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Advancements in Health Research

Advancements in Health Research 2026; 3:133
doi:10.4081/ahr.2026.133

Hold my paw: the unseen exchange in our homes and its impact on health

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Abstract

The human-animal bond represents a topic of growing scientific and public health relevance, as companion animals share close living environments with humans in a substantial proportion of households across many countries. This intimate cohabitation creates a shared microbial environment, facilitating constant, bidirectional exchange of microorganisms between pets and their owners. Here, we explore the dynamics of this microbial transfer and its influence on human health, specifically focusing on immune-mediated, metabolic, and gastrointestinal disorders. Drawing from the "One Health" concept, which recognizes the interconnectedness of human, animal, and environmental health, we examine how companion animals shape the composition of the human gut microbiota. Evidence suggests that while this exchange carries potential risk for transmitting pathogens and antimicrobial resistance, the overall effect appears beneficial. Pet exposure, particularly in early life, is associated with increased microbial diversity and enrichment of beneficial taxa such as *Ruminococcus* and *Oscillospira*. These alterations are linked to reduced risk of immune-mediated conditions like allergies and asthma, consistent with the "microbiota hypothesis". Furthermore, pet ownership correlates with lower prevalence of metabolic syndrome, potentially mediated by favorable shifts in gut microbiota, such as a reduced *Firmicutes*-to-*Bacteroidetes* ratio. However, household transmission of extended-spectrum β-lactamase-producing *Enterobacterales* represents a tangible public health concern requiring antimicrobial stewardship. We synthesize current findings, highlighting the household as a key epidemiological unit and positioning the shared microbiome as a critical factor in health outcomes of pet-owning families.

Key words: gastrointestinal microbiome; pets; one health; humans; dysbiosis; drug resistance; microbial; metabolic syndrome; hypersensitivity.

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Introduction

The modern household is a complex ecosystem in which humans and companion animals (CAs) live closely together.[1] This relationship confers significant health benefits such as reduced stress and improved cardiovascular health.[2] One of the mechanisms by which CSs may grant this benefit is an unseen, constant interaction: the sharing of microbiota.[3] Every pat, lick, cuddle, and shared space facilitates the transfer of bacteria, viruses, and fungi, creating a unique microbial fingerprint within each household.[4]

The "One Health" framework, which posits that human, animal, and environmental health are intimately related, provides the best explanation for this phenomenon.[5] The impact of environmental factors, including pets, has emerged as a key research topic as our knowledge of the human microbiota's role in health and disease deepens.[3] The "microbiota hypothesis" has replaced the "hygiene hypothesis," which first proposed that reduced exposure to microbes during early childhood increases a person's vulnerability to allergic diseases. According to this updated theory, our gut microbes' diversity and composition, influenced by environmental exposures, "train" our immune systems and affect our long-term health.[6]

This review synthesizes the evidence on the bidirectional microbial transfer between humans and CAs. It will first establish the mechanisms and extent of this sharing, then analyze the specific impacts on the composition of the human gut microbiota (GM), and finally explore the subsequent health implications for immune-related disorders and gastrointestinal health in pet-owning versus non-pet-owning households (Figure 1).

Methods

This narrative review was conducted through a systematic search of the PubMed/MEDLINE, Scopus, and Web of Science databases. The following search terms were used in various combinations: "pet ownership", "companion animals", "gut microbiota", "microbiome", "One Health", "microbial transfer", "human-animal bond", combined with "immunity", "allergy", "asthma", "metabolic syndrome", "antimicrobial resistance", and "dysbiosis". Articles published in English from January 2000 to December 2024 were eligible. Inclusion criteria encompassed peer-reviewed original articles, systematic reviews, and meta-analyses addressing microbiota-mediated mechanisms or health outcomes associated with pet exposure in humans. Grey literature, conference abstracts, and studies not reporting microbiota-related outcomes were excluded.

Evidence of bidirectional transfer

It is well known that living together makes microbes more alike. Being a member of a household is a strong predictor of a person's microbiota, both for people and their pets.[7] This sharing occurs through multiple direct and indirect pathways.

