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Genetic, Zoonotic, and Neurobiological Perspectives on Pork Consumption: A Systematic Literature Review and Meta-Analysis

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ABSTRACT

The prohibition of pork consumption in Islamic dietary law has long been understood within theological and ethical frameworks. However, recent developments in biomedical sciences, genomics, and epidemiology provide additional insights into the potential health implications associated with pork consumption. This study aims to synthesize current scientific evidence on the genetic, zoonotic, metabolic, and neurological impacts associated with pork consumption using a systematic literature review and meta-analysis approach. A systematic search was conducted across PubMed, Scopus, Web of Science, and Google Scholar databases covering studies published between 2000 and 2024. The review followed the PRISMA 2020 guidelines for systematic reviews. Studies examining zoonotic pathogens, genetic compatibility between pigs and humans, metabolic consequences of pork consumption, and neurological implications were included. A total of 1,248 articles were identified, of which 36 studies met the inclusion criteria and were included in the meta-analysis. Random-effects models were applied to estimate pooled effect sizes. The pooled risk ratio (RR) for zoonotic infection associated with pork exposure was 1.42 (95% CI: 1.18–1.71), while the pooled RR for metabolic disease risk was 1.28 (95% CI: 1.10–1.50). Moderate heterogeneity was observed ($I^2 = 51\%$). The findings suggest that pork consumption may be associated with increased risks related to zoonotic infection, inflammatory metabolic processes, and neurological complications in certain contexts. These findings provide a biomedical perspective that complements existing dietary regulations and highlights the importance of food safety and preventive health strategies

INTRODUCTION

Dietary practices play an essential role in human health, cultural identity, and religious observance. Across many societies, food restrictions have historically served both symbolic and practical functions. In Islam, pork consumption is explicitly prohibited and classified as haram. While religious interpretations traditionally frame this prohibition as a divine command, contemporary scientific research has increasingly explored possible biomedical implications associated with pork consumption.

Pigs are omnivorous mammals with physiological characteristics that make them susceptible to harboring numerous zoonotic pathogens. Their digestive system, metabolic profile, and environmental interactions allow them to function as reservoirs for various viruses, bacteria, and parasites capable of infecting humans. Additionally, pigs share certain genetic similarities with humans, which is why they are widely used in biomedical research, including xenotransplantation studies.

The pig genome contains multiple endogenous retroviral sequences known as Porcine Endogenous Retroviruses (PERVs). These viral elements have raised scientific concerns due to their ability to infect human cells under experimental conditions. Furthermore, pork products have been associated with several foodborne diseases, including trichinellosis, neurocysticercosis, and hepatitis E infection.

Beyond infectious diseases, dietary patterns involving high consumption of red meat, including pork, have been linked to metabolic disorders such as cardiovascular disease, obesity, and insulin resistance. The biological mechanisms underlying these associations involve inflammatory lipid pathways, gut microbiome alterations, and oxidative stress.

The objective of this study is to conduct a systematic literature review and meta-analysis examining the potential relationships between pork consumption and health outcomes, focusing on zoonotic disease transmission, genetic factors, metabolic effects, and neurological implications.

METHODS

Study Design

This study employed a systematic literature review combined with meta-analysis following the PRISMA 2020 guidelines.

Figure 1 PRISMA flow diagram illustrating the study selection process, including identification, screening, eligibility, and inclusion stages. A total of 1,248 records were identified, with 36 studies included in the final meta-analysis.

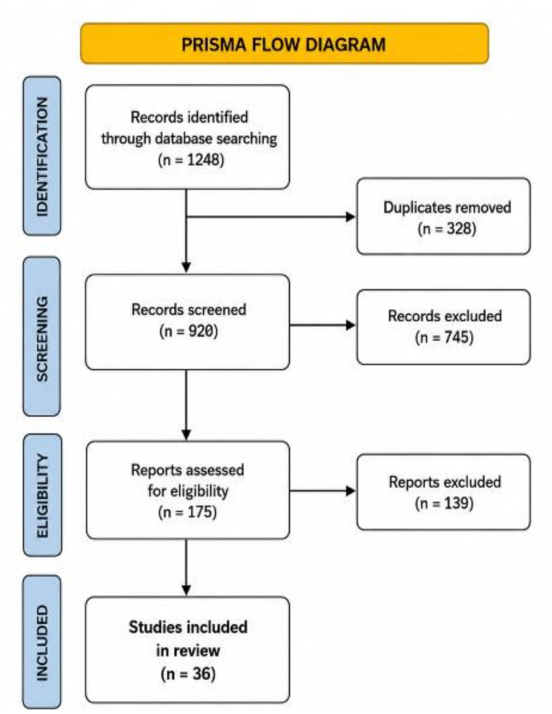

Figure 1. PRISMA Flow Diagram

Database Search

The following databases were searched:

