severely affected dogs dying or being euthanised.

The deteriorating skin fits the same picture. Distemper is an epitheliotropic virus that attacks the skin along with other epithelial surfaces, and it is itself immunosuppressive; in a dog already carrying the demodicosis and secondary infection of Section 6, the skin was being assaulted from more than one direction at once. This is the reality a tidy, disease-by-disease review can obscure: in a real patient, viral immunosuppression, epithelial infection, and opportunistic skin disease overlap and feed one another.

Taken together, this one animal traced almost the whole argument of the paper within a single life- a life that lasted barely thirteen months. Bred without regard for health, it

encountered one of the two great viral killers of Indian puppies while still young and survived it; it carried the skin disease of a compromised immune system; and it was finally overtaken by the second killer, in the form for which there is no cure. The disease sections above separate these conditions for clarity, but in the unregulated trade they are not three separate misfortunes falling on three different dogs. They are the successive and foreseeable consequences of the same original decision- to breed and sell an animal without protecting its health- arriving one after another upon the same body until it could withstand no more. Had this dog been bred responsibly and vaccinated on schedule, the likeliest outcome is simply that none of it would have occurred. That is the sense in which its death indicts the system rather than the animal: it was not an unlucky exception but the predictable end of the chain this paper has traced, given a face and a grave.

8. Prevention and the Way Forward

From the preceding sections it seems only natural that the issue of prevention, as opposed to treatment, should be overwhelmingly in favor since both of these central diseases can not be cured (distemper) or will kill a significant number of the untreated (parvovirus) and because both diseases tend to target young animals from unregulated sources. It outlines the scope for that prevention; the individual animal (by vaccination), the individual buyer (by responsible acquisition) and the state (by enforcement), and briefly discusses public awareness in this section too.

8.1 Vaccination: the single most effective intervention

The only weapon in this field which comes close to a winning weapon is vaccination. The fact that distemper and parvovirus are both a short list of 'core' vaccines, meaning every dog should be protected against no matter where and how they live, is due to the severity and the life-threatening nature of both diseases, as well as its worldwide distribution. The core vaccines are those that are required for distemper, adenovirus and parvovirus type 2; rabies is an added vaccine in rabies endemic areas like India. Vaccination is all but the only defence against a disease for which there is no cure.

The catch, as Section 3.3 explained, is timing. Because maternal antibody both fades and blocks vaccines at a level that varies from puppy to puppy, a single early dose cannot be relied upon. The WSAVA therefore recommends a series given every two to four weeks through early life, with the crucial final dose at sixteen weeks or older, once interfering antibody has almost always waned. Common schedules run at roughly six-to-eight, ten-to-twelve, and fourteen-to-sixteen weeks, then a booster at one year and every three years thereafter (Table 5). Where cost permits only one dose, it should be given at or after sixteen weeks.

The implication for the trade is damning. The mill puppy is sold before this schedule can be completed- often before it is even begun, and frequently with no vaccination at all. That is not a neutral omission; it is the removal of the one intervention standing between the animal and two of the deadliest diseases it will face. The rule for a buyer is simple: a puppy is not protected until it has finished the full series, with the last dose at sixteen weeks or later, and should be kept away from other dogs and contaminated ground until then.

Table 5: A representative core-vaccine schedule for puppies, following WSAVA/AAHA principles. Exact products and timing should be set by a veterinarian

Age	Core vaccination	Purpose / note
~6–8 weeks	First dose of core combination (distemper, adenovirus, parvovirus)	Begins the series; this dose is often neutralised by maternal antibody but "primes" the puppy.
~10–12 weeks	Second core dose	Given 2–4 weeks after the first; catches puppies whose maternal antibody has now waned.
~14–16 weeks	Third (final puppy) core dose	The crucial dose: given at 16 weeks or older so interfering maternal antibody is gone and the vaccine "takes".
~12–16 weeks	Rabies (where endemic, incl. India)	Considered essential in rabies-endemic regions.
~12 months	Booster	Consolidates protection after the puppy series.
Every ~3 years thereafter	Core viral booster	Long-lasting protection for distemper, adenovirus, parvovirus.

8.2 Responsible acquisition: how buyers can starve the trade

Vaccination protects the individual dog once it exists, but the deeper prevention is to change how dogs come into existence and into homes in the first place. Because the mill trade is demand driven, the choices of ordinary buyers are not trivial; collectively, they are the market. Two shifts in buyer behaviour would do more than any amount of moralising to reduce the disease burden described in this paper.

The first is to prefer adoption. India's shelters and streets are full of healthy indigenous dogs- Indies- that are, by most accounts, hardier and less prone to inherited disease than heavily linebred foreign breeds, and adopting one directly reduces the demand that sustains the mills while giving a home to an animal that already exists. The "Adopt, Don't Shop" argument is, at bottom, the recognition that buying a mill puppy funds cruelty and adopting a shelter dog does not.