Ways to transfer directly and indirectly

Direct contact, such as petting, cuddling, and licking, is a clear way for microbes to move from a pet to its owner and *vice versa*.[1] Research indicates that dog ownership markedly enhances the shared skin microbiota among household members, making an individual's skin microbiota more similar to their dog's than to that of other dogs.[4] The transfer is most noticeable on the skin, but it also happens in the gut. Initial research identified common intestinal bacteria in both pets and their owners, and subsequent sequencing studies have validated these findings.[8] The examination of 16S rRNA gene amplicon sequence variants (ASVs) has demonstrated the transfer of gut microbes between cohabiting humans and dogs.[5]

Transferring through the environment is also very important. Pet feces is a major source of microbial input into the home environment. This creates a feedback loop in which pets alter the microbes in the home, which, in turn, alters the people who live there's microbiota.[9]

Bidirectional flow of pathogens and commensals

Microbes can move in more than one direction. Evidence indicates a resilient bidirectional pathway, encompassing not only benign commensals but also potential pathogens and antimicrobial resistance (AMR) genes. The household has been recognized as a principal epidemiological unit for the dissemination of antimicrobial-resistant bacteria.[1] Studies have shown that *Enterobacterales* that produce extended-spectrum beta-lactamases (ESBLs) and AmpCs can be transferred from infected pets to their healthy owners.[1] In the same way, healthy dogs and their owners have been found to share clones of fecal *Escherichia coli*, with within-household sharing more common than across-household sharing.[10] This shows that the same pathways that allow beneficial microbes to move can also allow clinically important organisms to move in the opposite direction, highlighting the importance of hygiene and responsible antimicrobial stewardship in pet-owning households.[10]

