

Epidemiology and risk factors for Human Rhinovirus infections and severe outcomes in Burkina Faso: A cross-sectional analysis of sentinel surveillance data, 2016-2019

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Abstract

Background

Human rhinovirus (HRV) is a significant contributor to severe acute respiratory infections (SARI). We aimed to identify factors associated with HRV infection and HRV infection with severe outcomes using Burkina Faso country-wide SARI surveillance data.

Methods

A cross-sectional study was conducted from October 2016 to April 2019, using data from four hospitals in Burkina Faso that were enrolled in the SARI sentinel surveillance program. Hospitalised children and adult patients fulfilling the 2014 WHO SARI definition (acute respiratory infection with fever ($\geq 38^{\circ}\text{C}$) and cough < 10

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days) were included, with specimens collected and tested for HRV and other pathogens using multiplex real-time RT-PCR. Demographic, clinical, and laboratory data were analysed using descriptive statistics and logistic regression.

Results

Among 1,540 enrolled patients, children under four years comprised the majority (49.3%). Most participants were from rural areas, with a predominance of male enrollment. HRV prevalence ranged from 29.8% to 38.1% in different sentinel sites. Young age, urban residence, early consultation (within 2 days), chronic conditions, and viral-bacterial coinfections were independently associated with an increased risk of HRV infection. Severe outcomes were significantly associated with site location and specific viral and bacterial coinfections, especially with Adenovirus, *Haemophilus influenzae*, *Haemophilus influenzae* type b, and any bacterial coinfection. Influenza A and viral coinfection alone appeared protective against severe outcomes.

Conclusion

HRV represents an important etiologic agent of SARIs in Burkina Faso. Identifying demographic and clinical predictors such as young age, coinfection status, and geographical patterns can guide targeted interventions and improve outcomes in high-risk populations, especially in resource-limited settings.

Keywords: Human rhinovirus, Severe acute respiratory infections, severe outcomes, risk factors, Burkina Faso

Épidémiologie et facteurs de risque des infections à rhinovirus humain et de leurs formes graves au Burkina Faso : analyse transversale des données de surveillance sentinelle, 2016-2019

Résumé

Contexte

Le rhinovirus humain (HRV) est une cause importante des infections respiratoires aiguës sévères (IRAS). Cette étude vise à identifier les facteurs associés à l'infection et à la gravité des formes de HRV en analysant les données nationales de surveillance des IRAS au Burkina Faso.

Méthodes

De 2016 à 2019, une étude transversale a été menée dans quatre sites sentinelles, incluant des enfants et des adultes hospitalisés atteints d'IRAS selon la définition de l'OMS (fièvre ≥ 38 °C et toux < 10 jours). Les échantillons ont été testés par RT-PCR multiplex pour HRV et d'autres agents pathogènes. Les facteurs sociodémographiques, cliniques et biologiques ont été étudiés à l'aide de statistiques descriptives et d'une régression logistique.

Résultats

Parmi les 1540 patients enrôlés, les enfants de moins de 5 ans représentaient 49,3 % et 56,3 % étaient de sexe masculin. La prévalence de HRV était comprise entre 29,8 % et 38,1 % selon les sites. Les facteurs de risque d'infection à HRV comprenaient le jeune âge, la résidence en zone urbaine, la consultation précoce (≤ 2 jours), la présence de maladies chroniques et les co-infections virales ou bactériennes. Les facteurs associés aux formes graves comprenaient certains sites et des co-infections spécifiques, notamment avec l'adénovirus, *Haemophilus influenzae* et *Haemophilus*

influenzae type b. En revanche, la grippe A et certaines co-infections virales étaient des facteurs protecteurs contre la gravité.

Conclusion

Le HRV constitue un agent étiologique majeur des IRAS au Burkina Faso. L'identification des facteurs de risque nécessite des actions ciblées et l'amélioration de la prise en charge des populations à risque, notamment dans des contextes de ressources limitées.

Mots-clés : Rhinovirus humain, infections respiratoires aiguës sévères, formes graves, facteurs de risque, Burkina Faso

Background

Acute respiratory infections (ARIs) remain a leading cause of morbidity and mortality worldwide, particularly affecting vulnerable populations such as young children and the elderly (1). Among the viral agents responsible, human rhinovirus (HRV) is traditionally recognised as the primary cause of the common cold (2). It is, along with other viral respiratory infections, a significant public health concern in many countries because it represents a heavy financial burden on health systems worldwide (3). However, its role in more severe respiratory infections has often been underestimated. Recent findings have expanded the understanding of HRV beyond mild upper respiratory tract infections (4). HRV is now recognised as an important etiologic agent in severe lower respiratory tract infections (LRTIs), including bronchiolitis, pneumonia, and exacerbations of wheezing and asthma, especially in pediatric populations (5-7).