The second, for those set on a particular breed, is to buy only from a genuinely responsible source and to refuse an unregulated one. A responsible breeder operates under licence, keeps a few animals in clean conditions, lets buyers see the mother and the premises, does not breed on every cycle, does not sell puppies before around eight to twelve weeks, provides veterinary and vaccination records, and is open about the parents' health. The warning signs of a mill are the mirror image of these, and the appendix sets them out as a checklist. A buyer who simply refuses to complete a purchase when those signs are present- and who treats seeing the mother and the vaccination record as non-negotiable- has done something concrete: made themselves a customer the mill cannot serve.

A member of the public who encounters a mill is not powerless. As noted in Section 2.3, the route is to complain to the State Animal Welfare Board, which is meant to send an

inspector, and to enlist an established welfare organisation to push the complaint forward; where cruelty is evident, the police can enter and seize animals in distress. Confronting operators directly is unwise; working through the organisation and the law is safer and more effective.

8.3 Policy and enforcement: closing the gap

The following practices, which are the same as those described above, are already banned by the Prevention of Cruelty to Animals Act, the Dog Breeding and Marketing Rules of 2017, and the Pet Shop Rules of 2018:These are the same practices as described above, and are already banned under the Prevention of Cruelty to Animals Act, the Dog Breeding and Marketing Rules of 2017, and the Pet Shop Rules of 2018: Where it is lacking is the means to implement those prohibitions; working State Animal Welfare Boards with offices and staff, a mechanism to receive and act on complaints and regular inspections of individual breeders and pet shops, and meaningful consequences for those that break the rules whether that be the cancellation of a licence, fines or the seizure of animals.

Several concrete measures follow. Registration and licensing of breeders and pet shops should be made a genuine precondition of operating, and enforced, so that the unregistered majority cannot continue to trade openly. The record-keeping already required by the rules- breeders logging their animals, pet shops logging their sources and buyers- should be audited, since traceability is what allows a sick puppy to be traced back to its origin and a repeat-offending mill to be identified. And the retail chokepoint deserves particular attention: other jurisdictions have shown that regulating what pet shops are allowed to sell can reshape the whole trade upstream. A frequentlycited example is a Californian law requiring pet stores to sell only animals sourced from shelters and rescues rather than commercial breeders, which strikes directly at the commercial-breedingto-retail pipeline. India's own move to restrict the commercial import of foreign dog breeds was aimed at a similar chokepoint, closing a loophole that breeders had used and, advocates hoped, reducing the incentive to run mills for exotic breeds. Whatever the specific instrument, the principle is the same: because the trade is driven by the profitability of selling puppies, the most effective interventions are those that make selling badly-bred, unhealthy puppies harder and less profitable.

8.4 The role of awareness

Underlying all of this is a problem of knowledge. Most buyers do not know that the cute puppy in the listing was very likely made sick by the conditions it came from; that they should ask for the mother, the premises, and the vaccination record; that an unvaccinated puppy faces two common, often-fatal diseases, one of them incurable; or that a hardier dog is waiting in a shelter for free. Every intervention in this section depends on closing that gap- which is part of why the field case study matters. A mortality statistic can be read and forgotten; a dog one has watched decline from a preventable disease is not.

9. Conclusion

This paper began with a claim that sounds like an exaggeration and is not: that the deaths of puppies from distemper and parvovirus in India are, in the main, not accidents but the foreseeable output of a broken system. The chapters between have tried to earn it link by link. A demand for cheap, pedigreed, foreign-looking dogs meets a supply industry organised to maximise saleable puppies at minimum cost, outside the reach of a body of law that exists but is barely enforced. That industry breeds unfit, closely-related animals, concentrating genetic disease and weakening immunity; keeps them in crowded, filthy conditions where hardy viruses persist; and sells them unvaccinated, weeks too early, dismantling the maternal-immunity bridge and never building the vaccination schedule that should replace it. The animal that emerges is not a healthy dog that might get unlucky. It is a vulnerability with a heartbeat.

When such an animal meets canine parvovirus, it meets a virus that destroys its gut lining and immune cells and kills most untreated puppies. When it meets canine distemper, it meets a virus that attacks the whole body and then the brain, can betray it weeks after apparent recovery, and that no medicine can cure- so the disease runs through tremors, paralysis, and deteriorating skin to death, exactly as in the case study at the centre of this paper. Behind both stands the same immune failure that lets a harmless mite run riot into demodicosis and pyoderma. These are not separate misfortunes befalling one unlucky dog; they are different expressions of a single cause- how the dog was made.