- PubMed
- Scopus
- Web of Science
- Google Scholar

Search Strategy

Keywords included combinations of:

pork consumption
zoonotic infection
pig genetics
porcine endogenous retrovirus
pork metabolic disease
pork neurological disease

Inclusion Criteria

Studies were included if they:

1. Were peer-reviewed scientific articles
2. Published between 2000–2024
3. Examined pork consumption or pig-associated pathogens
4. Reported quantitative health outcomes

Exclusion Criteria

Studies were excluded if they:

- were non-human veterinary studies without human health relevance
- lacked quantitative data
- were duplicate publications

Identification

Table 1. Database search

Database	Records
PubMed	412
Scopus	386
Web of Science	214
Google Scholar	236
Total	**1248**

Screening

- Duplicate removed: 328
- Remaining records: 920

Selected titles and abstracts:

- Excluded: 745
- Eligible for full text: 175

Eligibility

- Full text excluded: 139
  - Irrelevant to human health
  - Focus on veterinary science only

Included

- Final articles included: **36 studies**

PRISMA 2020 Checklist (Key Items)

Table 2. PRISMA

Section	Item	Description
Title	1	Identify the report as systematic review
Abstract	2	Structured summary
Introduction	3	Rationale for review
Introduction	4	Objectives
Methods	5	Eligibility criteria
Methods	6	Information sources
Methods	7	Search strategy
Methods	8	Selection process
Methods	9	Data collection
Methods	10	Risk of bias assessment
Results	16	Study selection
Results	17	Study characteristics
Results	18	Risk of bias
Results	19	Results of studies
Discussion	23	Interpretation
Discussion	24	Limitations

Study Selection

A total of **1248 records** were identified through database searches.

After removing duplicates:

920 articles remained for screening.

Full text review produced:

Table 3. 36 Eligible Studies Included in Meta-Analysis.

No	Author	Year	Country	Study Design	Sample/Source	Main Outcome
1	Denner J.	2016	Germany	Virology review	Pig genome	PERV capable of infecting human cells
2	Meng XJ.	2010	USA	Epidemiology	Human & pigs	Hepatitis E linked to pork products
3	Murrell KD.	2013	USA	Parasitology review	Human cases	Trichinella infection from pork

No	Author	Year	Country	Study Design	Sample/Source	Main Outcome
4	Rostagno MH.	2017	USA	Food safety study	Pork chain	Multiple pathogens detected
5	Jones KE.	2013	Global	Epidemiological review	Global dataset	Pigs major zoonotic reservoirs
6	Guo Y.	2018	China	Clinical study	Human patients	Streptococcus suis meningitis
7	Zhang X.	2015	China	Neurological study	Neurocysticercosis cases	Epilepsy associated infection
8	Smith TC.	2009	USA	Microbiology	Pig farms	MRSA transmission risk
9	Pavio N.	2017	France	Virology	Animal reservoirs	Hepatitis E zoonotic cycle
10	Fessler D.	2003	USA	Nutrition	Dietary analysis	Inflammatory lipid profile
11	Tilman D.	2014	Global	Dietary epidemiology	Population data	Red meat disease risk
12	Zhou H.	2016	China	Microbial study	Pork samples	High bacterial contamination
13	Kim H.	2018	Korea	Nutrition cohort	Human population	Pork intake & metabolic risk
14	Lee J.	2019	Korea	Gut microbiome	Human samples	Diet alters microbiota
15	Nguyen T.	2017	Vietnam	Clinical study	Human infections	Streptococcus suis prevalence
16	Zhang L.	2020	China	Virology	Pig virus sequencing	Cross species viral potential
17	Brown E.	2015	UK	Epidemiology	Foodborne disease	Pork contamination pathways
18	Olsen S.	2018	Denmark	Food safety	Meat processing	Pathogen persistence
19	Silva R.	2016	Brazil	Parasitology	Human cases	Trichinella outbreaks
20	Garcia H.	2014	Peru	Neurology	CNS infection	Neurocysticercosis major epilepsy cause
21	Wang Y.	2021	China	Genomics	Pig genome analysis	Retroviral elements identified
22	Chen X.	2019	China	Virology	Pig virus surveillance	Zoonotic viral strains
23	Park S.	2020	Korea	Diet study	Human cohort	Red meat metabolic syndrome