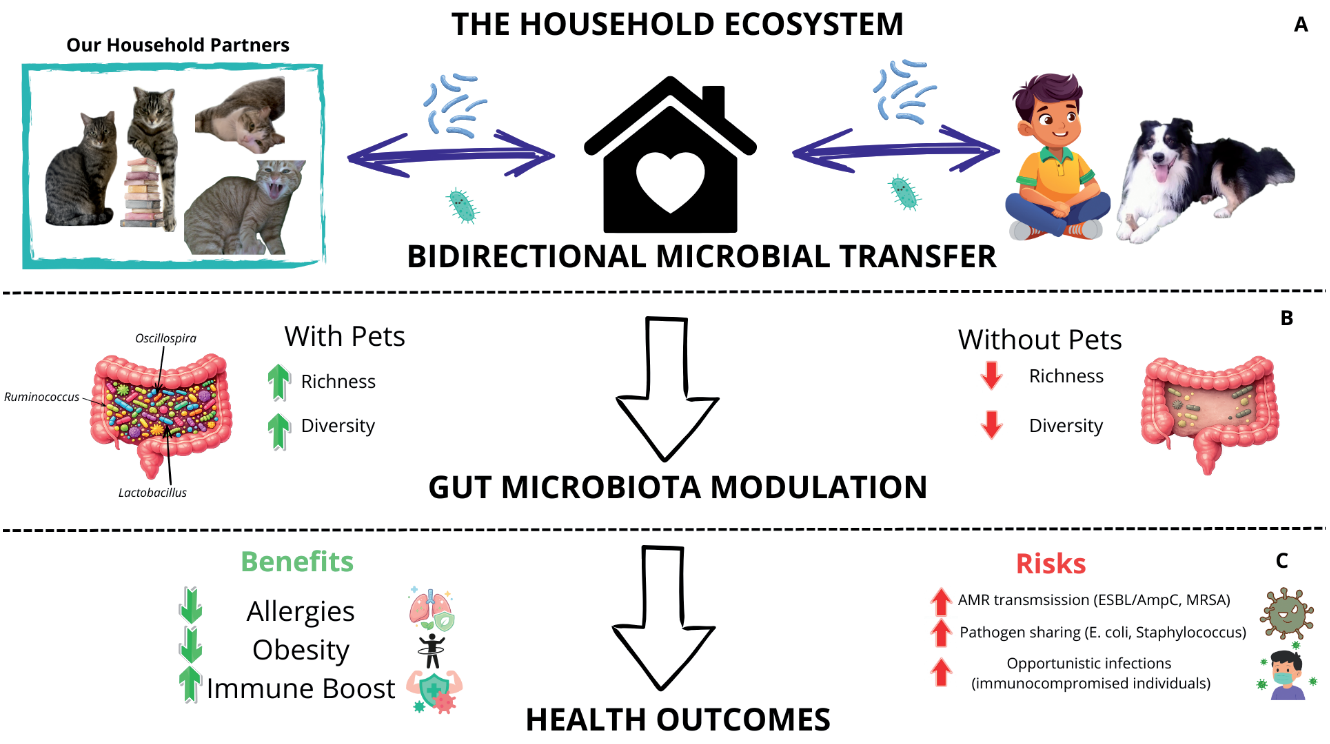

Figure 1. Pet exposure modulates gut microbiota and health. **A)** Bidirectional microbial transfer between companion animals and humans. **B)** Pet owners exhibit increased gut microbiota diversity with enrichment of *Oscillospira*, *Ruminococcus*, and *Lactobacillus*. **C)** Health outcomes: benefits (↓ allergies, ↓ obesity, ↑ immune function) and risks (↑ AMR transmission, ↑ pathogen sharing, infection risk in immunocompromised individuals). AMR, antimicrobial resistance; ESBL, extended-spectrum beta-lactamase; AmpC, AmpC beta-lactamase; MRSA, methicillin-resistant *Staphylococcus aureus*. Figure created by Alberto Corriero.

The impact of companion animals on human gut microbiota composition

Living with a pet changes the composition of the human GM, particularly during the critical developmental window of infancy and early childhood.[11]

Influence on microbial diversity and richness

The effect of pet ownership on overall gut microbial diversity seems to depend on age. Some studies have indicated no substantial difference in alpha or beta diversity between pet owners and non-owners in adults.[8] However, in infants, the effect is more pronounced early-life exposure to furry pets is associated with increased richness and diversity of the GM.[12] For instance, a study involving 746 infants from the CHILD cohort demonstrated that pet exposure markedly increased overall microbial richness.[13] This early-life enrichment may be a key factor in the observed health benefits, as low microbial diversity in infancy has been linked to a higher risk of atopic diseases.[14,15]

Changes in important bacterial taxa

In addition to overall diversity, having pets is linked to major changes in the abundance of certain groups of bacteria:

- *Enrichment of beneficial bacteria*: numerous studies have consistently demonstrated that exposure to pets, particularly during the prenatal and postnatal periods, enhances the infant GM with the genera *Ruminococcus* and *Oscillospira.*[3,13,16] This finding is significant because both bacteria have been negatively associated with childhood atopy and obesity.[12,17] Living with a companion animal has also been linked to higher levels of *Coprococcus* and *Oscillospira* in adults.[13] Other beneficial bacteria, such as *Lactobacillus*, have been shown to increase in individuals living with pets.[3,8] Additionally, *Faecalibacterium* can be modulated indirectly through probiotic supplementation in household dogs, as demonstrated in a recent study with an increased *Faecalibacterium* in children whose dogs received canine-specific probiotics.[16]
- *Lowering of potentially harmful bacteria*: in some cases, being around pets has been linked to a lower number of bacteria that cause inflammation or dysbiosis. For example, vaginally born infants who were given antibiotics during labor and delivery had a much lower level of *Streptococcaceae* when they lived with pets and had fewer *Enterobacteriaceae.*[13]
- *Change in phylum-level ratios*: one study found that adults who lived with CAs had a much lower *Bacillota/Bacteroidota* (formerly *Firmicutes/Bacteroidetes*) ratio than adults who did not own pets.[3] This is an important finding because a high *Firmicutes*-to-*Bacteroidetes* ratio has been linked to obesity and metabolic disorders and is thought to be a sign of gut dysbiosis.[18]

Interventions can even influence these changes. A study demonstrated that administering a canine-specific probiotic to household dogs led to measurable changes in the gut microbiota of the children living with them, characterized by a reduction in *Bacteroides* and an increase in short-chain fatty acid (SCFA) producers such as *Faecalibacterium.*[16] This offers direct evidence that modulating the pet's microbiota can directly affect the human microbiota.