The virus belongs to the genus *Enterovirus* within the *Picornaviridae* family and comprises small, non-enveloped, single-stranded RNA viruses. To date, nearly 170 genotypes have been identified, classified into three species: rhinovirus A, B, and C (8,9). Transmission of HRV occurs primarily through direct exposure to respiratory secretions by inhaling droplets from coughing or sneezing, and by contacting contaminated surfaces or hands. Clinically, HRV infections present a broad spectrum of manifestations, ranging from upper respiratory conditions such as sinusitis and sore throat to more severe lower respiratory illnesses like bronchitis, bronchiolitis, and pneumonia (10-12). In addition, HRV infections are implicated in triggering and exacerbating asthma and wheezing episodes in both children and adults. Risk factors associated with severe disease outcomes in HRV infections include individual and familial atopic predispositions, environmental factors such as pollution and tobacco smoke exposure (13-16). It is

important to note that HRV can be detected in 12 to 22% of asymptomatic individuals, highlighting the potential for asymptomatic carriage and questioning the direct causal role of HRV in respiratory symptoms, especially when co-pathogens are present (17, 18).

In sub-Saharan Africa, including Burkina Faso, the burden of HRV and other respiratory viral infections is poorly documented, leading to underdiagnosis and a limited understanding of their impact, especially in severe cases. Prevalence studies in the region report variable detection rates of HRV among patients with respiratory infections: 58.2% in South Africa (19), 33.7% in Senegal (20), 44.1% in Kenya (21), 9.4% in Niger (22), and 18.6% in Ethiopia (23). In Burkina Faso, few studies have focused explicitly on HRV as a leading cause of respiratory illnesses; prevalences of 29.8% (24) and 38.1% (25) were found in hospital-based studies. It remains essential not only to determine the prevalence of HRV in different contexts, but also to investigate the risk factors for infection and severe outcomes, especially among at-risk groups in middle and low-income settings such as Burkina Faso.

This study aims to study the epidemiology and identify predictors of HRV infections and severe outcomes associated with HRV infections among patients with acute respiratory infections in Burkina Faso, using historical data from the national Severe Acute Respiratory Infection (SARI) surveillance system collected during the pre-COVID-19 period.

I. Methods

SARI surveillance organization, study design, sites, and data collection

A cross-sectional study was conducted within the existing SARI sentinel surveillance framework in Burkina Faso from October 2016 to April 2019. Four hospitals representing diverse geographic regions (National Teaching Hospital of Bogodogo (located in Ouagadougou), District Hospitals of Boussé, Kongoussi, and Houndé) were selected based on patient volume, geographic coverage, accessibility, and staff willingness to participate. Trained health workers enrolled patients meeting the 2014 WHO SARI case definition: acute respiratory infection with fever ($\geq 38^{\circ}\text{C}$) and cough within 10 days requiring hospitalisation (27). Oropharyngeal and/or nasopharyngeal specimens were collected, stored at $4-8^{\circ}\text{C}$, and transported within 48 hours to the national reference laboratory for testing. Socio-demographic and clinical data were recorded using standardised case report forms.

Study participants and variables definition

We included patients of all ages who met the SARI case definition. Children were excluded if parental or legal guardian consent was not obtained or if their medical condition prevented specimen collection. Patients without complete laboratory results or unknown discharge status were also excluded.

An HRV infection was defined as any patient fulfilling the inclusion criteria with a positive laboratory test for HRV from oropharyngeal or nasopharyngeal specimens.

Severe HRV-associated infection was defined as HRV cases with fatal outcomes or requiring intensive care due to danger signs such as dyspnea, lethargy, convulsions, stridor, inability to eat or drink, intercostal indrawing, or oxygen saturation below 90%.

Among viral SARI cases, hospital stays were considered prolonged if the length of stay was equal to or greater than the study mean of seven days; otherwise, the stay was considered normal.

Viral codetection: HRV cases with concomitant detection of at least one other virus in the same episode of disease during the laboratory testing

Bacterial codetection: HRV cases with concomitant detection of at least one bacterium in the same episode of disease during laboratory testing

Virus and bacterial co-detection: coexistence of detection of at least one virus and one bacterium during laboratory testing.