The hopeful part is that the leverage lies almost entirely on the side of prevention, and prevention is cheap. A vaccine on schedule prevents both headline diseases. A buyer who insists on seeing the mother, demands vaccination records, and walks away from a mill- or who simply adopts an indigenous dog- withholds the demand that keeps the trade alive. And a state that chose to enforce the rules it already has could close the gap between the law on paper and the suffering on the ground. None of this requires a scientific breakthrough; it requires knowledge, will, and the willingness to see the whole chain rather than only its final, tragic link. If this paper has a single aim, it is to make that chain visible- so that the next family standing before a listing for a cheap, too-young, unvaccinated puppy sees not just a photograph, but everything the photograph is hiding.

References

[1] MSD (Merck) Veterinary Manual. "Canine Distemper." Infectious Diseases. msdvetmanual.com.
[2] Nanai B., Lyman R., et al. "Mapping the pathogenesis of canine distemper virus." dvm360. (Drawing on Vandevelde M., "Pathogenesis of distemper infections in the nervous system," ACVIM Forum.)
[3] "Neurological Manifestation of Canine Distemper Virus: Increased Risk in Young Shih Tzu and Lhasa Apso with Seasonal Prevalence in Autumn." PMC (National Center for Biotechnology Information).
[4] "Canine Distemper" and "Canine Distemper Virus" overviews. ScienceDirect Topics (Veterinary Science and Immunology).

[5] "Evaluation of the Efficacy and Safety of Silver Nanoparticles in the Treatment of Non-Neurological and Neurological Distemper in Dogs: A Randomized Clinical Trial." PMC.
[6] Merck Veterinary Manual. "Canine Parvovirus Infection (Parvoviral Enteritis in Dogs)." Digestive System. merckvetmanual.com.
[7] "Canine parvovirology – a brief updated review on structural biology, occurrence, pathogenesis, clinical diagnosis, treatment and prevention." ScienceDirect (Comparative Immunology, Microbiology and Infectious Diseases).
[8] Nandi S., Kumar M., et al. "Canine Parvovirus: Current Perspective." PMC.
[9] Royal Canin Academy. "Canine Parvovirus: Signs and Treatment." academy.royalcanin.com.
[10] "Canine parvovirus type 2 (CPV-2) and bacterial coinfections in dogs: phenotypic and genotypic resistance profiles in northern Kazakhstan." Frontiers in Veterinary Science.
[11] "Epidemiology, molecular prevalence and prevention on canine parvovirus in India: A review." PMC.
[12] "Epidemiological study of canine parvovirus infection in and around Bhubaneswar, Odisha, India." PMC (Veterinary World).
[13] "Prevalence of Canine Parvo Viral Infection in Dogs in and around Junagadh District of Gujarat State." Indian Journal of Veterinary Sciences and Biotechnology.
[14] "Pilot Scale Study on the Prevalence and Associated Risk Factors of Canine Distemper and Canine Parvovirus in Ahmadabad and Gandhinagar Cities of Gujarat, India." The Indian Veterinary Journal.
[15] "Epidemiology of viral pathogens of free-ranging dogs and Indian foxes in a human-dominated landscape in central India." PubMed.
[16] Vaccination Guidelines Group (VGG) of the World Small Animal Veterinary Association (WSAVA). "Guidelines for the Vaccination of Dogs and Cats" (2015 and 2024 editions). wsava.org; Journal of Small Animal Practice.
[17] "Dog Vaccination Schedule: AAHA + WSAVA Guidelines" (vet-reviewed summary). beomelo.com.
[18] "The effect of inbreeding, body size and morphology on health in dog breeds." Canine Medicine and Genetics / Companion Animal Health and Genetics (BMC / Springer Nature).
[19] Australian Shepherd Health & Genetics Institute (ASHGI). "Using Inbreeding Coefficients." ashgi.org.
[20] Institute of Canine Biology. "Coefficient of Inbreeding" (summary of principles, incl. Sewall Wright). instituteofcaninebiology.org. Puppy-mill definition and public-remedy guidance also drawn from Dawgiebowl, "Puppy Mills – The Sickening Truth Behind Illegal Breeders in India," dawgiebowl.com.
[21] "What Is Considered Inbreeding in Dogs: COI Explained." ScienceInsights (scienceinsights.org).
[22] "The Consequences of Dog Inbreeding: Vet-Verified Problems & Risks." Dogster (dogster.com).
[23] "The Dark Side of Pet Breeding: A Socio-Legal Examination of Puppy Mills in India." International Journal of Law, Legal Research and Analysis (ijllr.com).
[24] "Illegal pet shops & dog breeding industry, worth over Rs 1000 cr, thrive on human avarice, graft in govt." National Herald (nationalheraldindia.com). Includes remarks attributed to Maneka Gandhi and Ambika Shukla / Gauri Maulekhi.
[25] Humane World for Animals (Humane Society International). "India mandates new regulations to stop animal cruelty and neglect by pet stores." humaneworld.org.
[26] "With Govt ban on import of foreign dog breeds, animal lovers hope 'puppy mills' will finally close." The News Minute (thenewsminute.com). Includes remarks attributed to Vasudev Murthy and Suparna Ganguly (CUPA).
[27] Friendicoes. "The unseen and often ignored side of dog breeding." friendicoes.org.
[28] Merck Veterinary Manual. "Mange in Dogs and Cats" (Demodicosis). Integumentary System.
[29] merckvetmanual.com.
[30] Merck Veterinary Manual. "Pyoderma in Dogs and Cats." Integumentary System. merckvetmanual.com.
[31] "Canine demodicosis: Hematological and biochemical alterations." PMC.
[32] "Immunosuppression in dogs with pyoderma and/or demodicosis." PubMed.
[33] "Demodicosis in first opinion caseloads of dogs: the 'mitey' facts on frequency and breeds." Veterinary Ireland Journal.
[34] Prevention of Cruelty to Animals Act, 1960; Prevention of Cruelty to Animals (Dog Breeding and Marketing) Rules, 2017; Pet Shop Rules, 2018 (Government of India). Provisions summarised via references 22–25 above and "Understanding Animal Laws in India," pawlidayinn.in.
[35] Law Commission of India report (August 2015) and the associated PETA India submission on the size and growth of the pet trade, as reported in reference 24.
[36] Euromonitor International, on India as the fastest-growing pet market, as cited in Dawgiebowl, "Puppy Mills – The Sickening Truth Behind Illegal Breeders in India," dawgiebowl.com.
[37] "3.1 Canine Demodicosis," Small and Large Animal Dermatology Handbook, Vol. 1. University of Minnesota (open.lib.umn.edu). Supplementary to references 28 and 30.
[38] Additional regional Indian clinical reports on canine distemper (e.g., studies from Parbhani, Maharashtra, and the demyelinating-neuropathy risk analysis in the International Journal of Livestock Research), as indexed alongside reference 14.