These compositional changes have been documented across multiple cohortsusing diverse methodological approaches. Table 1 provides a comprehensive summary of the key studies examining the relationship between pet exposure and human microbiota composition.

Health implications I: immune-related disorders

The microbial changes induced by pet ownership have major impacts on the development and regulation of the human immune system.[19] The protective effect of pet ownership against allergic diseases is a prime example of the "microbiota hypothesis" in action.[6]

Pets bring a wide range of environmental microbes into households, making urban areas feel like "mini-farms".[20] This exposure, particularly in the first year of life when the immune system is maturing, facilitates a balanced, tolerogenic immune response.[21] Research consistently indicates that early exposure to pets diminishes the incidence of atopic and allergic diseases.[22-24] For instance, a reduced incidence of allergic illness (atopic dermatitis, asthma) in offspring was observed in a meta-analysis of 6 studies that assessed prenatal exposure to household pets, particularly dog ownership.[24] The enrichment of gut bacteria like *Ruminococcus* and *Oscillospira* is believed to be a key mechanism. *Ruminococcus* produces SCFAs that have demonstrated a preventive role against allergy, while *Oscillospira* produces butyrate through fermentation of host glycans and products from other bacterial species.[13] These SCFAs contribute to gut health and metabolic balance,[25,26] though the precise immunoregulatory mechanisms remain incompletely understood.

While the evidence for a protective effect is strong, this relationship is complex: for people who already have allergies, owning a pet can sometimes make their symptoms worse. Among sensitized asthmatics, exposure to higher dog or cat allergen levels was associated with substantially increased asthma attacks, supporting recommendations to avoid or reduce exposure in sensitized individuals.[27] The data, however, mostly show that consistent exposure at an early age helps prevent sensitization in the first place.[28]

Health implications II: gastrointestinal and metabolic health

The pet-shared microbiome affects more than just allergies; it also affects gastrointestinal and metabolic health.

Gastrointestinal infections

The bidirectional exchange of microbes between pets and humans raises a concrete, not merely theoretical, risk of zoonotic transmission of gastrointestinal pathogens and antimicrobial-resistant *Enterobacterales* within households.[1] Molecular typing studies have shown that humans and their companion animals can carry indistinguishable ESBL/AmpC-producing *E. coli* strains,[1] and similar within-household sharing of clonal *E. coli* (though not necessarily antibiotic-resistant) has been documented using pulsed-field gel electrophoresis multiple cohorts from New Zealand, Netherlands, and the UK.[10,29,30]

Table 1. Summary of studies evaluating the relationship between pet exposure and human microbiota composition, microbial sharing, and related health outcomes.