Laboratory investigations

The laboratory testing procedure was already described in a previous published article by Cissé *et al.*,(28). Briefly, the national influenza reference laboratory of Burkina Faso tested oropharyngeal and/or nasopharyngeal specimens from enrolled patients using the FTD-33 multiplex real-time RT-PCR kit (Fast Track Diagnostics). According to the manufacturer's specifications, the assay allows the detection of 21 viruses, 11 bacteria, and one fungus, including human rhinovirus, influenza A/B/C, parainfluenza viruses, coronaviruses, respiratory syncytial virus, adenovirus, Enterovirus, and common bacterial respiratory pathogens such as *Mycoplasma pneumoniae* and *Streptococcus pneumoniae*. Specimens were processed following standardised protocols to identify a broad range of respiratory pathogens.

Statistical analysis

Data entry was performed with Epi-Info™ version 7.2.1.0 (CDC, Atlanta), while data cleaning and analysis were conducted using STATA® version 17. Descriptive statistics summarised participant characteristics. Seasonal trends of HRV cases over two full years (2017-2018) were visualised using epidemic curves generated in Microsoft Excel®. Bivariate analyses examined associations between outcomes (*HRV positivity* and *severe HRV-associated cases*) and independent variables using Chi-square tests. Univariable logistic regression identified factors linked to HRV infection and severity. Variables with p-values ≤ 0.2 were included in multivariable logistic regression models via forward stepwise selection. Multicollinearity was assessed and addressed using variance inflation factors. Both crude and adjusted odds ratios with 95% confidence intervals were reported.

Ethical considerations

The data used in this study were obtained from the Burkina Faso SARI sentinel surveillance program, implemented by the Ministry of Health as a public health initiative in accordance with national laws and regulations (Law No. 23/94/ADP and ARRET No. 2023-83/MSHP/CAB/PM/MSHP). Ethical approval was waived by the national ethics committee given the public health nature of the program. Verbal informed consent was obtained from parents or legal guardians of enrolled children. All procedures adhered to the Helsinki Declaration and relevant ethical guidelines (26).

II. Results

Demographic characteristics of the study population

The demographic and clinical characteristics of the study population, as summarised in **Table 1**, provide an overview of the 1,540 participants involved in the study. Children aged 1 to 4 years were the most significant group, making up 49.3% of the population, followed by infants aged 4-11 months (25.0%) and those aged 0-3 months (11.2%). Participants aged 50 years and older accounted for only 3.7%. The majority of the participants were from rural areas (56.6%), while sex distribution showed a higher enrollment of males (56.3%).

Analysis by season revealed that most cases were enrolled during the dry season (71.3%). Patterns of health-seeking behaviour also varied in the study population: 46.2% of individuals consulted within two days

of symptom onset (early consultation), 38.4% had a medium delay (3-5 days), and 15.3% presented late (after six days). Pre-hospital antibiotic use was reported by 60.8% of patients, while 98.6% received antibiotics during hospitalisation, reflecting common management practices for respiratory illnesses in resource-limited settings. Only 2.7% of the participants had a documented chronic health condition, such as sickle cell disease, asthma, diabetes, or obesity. Prolonged hospital stay (>7 days) was noted in 21.4% of the study population, while severe outcomes were reported in 28.9% of the total study population.

Patterns of rhinovirus infection

Figure 1 displays weekly rhinovirus case trends for 2017 and 2018 alongside the total number of respiratory specimens collected per epi-week. Both years show modest, fluctuating rhinovirus activity throughout the two-year period, with no pronounced seasonal peak. Despite higher overall specimen collection early in the year, rhinovirus detection remained stable with limited differences between years, suggesting stable rhinovirus circulation patterns, and overall incidence appears not to be strongly influenced by variations in sampling effort.

Factors associated with Human Rhinovirus infection

A detailed analysis presented in Table 2 highlights the key demographic and clinical risk factors associated with human rhinovirus (HRV) infection. Children aged 0-3 months, 4-11 months, and 1-4 years demonstrated significantly higher odds of HRV infection compared to older age groups, with adjusted odds ratios (aORs) ranging from 3.0 to 3.9 ($p < 0.05$). Additionally, urban residency was associated with an increased risk of HRV infection (aOR: 1.6, $p < 0.001$) compared to rural areas. Seasonal trends suggest a higher prevalence of HRV during the rainy season in the univariable analysis, although this was not significant after adjustment.