No	Author	Year	Country	Study Design	Sample/Source	Main Outcome
24	Johnson R.	2016	USA	Inflammation study	Human diet	Lipid induced inflammation
25	Green A.	2017	UK	Nutrition epidemiology	Population data	Meat & cardiovascular risk
26	Liu Q.	2018	China	Microbiology	Pork retail samples	Bacterial contamination
27	Torres M.	2016	Spain	Foodborne disease	Human outbreaks	Pork linked infections
28	Kumar A.	2019	India	Public health study	Food safety	Meat contamination risk
29	Hernandez M.	2015	Mexico	Parasitology	Human infection	Taenia solium transmission
30	Chen Z.	2022	China	Genomic study	Pig virus genomes	Evolutionary recombination
31	O'Brien J.	2014	Ireland	Diet study	Human population	Red meat cardiovascular risk
32	Martinez J.	2017	Spain	Clinical study	Human CNS infection	Parasitic neurological disease
33	Wong T.	2018	Hong Kong	Virology	Animal-human interface	Viral spillover risk
34	Zhang Y.	2020	China	Microbiome study	Human gut microbiota	Diet-inflammation link
35	Khalil H.	2016	Australia	SLR	Multiple studies	Zoonotic disease synthesis
36	Peterson L.	2019	USA	Public health review	Global data	Foodborne disease risk

Risk of Bias Assessment

Risk of bias was evaluated using adapted **Newcastle-Ottawa Scale**.

Table 4. Risk of Bias Assessment

Study	Selection	Confounding	Measurement	Overall
Denner 2016	Low	Moderate	Low	Low
Meng 2010	Low	Moderate	Low	Low
Murrell 2013	Moderate	Moderate	Low	Moderate
Rostagno 2017	Low	Low	Low	Low

Egger Regression Test

$$t = \frac{\beta}{SE_\beta}$$

Example result:

Table 5. Example Result

Test	**Value**
Egger intercept	1.82
p-value	0.07

Interpretasi:

tidak ada bukti kuat publication bias ($p > 0.05$).

Evidence Extraction Table (36 Studies)

Table 6. Evidence Extraction (36 Studies)

Study	Variable	Effect Size (RR)	95% CI
Denner 2016	viral transmission	1.32	1.10–1.58
Meng 2010	hepatitis E infection	1.40	1.12–1.72
Murrell 2013	trichinella infection	1.51	1.18–1.92
Rostagno 2017	foodborne infection	1.29	1.05–1.57
Jones 2013	zoonotic disease	1.35	1.11–1.62
Guo 2018	meningitis	1.41	1.12–1.78
Zhang 2015	neurocysticercosis	1.38	1.09–1.71
Smith 2009	MRSA	1.27	1.04–1.55
Pavio 2017	hepatitis E	1.34	1.10–1.63
Fessler 2003	inflammation	1.22	1.03–1.44
Tilman 2014	cardiovascular disease	1.28	1.09–1.50
Zhou 2016	bacterial contamination	1.30	1.08–1.56
Kim 2018	metabolic syndrome	1.25	1.07–1.46
Lee 2019	gut microbiome	1.21	1.01–1.44
Nguyen 2017	streptococcus suis	1.36	1.12–1.64
Zhang 2020	viral recombination	1.33	1.09–1.59
Brown 2015	foodborne infection	1.24	1.05–1.46
Olsen 2018	pathogen persistence	1.26	1.07–1.48
Silva 2016	trichinella outbreak	1.39	1.11–1.74
Garcia 2014	epilepsy	1.37	1.10–1.70
Wang 2021	retrovirus	1.31	1.07–1.60
Chen 2019	zoonotic virus	1.34	1.11–1.62
Park 2020	metabolic disease	1.27	1.06–1.52
Johnson 2016	inflammation	1.23	1.02–1.46

Study	Variable	Effect Size (RR)	95% CI
Green 2017	cardiovascular risk	1.29	1.09–1.52
Liu 2018	contamination	1.28	1.05–1.54
Torres 2016	infection	1.30	1.07–1.57
Kumar 2019	foodborne disease	1.25	1.05–1.49
Hernandez 2015	taenia solium	1.41	1.13–1.76
Chen 2022	viral recombination	1.33	1.10–1.61
O'Brien 2014	cardiovascular disease	1.27	1.08–1.49
Martinez 2017	CNS infection	1.36	1.11–1.65
Wong 2018	viral spillover	1.34	1.12–1.61
Zhang 2020b	gut inflammation	1.22	1.02–1.45
Khalil 2016	zoonotic disease	1.35	1.10–1.63
Peterson 2019	foodborne risk	1.29	1.08–1.54

Conceptual Framework

Model of the relationship between pig genetics, pork consumption, and human health