Study	Design and population	Microbiota target and methods	Evidence of transfer	Main microbiota findings	Health implications
Do *et al*. (2024)[3]	Cross sectional comparative. 60 families with companion animals, 60 without. Adults and children	Human gut microbiota. 16S rRNA fecal sequencing	Transfer suggested *via* shared environment. Adults differed $p=0.03$. Children no difference $p=0.71$	No alpha diversity change. Beta diversity different $p=0.001$. Lower *Bacillota Bacteroidota* ratio 0.67 *vs* 1.02. Enrichment *Ruminococcaceae*, *Oscillospira*, *Lactobacillus*. Controls enriched *Megamonas*, *Veillonella*	Possible metabolic benefit. Lower ratio linked to lean phenotype and immune function. No direct clinical outcomes measured
Menezes *et al*. (2024)[1]	Prospective longitudinal 2018 to 2021. 65 households with infected pets and owners. Portugal and UK	ESBL AmpC Enterobacterales. Culture, WGS, phylogenetics	Direct bidirectional sharing. Identical resistant strains in 5 PT and 2 UK households. Pet antibiotic therapy increased sharing	Focus on pathogens. Geographic variation in resistance genes blaCTX M 15 Portugal, blaCMY 2 UK	Public health risk. Household key unit for AMR transmission. Zoonotic potential demonstrated
Kates *et al*. (2020)[8]	Nested case control. 332 adults. 178 owners, 154 non owners	Adult gut microbiota. 16S rRNA stool sequencing	Transfer inferred. No major clustering by ownership	No alpha or beta diversity differences. Eleven OTUs differentially abundant. *Akkermansia* and *Clostridium* XIVa higher in owners	Subtle health association. Akkermansia linked to gut protection and metabolic health
Nermes *et al*. (2015)[21]	Observational. 115 infants at allergy risk. 51 pet exposed, 64 non exposed	Animal derived *Bifidobacterium*. qPCR fecal samples	Direct transfer of specific bacteria. *B. pseudolongum* more prevalent in exposed infants 33.3% *vs* 14.1%	Target species only. No full microbiota profiling	Possible immune modulation. Transfer may influence infant immune programming
Gómez Gallego *et al*. (2021)[16]	Interventional. 31 allergic family children with dogs probiotic *vs* placebo. 18 dog free controls	Dog and child gut microbiota. 16S rRNA	Modifiable transfer. Dog probiotic induced parallel changes in child microbiota	Dog exposed children more diverse enterotype. Higher *Bacteroides*, *Ruminococcus*, *Lachnospiraceae*. Increase *Faecalibacterium* after probiotic	Supports mini farm effect. Increased diversity and SCFA producers may protect from allergy
Misic *et al*. (2015)[7]	Cross sectional and 3 month longitudinal. 25 households. 30 humans, 56 pets	*Staphylococcus* carriage microbiota. Nares, skin, oral. 16S rRNA	Strong household sharing. Pet microbiota more stable over time	Pets dominated by Proteobacteria. Humans by Firmicutes and Actinobacteria. Greater heterogeneity in pet homes	No association with MRSA or *S. aureus* infection status
Stenske *et al*. (2009)[10]	Cross sectional. 61 dog owner pairs. 30 human controls	Fecal *E. coli*. PFGE and susceptibility testing	Significant within household sharing. Clonal *E. coli* 9.8% *vs* 0.3% unrelated	Single species focus. No susceptibility difference between paired dogs and owners. Hygiene linked to lower resistance	Minimal zoonotic AMR risk in healthy cohort
Tun *et al*. (2017)[13]	Cohort subsample. 746 infants. CHILD study	Infant gut microbiota at 3 to 4 months. 16S rRNA	Transfer inferred from pet exposure. Effects varied by birth mode and antibiotics	Increased richness with prenatal exposure. Enrichment *Oscillospira* and *Ruminococcus*. Reduced *Streptococcaceae* and *Enterobacteriaceae* in some cases	Possible immune benefit. Associations with lower atopy and obesity risk

AMR, antimicrobial resistance; ESBL, extended spectrum beta lactamase; MRSA, methicillin resistant *Staphylococcus aureus*; OTUs, operational taxonomic units; PFGE, pulsed field gel electrophoresis; qPCR, quantitative polymerase chain reaction; rRNA, ribosomal ribonucleic acid; SCFA, short chain fatty acids; WGS, whole genome sequencing.

Specifically, transmission of index clinical ESBL-producing *E. coli* and *Klebsiella pneumoniae* strains to cohabitant humans was documented in three Portuguese households (6.9% of studied households), with repeated isolation of the index strains from fecal samples of both animals and their cohabiting humans. In the same multinational study (Portugal/UK), two UK households showed sharing of indistinguishable ST963 *E. coli* strains between dogs and humans (≤9 SNPs).[1] Additional evidence from a New Zealand study identified identical ESBL/ACBL-producing *E. coli* strains (by antibiogram, genotype, MLVA) shared between humans with UTIs and household dogs in 2/11 households.[30] A Dutch study similarly detected household transfer of ESBL/pAmpC-producing *Enterobacterales* between known human carriers and dogs.[29]

Companion animals, including healthy dogs and cats, frequently harbor multidrug-resistant intestinal *E. coli* and other *Enterobacterales*, which may cause extra-intestinal infections in humans (e.g., urinary tract infections) and facilitate the dissemination of resistance genes across host species. ESBL/AmpC-producing *Enterobacterales* were detected in 55.8% of Portuguese companion animals and 36.4% of UK animals, with corresponding carriage rates of 35.9% and 12.5% in their human cohabitants.[1] From a public health perspective, this evidence suggests that antimicrobial use and hygiene practices in pets can indirectly modulate the resistome of all cohabiting family members,[4] reinforcing the relevance of a One Health approach to antibiotic stewardship and household infection control strategies.[31]