Other notable risk factors included delayed consultation (higher odds for earlier consultation, aOR: 1.7 reference, $p=0.006$), viral-bacterial coinfection (aOR: 13.3, $p < 0.001$), and absence of viral coinfection, which appeared protective (aOR=0.12, $p<0.001$). Chronic health conditions were found to increase the odds of HRV infection (aOR: 2.3, $p = 0.048$), though these were rare in the sample. Severe disease and antibiotic exposure, both before and during hospitalisation, did not significantly alter HRV risk in the adjusted models.

Risk factors for Human Rhinovirus infection with severe outcomes

When assessing factors associated with severe outcomes among HRV-infected individuals, multivariate analysis indicated that the location of the site (notably Houndé, aOR: 2.72, $p < 0.001$), coinfections with viral pathogens such as enterovirus (aOR: 3.20, $p < 0.001$), adenovirus (aOR: 1.95, $p = 0.005$), and bacterial pathogens such as *Haemophilus influenzae* (aOR: 1.62, $p = 0.004$), and *Haemophilus influenzae* type B (aOR: 6.2, $p < 0.001$) were positively associated with adverse outcomes. Existence of at least one Bacterial coinfection (aOR: 9.18, $p < 0.001$) also markedly increased the risk of severe disease, underscoring the multifactorial nature of prognostic factors in HRV infection. Conversely, viral coinfection alone was associated with decreased odds of severe outcome (aOR: 0.20, $p < 0.001$) (Table I).

Table I: Demographic and clinical characteristics of the study population

	Total number N=1540	Percentage (%)
Age groups		
0-3 months	172	11.2
4-11 months	384	25.0
1-4 years	757	49.3
5-14 years	100	6.5
15-49 years	65	4.2
50 and more	57	3.7
Residency		
Urban	668	43.4
Rural	872	56.6
Sex		
Male	867	56.3
Female	673	43.7
Season¹		
Dry season	1,097	71.3
Rainy season	441	28.7
Time spent before consultation		
Early consultation (≤ 2 days)	711	46.2
Medium consultation(3-5 days)	591	38.4
Late consultation (≥ 6 days)	236	15.3

	Total number N=1540	Percentage (%)
Malnutrition		
No	1,436	93.3
Yes	104	6.7
Received antibiotics before admission		
No	603	39.2
Yes	937	60.8
Received antibiotics during hospitalisation		
No	21	1.4
Yes	1,519	98.6
Chronic health condition²		
No	1,499	97.3
Yes	41	2.7
Outcome		
Discharged	1177	76.43
Dead	26	1.7
Unknown Outcome/Transferred to another hospital	337	21.9
Prolonged hospital stay		
No (<7 days)	1,211	78.6
Yes (7 days and more)	329	21.4
Severe Outcomes³		
No	1,095	71.1
Yes	445	28.9
Sites		
Bogodogo	378	24.6
Boussé	228	14.8
Kongoussi	466	30.3
Houndé	468	30.4

1:dry season = November-April; rainy season= May-October—2: sickle cell disease, asthma, diabetes, obesity, and other medical chronic conditions —3:severe outcomes included: death, hospitalisation in ICU,

Table II: Demographic and clinical factors associated with HRV infection

Variables	Human Rhinovirus-associated infections				
	Prevalence n (%HRV)	Crude OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
Age group					
0-3 months	62(36.1)	3.4(1.5-7.7)	0.003**	3.9 (1.6-9.8)	0.003**
4-11 months	146(38)	3.7(1.7-8.2)	0.001**	3.9(1.6-9.4)	0.002**
1-4 years	254(33.5)	3.1(1.4-6.6)	0.004**	3(1.3-7.1)	0.012*
5-14 years	22(22)	1.7(0.7-4.2)	0.226	1.9(0.7-5)	0.197
15-49 Years	9(13.8)	1(0.3-2.7)	0.976	0.9(0.3-2.8)	0.885
50 Years and more	8(14)	1(ref.)	----	1(ref.)	
Area of residence					
Urban	219(32.8)	1(0.8-1.2)	0.891	1.6(1.2-2.1)	0.001**
Rural	283(32.4)	1(ref.)	---	1(ref.)	---
Sex					
Male	295(34)	1(ref.)	---	1(ref.)	---
Female	207(30.8)	0.9(0.7-1.1)	0.175	0.8(0.6-1.1)	0.139
Season ¹					
Dry season	337(30.7)	1(ref.)	--	1(ref.)	---
Rainy season	164(37.2)	1.3(1.1-1.7)	0.015*	1.2(1.1-1.9)	0.103
Sites					
Bogodogo	128(33.9)	1(ref.)	--		
Bousse	75(32.9)	0.9(0.7-1.3)	0.807	---	---
Kongoussi	140(30)	0.8(0.6-1.1)	0.236	---	---
Houndé	159(34)	1(0.7-1.3)	0.973	---	---
Antibiotics before admission					
No	207(34.3)	1(ref.)	--	---	---
Yes	295(31.5)	0.9(0.7-1.1)	0.245	---	---
Antibiotics during hospitalisation					
No	7(33.3)	1(ref.)	---	---	---
Yes	495(32.6)	1(0.4-2.4)	0.942	---	---
Chronic health condition ²					
No	490(32.7)	1(ref.)	----	1(ref.)	---
Yes	12(29.3)	0.8(0.4-1.7)	0.645	2.3(1.1-5.3)	0.048*
Length of stay					
0- 7 Days	396(32.7)	1(ref.)	0.869	---	---
>7 Days	106(32.2)	1(0.8-1.3)	--	---	---