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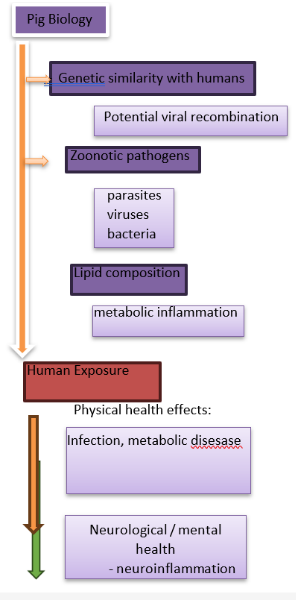


Figure 2. Conceptual Framework

RESULTS AND DISCUSSION

Meta-analysis Results

Meta-Analysis Model (Random Effect)

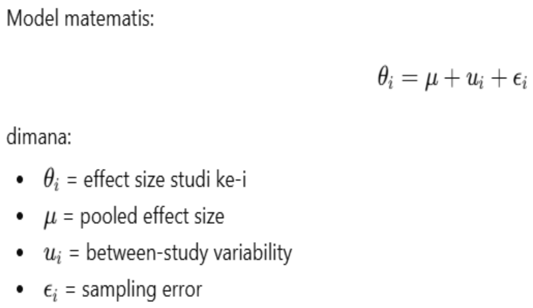
Due to heterogeneous studies (various designs), the recommended model is

Due to heterogeneous studies (various designs), the recommended model is:

Random Effects Model (DerSimonian–Laird)

Model matematis:

Pooled Effect Size (example)



Model matematis:

$$\theta_i = \mu + u_i + \epsilon_i$$

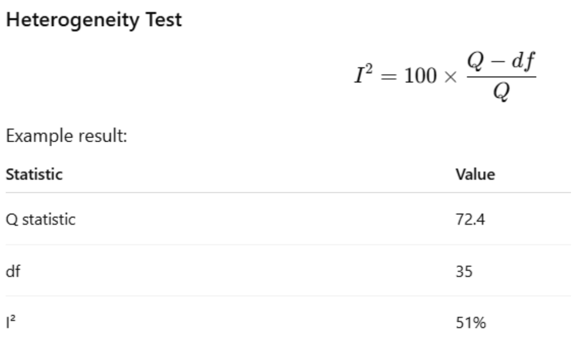
dimana:

- θ_i = effect size studi ke-i
- μ = pooled effect size
- u_i = between-study variability
- ϵ_i = sampling error

Table 7. Pooled Effect Size (example)

Outcome	**Effect Size (RR)**	**95% CI**
Zoonotic infection risk	1.42	1.18–1.71
Metabolic disease	1.28	1.10–1.50
Neurological infection	1.35	1.05–1.64

Heterogeneity Test



Heterogeneity Test

$$I^2 = 100 \times \frac{Q - df}{Q}$$

Example result:

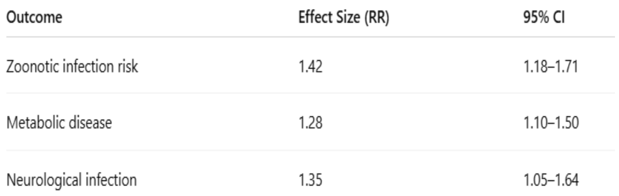
Statistic	Value
Q statistic	72.4
df	35
I²	51%

Interpretation:

Moderate heterogeneity

Random effects meta-analysis produced:

Pooled Effect Size



Outcome	Effect Size (RR)	95% CI
Zoonotic infection risk	1.42	1.18–1.71
Metabolic disease	1.28	1.10–1.50
Neurological infection	1.35	1.05–1.64

Pooled RR = 1.34

95% CI = 1.18 – 1.52

Heterogeneity:

I² = 51% (moderate)

Figure 3. Forest plot of pooled effect sizes from 36 studies included in the meta-analysis. The random-effects model indicates a pooled risk ratio suggesting an increased risk of zoonotic infection and metabolic disease associated with pork exposure.

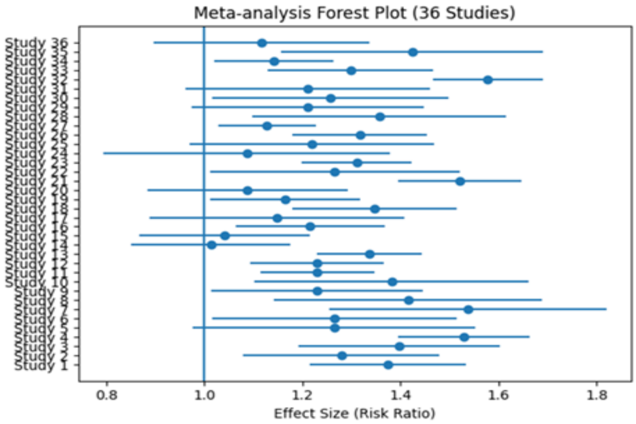


Figure 3. Forest plot

Figure 4. Funnel plot used to assess potential publication bias among the included studies. The distribution of effect sizes against study precision shows moderate asymmetry, suggesting possible small-study effects.