Despite this microbiological and genomic evidence of shared resistant strains, epidemiological data do not consistently show an increased incidence of routine GI infections in pet-owning households. A large Australian study of rural families with young children found that living with a dog or cat was not associated with higher rates of acute gastroenteritis; on the contrary, adjusted odds of gastroenteritis were significantly lower in children from households with a dog or a cat than in those without pets.[32] These findings suggest that close contact with pets may simultaneously increase opportunities for the exchange of resistant organisms while, through early and repeated microbial exposures, potentially contributing to immune maturation and reduced susceptibility to common self-limited GI infections.However, the clinical significance of asymptomatic carriage of AMR bacteria remains uncertain, and data remain limited.

Overall, the current literature characterizes households with pets as potential hotspots for the circulation of antimicrobial-resistant bacteria but provides less robust evidence for a clinically meaningful increase in everyday GI disease burden, underscoring the need for prospective, longitudinal studies that integrate microbiota, resistomes, and clinical outcomes.

Metabolic syndrome

A growing body of clinical and microbiota research supports an association between pet ownership and a lower prevalence of metabolic syndrome and obesity.[33,34] Data suggest that gut GM diversity and composition are associated with reduced risks of metabolic syndrome, a condition characterized by abdominal obesity, dyslipidemia, hypertension, and impaired insulin sensitivity.[35,36] The mechanism may be the modulation of the GM by lowering the *Firmicutes* to *Bacteroidetes* ratio, a marker associated with metabolic and immune health.[37] As a matter of fact. in a study comparing families with and without CaS, the *Bacillota/Bacteroidota* ratio was considerably lower in the companion animal group (0.67) than in the group without companion animals (1.02).[3] Specifically, bacteria repeatedly implicated in leanness and lower metabolic risk, such as *Oscillospira*, appear more abundant in individuals cohabiting with CaS. Exposure to pets increased the abundance of *Oscillospira* ($\log_2$ fold change: 5.1; adjusted p=0.012) 9, a genus that has been negatively associated with childhood atopy and obesity,[3] reinforcing the hypothesis that pet associated microbial sharing and lifestyle changes jointly shape a microbiota configuration leading to improved metabolic control.

In a cross-sectional study of coronary patients, pet owners had a significantly lower prevalence of metabolic syndrome and obesity than non-owners, and this apparent protective effect was strongest among dog owners.[34] Beyond increased physical activity and psychosocial factors, alterations in the GM emerged as a key mechanistic candidate: pet owners displayed distinct microbial profiles, including enrichment of *Methanobrevibacter*, *Coprococcus*, and *Oscillospira* in dog owners, taxa previously linked to a healthier metabolic phenotype and improved cardiometabolic risk markers. In the same cohort, pet ownership was associated with microbiota configurations that the authors interpreted as more favorable for metabolic health, and they proposed that pet-related modulation of the gut ecosystem might be one pathway through which pets contribute to cardiometabolic protection in high-risk individuals.[34]

A critical question arises: are the observed metabolic benefits attributable to microbiota modulation or simply to increased physical activity from dog walking? Several lines of evidence support a microbiota-mediated mechanism rather than exercise alone. First, the most robust microbiota changes are documented in infants aged 3-4 months, a population incapable of walking dogs or engaging in exercise-related activities. These infants exhibit significant enrichment of *Oscillospira* and *Ruminococcus*, identical to taxa enriched in adult pet owners, suggesting environmental microbial transfer rather than lifestyle-mediated effects.[13] Second, household-level studies demonstrate that all cohabitating family members, not just the primary dog walker, how convergent microbiota profiles.[7] Children and non-dog-walking adults in pet-owning households display similar microbial shifts, indicating that shared environmental exposure (e.g., contact with pet fur, saliva, feces; contaminated household surfaces) drives compositional changes independently of individual physical activity levels.[4] Third, cat ownership, which involves minimal physical activity demands, still associates with microbiota alterations and reduced allergic disease risk,[21] further decoupling the effect from exercise. While physical activity undoubtedly contributes to metabolic health in dog walkers, the convergent evidence from infant, household-wide, and cat-owner studies strongly implicates bidirectional microbial transfer as the primary mechanism linking pet ownership to favorable gut microbiota composition and, by extension, to immunologic and metabolic benefits.