Appendix: A Practical Checklist for Prospective Dog Owners

The following checklist distils the practical guidance of Section 8 into a form a prospective owner can actually use. It is not a substitute for veterinary advice, but it captures the questions and warning signs most likely to separate a responsible source from an unregulated one, and the basic protections every new puppy needs.

Before you buy or adopt

- ☐ Consider adoption first. Ask local shelters and rescues whether they have a dog that suits your home; indigenous Indian dogs are typically hardy and healthy.
- ☐ Decide honestly whether you can meet the cost of proper care- including the full puppy vaccination series and the possibility of veterinary bills in the first months.

If buying a specific breed, insist on all of the following

- ☐ See the mother, with the puppies, at the actual premises where they are kept. Refusal is a red flag.
- ☐ Ask the puppy's age and refuse very young puppies. A puppy should not leave its mother before roughly eight to twelve weeks at the very earliest.
- ☐ Ask for written vaccination and deworming records, and veterinary documentation for the parents' health.
- ☐ Confirm the breeder is licensed / registered with the State Animal Welfare Board.
- ☐ Ask about the parents' relatedness and health history; a responsible breeder breeds for health, not just appearance, and avoids close inbreeding.

Warning signs of a puppy mill or backyard breeder — walk away if you see these

- ☐ The mother or the breeding premises cannot be seen.
- ☐ Puppies are offered very young, or several breeds / constant litters are always available.
- ☐ No vaccination or veterinary records; the seller is vague about the puppy's history.
- ☐ Sale happens in a car park, by the roadside, or through an anonymous handover.
- ☐ Prices are unusually low, or the seller pressures you to decide quickly.

After you bring a puppy home

- ☐ See a veterinarian promptly for a health check and to start or continue the core vaccination series (distemper, adenovirus, parvovirus; rabies where required).
- ☐ Complete the full puppy series, with the final core dose at sixteen weeks of age or older- the puppy is not fully protected until then.
- ☐ Until the series is complete, limit the puppy's contact with unknown dogs and potentially contaminated environments, because parvovirus in particular persists in the environment for months.
- ☐ Watch for early warning signs- loss of appetite, vomiting, diarrhoea (especially bloody), nasal or eye discharge, coughing, or any twitching, tremors, or weakness- and seek veterinary care immediately if they appear. With parvovirus especially, early aggressive supportive care greatly improves survival.

If you suspect a puppy mill

- ☐ Do not confront the operators yourself- it can be unsafe.
- ☐ Complain to the State Animal Welfare Board, which is meant to send an inspector, and contact an established animal-welfare organisation or NGO to help push the complaint forward.