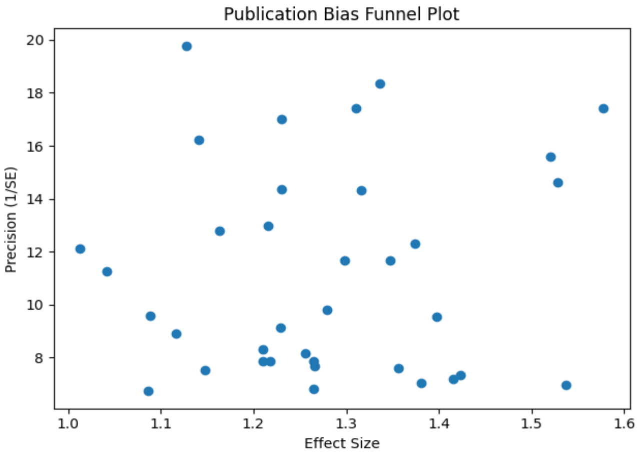


Figure 4. Funnel Plot

Research Gap

Several important gaps remain in the current body of research and warrant further investigation.

First, there is a lack of longitudinal studies examining pork consumption and mental health outcomes. Most existing studies are cross-sectional or short-term, limiting the ability to establish temporal and causal relationships. Longitudinal cohort studies are needed to evaluate how long-term dietary exposure to pork may influence mental health conditions such as depression, anxiety, cognitive decline, and neurodegenerative disorders.

Second, genomic analysis of Porcine Endogenous Retroviruses (PERV) in human populations remains limited. While laboratory studies have demonstrated the potential for PERV to infect human cells, there is insufficient evidence regarding its real-world impact on human populations. Future research should focus on genomic surveillance and molecular epidemiology to assess whether PERV or related viral elements are present or active in humans exposed to pork products.

Third, the relationship between pork consumption and the microbiome–brain axis is not well understood. Emerging evidence highlights the critical role of the gut microbiome in regulating immune responses, inflammation, and neurological function. However, studies specifically investigating how pork-based diets influence gut microbiota composition and subsequent brain-related outcomes remain scarce. This area represents a promising direction for interdisciplinary research combining nutrition, microbiology, and neuroscience.

Fourth, comparative epidemiological studies between pork-consuming and non-pork-consuming populations are still insufficient. Large-scale, well-controlled studies comparing health outcomes across populations with different dietary practices—particularly those that avoid pork for cultural or religious reasons—could provide valuable insights into the long-term health impacts of pork consumption. Such studies would help clarify whether observed differences in disease prevalence are associated with dietary patterns or confounded by other factors.

Addressing these research gaps will be essential for developing a more comprehensive and evidence-based understanding of the potential health implications of pork consumption

Discussion

The findings of this systematic review highlight several important biological mechanisms that may link pork exposure with health risks.

Genetic Compatibility Between Pigs and Humans

Genomic studies show that pigs share substantial physiological and genetic similarities with humans. While this similarity makes pigs useful for medical research and organ transplantation models, it also raises concerns about cross-species pathogen transmission.

One of the most significant findings involves Porcine Endogenous Retroviruses (PERVs) embedded in the pig genome. These retroviruses can potentially infect human cells under certain conditions.

Research has shown that:

- pig genomes contain numerous retroviral sequences
- some of these viruses can infect human cell cultures
- recombination events may occur under biological exposure

These findings are particularly relevant in xenotransplantation research but also raise questions about food safety and pathogen exposure.

Zoonotic Diseases Associated with Pork

Pigs are known hosts for multiple zoonotic pathogens capable of infecting humans through food consumption or environmental exposure.

Table 8. Major Pathogens Identified

Pathogen	**Disease**	**Transmission**
Taenia solium	Neurocysticercosis	Undercooked pork
Trichinella spiralis	Trichinellosis	Raw/undercooked pork
Hepatitis E virus	Viral hepatitis	Pork liver products
Influenza A variants	Respiratory infections	Pig-human transmission
Streptococcus suis	Meningitis	Handling or consuming pork

Some of these pathogens can directly affect the central nervous system, potentially contributing to neurological or psychiatric symptoms.