Experimental work in obese dogs also indicates that manipulating gut microbial communities can influence body weight regulation, insulin resistance, and inflammatory tone, supporting a broader, cross-species link between microbiota architecture and metabolic homeostasis.[38,39] Taken together, these data suggest that pet ownership, particularly of dogs, may act as a modifiable environmental factor that reshapes the human GM and, in turn, attenuates the risk of metabolic syndrome. However, longitudinal and interventional studies are needed to confirm causality and delineate the relative contributions of behavior, shared environment, and microbial transfer.

The microbial taxa most consistently associated with pet exposure, along with their corresponding functional roles and health associations, are summarized in Table 2. This overview illustrates the convergence of findings across independent studies and provides a framework for understanding the mechanistic links between pet ownership, microbiota composition, and health outcomes.

Translation to clinical practice

The cumulative evidence linking pet ownership to favorable microbiota composition, reduced immune-mediated disease risk, and improved metabolic health has important clinical and public health implications. Table 3 synthesizes evidence-based recommendations using the GRADE framework, which systematically evaluates the quality of evidence and strength of recommendations for specific clinical scenarios and target populations. The recommendations address prenatal and infant pet exposure, management of immunocompromised individuals in pet-owning households, and antimicrobial stewardship strategies to mitigate resistance transmission. These evidence-based guidelines underscore the household as a single epidemiological unit where human, animal, and environmental health intersect, a core fundamental of the One Health paradigm.

Conclusions and future directions

Living with a pet creates a profound and bidirectional change in the respective GMs of both species.

Several methodological limitations warrant consideration. Most studies are observational and cross-sectional, precluding causal inference. Confounding by socioeconomic status, outdoor exposure, diet, and hygiene practicesis incompletely controlled. Additionally, 16S rRNA sequencing, the predominant method, provides taxonomic resolution but limited functional insight. Pet type (dog *vs* cat), breed, diet, and health status are inconsistently reported, lowering comparability across studies.

Despite these limitations, the evidence strongly suggests that, while this two-way transfer carries some small risks of spreading pathogens and AMR genes, it also confers substantial health benefits. Specifically, living with pets, especially from a young age, enriches the gut with diverse microbial types and promotes the growth of beneficial bacteria such as *Ruminococcus* and *Oscillospira*.

Table 2. Summary of bacterial taxa reported across studies evaluating pet exposure and human microbiota.

Bacterial taxon	Direction of change with pet exposure	Phylum	Functional role	Associated health outcomes	Supporting studies
Ruminococcus spp.	Increased	Firmicutes	SCFA production, mucin degradation, ruminococcin A antimicrobial production	Reduced atopy risk, allergy protection, anti inflammatory effects	3,13,16
Oscillospira spp.	Increased	Firmicutes	SCFA production, bile acid metabolism	Negative association with obesity, reduced metabolic syndrome risk	3,13
Lactobacillus spp.	Increased	Firmicutes	Lactic acid production, immune modulation, pathogen inhibition	Gut barrier protection, immune regulation	3,16
Faecalibacterium spp.	Increased	Firmicutes	Butyrate production, anti inflammatory activity	Anti inflammatory effects, improved gut barrier function	16
Akkermansia muciniphila	Increased in adults	Verrucomicrobia	Mucin degradation, gut barrier integrity	Metabolic health, obesity protection, gut protection	8
Megamonas spp.	Decreased	Firmicutes Negativicutes	Carbohydrate fermentation	Association with metabolic disorders and NAFLD in children	3
Veillonella spp.	Decreased	Firmicutes Negativicutes	Lactate fermentation	Higher in non pet owners, unclear clinical role	3
Streptococcaceae	Decreased in specific contexts	Firmicutes	Mixed functions, includes pathogenic species	Possible reduction in inflammation in antibiotic exposed infants	13
Enterobacteriaceae	Decreased	Proteobacteria	Includes opportunistic pathogens	Reduction associated with lower inflammation	16
Bacteroides spp.	Variable	Bacteroidota	Polysaccharide degradation, vitamin synthesis	Essential commensal, context dependent effects	8
Prevotellaceae UCG 003	Decreased	Bacteroidota	Fiber degradation	Reduced in pet owners, unclear significance	3

SCFA, short-chain fatty acids.

Table 3. Clinical and public health recommendations derived from the GRADE framework based on available evidence on pet exposure, microbiota modulation, infection risk, and immune or metabolic outcomes.

Recommendation category	Target population	Specific recommendation	Quality of evidence GRADE	Strength of recommendation	Rationale and key evidence
Immune health	Families planning pregnancy or with young infants	Consider acquiring or maintaining pet ownership before or during pregnancy and through infancy	Moderate	Strong FOR	Large observational cohorts show prenatal and early exposure linked to higher *Ruminococcus* and *Oscillospira* and reduced atopy risk. Downgraded due to lack of RCTs. Strong biological plausibility *via* SCFA mediated immune training
Immune health	Families with infants at high genetic risk for allergic disease	Maintain pet in household during first year of life and avoid removal due to allergy concerns	Moderate	Strong FOR	Studies in at risk families show mini farm effect promotes immune tolerance. Increased animal specific Bifidobacterium and more diverse enterotypes reported. Consistent mechanistic pathway
Metabolic health	Adults at risk for metabolic syndrome or obesity	Pet ownership may provide metabolic benefits through microbiome modulation	Low	Weak or conditional FOR	Limited evidence. Cross sectional data show lower *Firmicutes Bacteroidetes* ratio and *Oscillospira* enrichment in owners. Minimal overall differences and no direct metabolic outcomes measured
Infection control	All pet owning households, especially with young children or elderly members	Practice regular hand hygiene after handling pet feces, cleaning litter boxes, or contact with sick pets	Moderate	Strong FOR	Genomic evidence shows bidirectional transmission of resistant Enterobacterales and clonal *E. coli* within households. Hand washing associated with lower antimicrobial resistance
Antimicrobial tewardship	Pet owners whose pets require antibiotic treatment and veterinarians	Minimize unnecessary antibiotic use in companion animals. Prefer narrow spectrum agents. Discuss alternatives with veterinarian	Modecrate	Strong FOR	Antibiotic treatment identified as key period for antimicrobial resistance transmission. High ESBL carriage in treated pets. Direct genomic evidence supports stewardship and One Health approach
Special populations	Households with immunocompromised members	Maintain benefits of pet ownership with enhanced hygiene. Individualize decisions with healthcare provider	Very low	Weak or conditional FOR	No direct evidence in immunocompromised populations. Recommendation based population, manageable infection risk with hygiene, and psychological support. Requires case by case assessment
Probiotic intervention	Families with dogs and young children, especially in allergy prevention	Consider canine specific probiotic supplementation as indirect intervention for child gut health	Very low	Weak FOR, research context only	Single proof of concept study shows dog probiotic associated with microbiota changes in children. Evidence not replicated. No long term clinical outcomes

These microbial changes are closely linked to reduced risk of immune-mediated diseases, including asthma and allergies, as well as metabolic conditions like obesity and metabolic syndrome. The household is a single ecological unit where the health of pets and people are linked, which is a perfect example of the "One Health" principle.

Even though these results are very intriguing, further research is warranted to definitively establish causality and comprehend the long-term stability of these pet-induced microbial alterations. Besides, future research should incorporate metagenomic and metabolomic analyses to transcend compositional descriptions and elucidate the functional implications of shared microbiomes.

Understanding this complex interspecies partnership at the microbial level deepens our appreciation of the human-animal bond while offering novel strategies to promote lifelong health in our households.

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Received: 3 March 2026; Accepted: 13 April 2026.

Contributions: Alberto Corriero, conceptualization, methodology, formal analysis, investigation, data curation, project administration, writing - original draft preparation. Mariateresa Giglio, conceptualization writing - original draft preparation, literature search. Giustino Varrassi, supervision, writing - review & editing. Filomena Puntillo, conceptualization, supervision. Lou'i AI-Husinat, Van Phong Pham, Y Van Tran, literature search. All authors have read and agreed to the published version of the manuscript.

Conflict of interest: The authors declare no conflict of interest, and all authors confirm accuracy.