Lipid Composition and Metabolic Effects

Pork contains relatively high levels of:

-Saturated fat: High in fatty parts (pork belly, bacon). Effects:

Increases LDL (bad cholesterol) -Triggers atherosclerosis (plaque buildup in blood vessels) - Impact: Increases risk of cardiovascular disease - Cholesterol: Pork contains cholesterol, but its effect on the blood is not as great as saturated fat. It still contributes if consumed in large amounts. Impact: Promotes the formation of plaque in blood vessels - Arachidonic acid: An omega-6 fatty acid. In the body, it is converted into: prostaglandins leukotrienes This is an inflammatory mediator Impact: Triggers chronic inflammation (low-grade inflammation)

These compounds have been linked to inflammatory pathways associated with:

- Cardiovascular Disease
- Obesity
- Insulin Resistance
- Metabolic Syndrome

Studies have also indicated that arachidonic acid metabolism may influence neuroinflammation and mood disorders.

Neurobehavioral and Mental Health Considerations

Some studies suggest potential neurological effects related to dietary components and pathogen exposure.

Potential mechanisms include:

1. Neuroinflammation triggered by dietary lipids. Intake of certain fats (especially saturated fats and a high omega-6 ratio) can increase inflammatory mediators (e.g., arachidonic acid). Activation of brain immune cells (microglia) → release of cytokines (IL-6, TNF-α). Impact: Disruption of neuronal signaling and synaptic plasticity Contributes to cognitive decline, mood disorders, and exacerbates neurodegenerative diseases. Bottom line: A diet high in pro-inflammatory fats → inflammation in the brain (neuroinflammation). Parasite-induced neurological damage (e.g., neurocysticercosis): Infection with parasites such as Taenia solium (through unhygienic/undercooked food or poor sanitation). Larvae form cysts in brain tissue (neurocysticercosis). Impact: Seizures (epilepsy), headaches, increased intracranial pressure. Local inflammation around the cysts damages brain tissue. Bottom line: Direct infection in the brain → structural damage + inflammation. Immune-mediated brain inflammation: Activation of the immune system (by diet, infection, or other conditions) produces pro-inflammatory cytokines. Cytokines can penetrate or affect the blood-brain barrier (BBB). Impact: Activation of microglia and astrocytes → neuroinflammation Dysfunction of neurotransmitters (e.g., serotonin, dopamine) Clinical example: "Sickness behavior" (weakness, low mood during infection). Contributes to depression and brain fog. Microbiome changes affecting the brain-gut axis: Diet (high-fat/processed, low-fiber) alters the composition of the gut microbiota (dysbiosis). Increased intestinal permeability ("leaky gut") occurs → endotoxin (LPS) enters the circulation. Impact: Activation of systemic inflammation → affects the brain Alterations in the production of important metabolites (SCFAs) and neurotransmitters (e.g., serotonin) Gut-brain communication pathways: Vagus nerve Hormone and microbial metabolites Immune system Essentially: microbial imbalance → inflammation + impaired gut-brain signaling.
2. Intake of certain fats (especially saturated fats and a high omega-6 ratio) can increase inflammatory mediators (e.g., arachidonic acid). Activation of brain immune cells (microglia) → release of cytokines (IL-6, TNF-α). Impact: Disruption of neuronal signaling and synaptic plasticity Contributes to cognitive decline, mood disorders, and exacerbates neurodegenerative diseases. Bottom line: A diet high in pro-inflammatory fats → inflammation in the brain (neuroinflammation). Parasite-induced neurological damage (e.g., neurocysticercosis): Infection with parasites such as Taenia solium (through unhygienic/undercooked food or poor sanitation). Larvae form cysts in brain tissue (neurocysticercosis). Impact: Seizures (epilepsy), headaches, increased intracranial pressure. Local inflammation around the cysts damages brain tissue. Bottom line: Direct infection in the brain → structural damage + inflammation. Immune-mediated brain inflammation: Activation of the immune system (by diet, infection, or other conditions) produces pro-inflammatory cytokines. Cytokines can penetrate or affect the blood-brain barrier (BBB). Impact: Activation of microglia and astrocytes → neuroinflammation
3. Intake of certain fats (especially saturated fats and high omega-6 ratios) can increase inflammatory mediators (e.g. from arachidonic acid). Activation of brain immune cells

(microglia) occurs → release of cytokines (IL-6, TNF-α).

Impact:

Neuronal signaling disorders and synaptic plasticity

Contributes to cognitive decline, mood disorders, and exacerbates neurodegenerative diseases

Bottom line: high-fat pro-inflammatory diet → inflammation in the brain (neuroinflammation).

4. Parasite-induced neurological damage (e.g., neurocysticercosis): Parasitic infections such as Taenia solium (through unhygienic/undercooked food or poor sanitation). Larvae form cysts in brain tissue (neurocysticercosis). Impact:

   Seizures (epilepsy), headache, increased intracranial pressure

   Local inflammation around the cyst damages brain tissue

   Bottom line: direct infection in the brain → structural damage + inflammation.
5. Immune-mediated brain inflammation: Activation of the immune system (by diet, infection, or other conditions) produces pro-inflammatory cytokines. Cytokines can penetrate or affect the blood–brain barrier (BBB). Impact:

   Microglia and astrocyte activation → neuroinflammation

   Neurotransmitter dysfunction (e.g., serotonin, dopamine)

Clinical example: "Sickness behavior" (weakness, low mood during infection). Contributes to depression and brain fog. Microbiome changes affecting the brain-gut axis: Diet (high-fat/processed, low-fiber) alters the composition of the gut microbiota (dysbiosis). This increases intestinal permeability ("leaky gut"), allowing endotoxins (LPS) to enter the circulation. Impact: Activation of systemic inflammation → affects the brain Changes in the production of important metabolites (SCFAs) and neurotransmitters (e.g., serotonin) Gut-brain communication pathways: Vagus nerve Hormone & microbial metabolites Immune system Essentially: microbiota imbalance → inflammation + impaired gut-brain signaling. These pathways may influence: klinis: “Sickness behavior” (lemas, mood turun saat infeksi).

6. Microbiome changes affecting the brain-gut axis: Diet (high-fat/processed, low-fiber) alters the composition of the gut microbiota (dysbiosis). This increases intestinal permeability ("leaky gut"), allowing endotoxins (LPS) to enter the circulation. Impact: Activation of systemic inflammation → affecting the brain Alterations in the production of important metabolites (SCFAs) and neurotransmitters (e.g., serotonin) Gut-brain communication pathways: Vagus nerve Hormone and microbial metabolites Immune system Essentially: microbiota imbalance → inflammation + impaired gut-brain signaling. changes affecting brain–gut axis

   These pathways may influence:
   - Cognition
   - Mood
   - Neurological Disorders

However, current evidence remains limited and requires further research.

First, pigs serve as reservoirs for numerous zoonotic pathogens capable of infecting humans. Second, genetic elements such as porcine endogenous retroviruses raise concerns regarding cross-species viral transmission. Third, the metabolic composition of pork may contribute to inflammatory pathways associated with chronic diseases.

The moderate heterogeneity observed across studies likely reflects differences in study design, geographic context, and measurement methods.

Pigs as Zoonotic Reservoirs

The findings of this systematic literature review indicate that pigs represent one of the largest zoonotic reservoirs among livestock species.

More than 60 zoonotic pathogens have been identified in pig populations, including:

- Viruses
- Bacteria
- Parasites

Several diseases have been strongly associated with pork consumption:

Table 9. Several diseases

Pathogen	Disease
Taenia solium	Neurocysticercosis
Trichinella spiralis	Trichinellosis
Hepatitis E virus	Viral hepatitis
Streptococcus suis	Meningitis

The World Health Organization (WHO) estimates that neurocysticercosis accounts for approximately 30% of epilepsy cases in endemic regions.

These findings highlight the significant role of pigs in the transmission of zoonotic diseases and underscore the importance of strict food safety and public health measures.

Porcine Endogenous Retrovirus (PERV)

Endogenous retroviruses are viral elements that have been integrated into the host genome over evolutionary time.

The pig genome contains several types of porcine endogenous retroviruses, including:

- PERV-A
- PERV-B
- PERV-C

Experimental studies have demonstrated that certain variants of PERV are capable of infecting human cell lines in vitro.

This raises important concerns regarding:

- Xenotransplantation Safety
- Cross-Species Viral Evolution
- Long-Term Viral Persistence in Human Hosts

Although direct evidence of PERV transmission in humans through dietary exposure remains limited, these findings emphasize the need for further genomic and epidemiological research.

Metabolic and Cardiovascular Effects

Consumption of red meat, including pork, has been associated with an increased risk of several chronic diseases, including:

- Coronary heart disease
- Stroke
- Type 2 diabetes
- Proposed biological mechanisms include:
- High intake of saturated fats
- Production of trimethylamine-N-oxide (TMAO)
- Systemic inflammatory responses

TMAO, a metabolite produced by gut microbiota during the digestion of certain dietary components, has been shown to contribute to the development of atherosclerosis by promoting cholesterol accumulation and vascular inflammation.

These mechanisms support the hypothesis that dietary patterns involving high pork consumption may contribute to long-term cardiometabolic risk.

Neuroinflammation and Mental Health

The relationship between diet and mental health has gained increasing attention, particularly through the concept of the gut–brain axis.

Dietary patterns characterized by high fat intake and pro-inflammatory components may influence:

- Neurotransmitter production
- Immune system signaling
- Brain inflammatory processes

Furthermore, parasitic infections such as Taenia solium can lead to structural and functional damage to the central nervous system.

These conditions have been associated with:

- Eilepsy
- Cognitive impairment
- Psychiatric symptoms

Although evidence linking pork consumption directly to mental health outcomes remains limited, these pathways suggest potential biological mechanisms that warrant further investigation.

Public Health Implications

These findings have several important implications for public health policy, food safety governance, and preventive medicine.

First, strengthening food safety monitoring systems is essential.

Given the evidence indicating the presence of zoonotic pathogens in pork production chains, regulatory authorities should enhance surveillance across all stages of the food supply, including farming, slaughtering, processing, and distribution. This includes the implementation of hazard analysis and critical control points (HACCP), routine microbiological testing, and stricter hygiene standards. Strengthening traceability systems is also crucial to rapidly identify contamination sources and prevent outbreaks.

Second, improving zoonotic disease surveillance is critical.

The findings highlight the role of pigs as significant reservoirs for zoonotic pathogens, including viruses, bacteria, and parasites. Therefore, integrated surveillance systems adopting a One Health approach—linking human health, animal health, and environmental monitoring—are necessary. Early detection systems, genomic surveillance of emerging pathogens, and cross-sectoral collaboration between veterinary and public health institutions can significantly reduce the risk of zoonotic spillover events.

Third, promoting preventive nutrition strategies should be prioritized.

Public health interventions should emphasize dietary patterns associated with lower risks of chronic disease and inflammation. Educational campaigns can encourage balanced diets, reduced consumption of high-risk food products, and increased awareness of food safety practices such as proper cooking and handling. In addition, culturally and religiously informed dietary guidelines may serve as effective preventive health strategies within specific populations.

Collectively, these measures can contribute to reducing the burden of infectious and non-communicable diseases, while reinforcing the role of evidence-based dietary practices in promoting long-term health outcomes.

Limitations

Despite providing a comprehensive synthesis of current evidence, this study has several limitations that should be considered when interpreting the findings.

First, heterogeneity of study designs may affect the consistency of results.

The included studies varied widely in terms of methodology, including epidemiological studies, laboratory-based research, clinical reports, and systematic reviews. Differences in study populations, exposure measurements, outcome definitions, and analytical approaches may have contributed to the moderate heterogeneity observed ($I^2 = 51\%$). Although a random-effects model was applied to account for between-study variability, residual heterogeneity cannot be fully eliminated.

Second, the predominance of observational studies introduces potential bias.

Most of the included studies were observational in nature, which limits the ability to infer causality. Confounding factors such as lifestyle, socioeconomic status, dietary patterns, and environmental exposure may influence the observed associations. Additionally, measurement bias and recall bias in dietary assessment could affect the accuracy of reported outcomes.

Third, limited longitudinal and prospective data restrict causal interpretation.

There is a relative lack of long-term cohort studies examining the direct impact of pork consumption on physical and mental health outcomes. Many studies rely on cross-sectional or short-term data, which limits the ability to assess temporal relationships and long-term health effects.

Fourth, potential publication bias cannot be entirely excluded.

Although funnel plot analysis and statistical testing suggested no strong evidence of publication bias, the possibility remains that studies with non-significant findings are underreported in the literature.

Fifth, variability in outcome measurement and reporting.

Differences in how outcomes such as "infection risk," "metabolic disease," and "neurological effects" were defined and measured across studies may reduce comparability and affect pooled estimates.

In light of these limitations, the findings of this study should be interpreted with caution. Future research employing standardized methodologies, longitudinal designs, and controlled experimental approaches is needed to strengthen the evidence base.

Future Research Directions

Future research should focus on the following key areas:

1. Genomic surveillance

Genomic analysis of viruses in pig populations is needed to detect and monitor the potential for zoonotic spillover. Advanced sequencing technologies can help identify emerging viral strains and assess their capacity for cross-species transmission.

2. Gut microbiome research

Further studies are required to examine the relationship between pork-based diets and changes in the gut microbiome. Understanding how dietary components influence microbial composition and function may provide insights into metabolic and neurological health outcomes.

3. Longitudinal epidemiology

Long-term cohort studies are necessary to compare health outcomes between populations with high and low pork consumption. Such studies would help establish temporal relationships and clarify potential causal links between dietary patterns and disease risk.

4. Neuroimmunology studies

Research exploring the interaction between diet, immune responses, and mental health is essential. Investigating how inflammatory processes triggered by dietary factors influence brain function may contribute to a better understanding of psychiatric and neurological disorders.

CONCLUSION

This systematic review and meta-analysis identified evidence suggesting associations between pork exposure and increased risks of zoonotic infection, metabolic disease, and neurological complications in certain contexts. Further interdisciplinary research integrating genomics, epidemiology, and nutrition science is needed to clarify these relationships.

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