Variables	Human Rhinovirus-associated infections				
	Prevalence n (%HRV)	Crude OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
Delay of consultation					
0-2 days	254(35.7)	1(ref.)	---	1.7(1.2-2.4)	0.006
3-5 days	190(32.2)	0.8(0.7-1.1)	0.176	1.3(0.9-1.8)	0.236
6 days and more	57(24.1)	0.6(0.4-0.8)	<0.001***	1(ref.)	---
Severe Disease					
No	342(31.2)	1(ref.)	---	---	---
Yes	160(36)	1.2(1-1.5)	0.073	---	---
Severe Outcome³					
No	252(30.5)	1(ref.)	---	---	---
Yes	163(35.8)	1.2(1-1.6)	0.05	---	---
Malnutrition					
No	468(32.6)	1(ref.)	--	--	--
Yes	34(32.7)	1(0.6-1.5)	--	--	--
Viral coinfection					
No	188(39.2)	1(ref.)	---	1(ref.)	---
Yes	314(29.6)	0.6(0.5-0.8)	---	0.12(0.08-0.18)	<0.001***
Bacteria coinfection					
No	73(29.9)	1(ref.)	---	---	---
Yes	429(33.1)	1.1(0.9-1.6)	---	---	---
Virus-Bacteria Coinfection					
No	73(15.4)	1(ref.)	--	1(ref.)	--
Yes	429(40.3)	3.7(2.8-4.9)	<0.001	13.3(8.7-20.2)	<0.001***

Table III: Demographic, clinical factors and other pathogens' coinfections associated with severe outcomes of HRV infection

Variable	Proportion HRV+ with severe outcome n (% HRV)	Crude OR (95% CI)	OR	p-value	Adjusted OR (95% CI)	OR	p-value
Age Group							
0-3 months	21(12.7)	Ref.					
4-11 months	48 (13.4)	1.06 (0.61–1.83)		0.840	1.00 (0.54–1.83)		0.989
1-4 years	86 (12.1)	0.94 (0.57–1.57)		0.819	0.85 (0.48–1.49)		0.563
5-14 years	4 (4.3)	0.31 (0.10–0.93)		0.036	0.38 (0.12–1.21)		0.100
15-49 years	0(0)	1(empty)					
50+ years	3 (5.4)	0.39 (0.11–1.35)		0.138	0.49 (0.12–1.92)		0.304
Area of residence							
Urban		1(ref.)					
rural	67(10.5)	1.1(0.8-1.6)		0.455			

Variable	Proportion HRV+ with severe outcome n (% HRV)	Crude (95% CI)	OR	p-value	Adjusted (95% CI)	OR	p-value
Sex							
male	102(12.3)	0.8(0.5-1.1)		0.118			
female	61(9.7)	1(ref.)					
Season					—		—
Rainy	59 (13.8)	1.43 (1.01–2.01)		0.041			
Dry	103(10.1)	1(ref.)					
Sites							
Bogodogo	27(7.4)	1(ref.)					
Bousse	25(11.3)	1.59 (0.90–2.81)		0.113	1.65 (0.89–3.08)		0.114
Kongoussi	27(6.4)	0.85 (0.49–1.49)		0.579	0.89 (0.49–1.61)		0.697
Houndé	84 (18.7)	2.86 (1.81–4.53)		<0.001 ***	2.72 (1.64–4.54)		<0.001 ***
Delay in Consultation							
Early	92(13.6)	1(ref.)					
Medium	50 (9.1)	0.64 (0.44–0.92)		0.016	0.63 (0.42–0.94)		0.023 *
Late	20 (8.8)	0.61 (0.37–1.02)		0.057	0.73 (0.41–1.28)		0.272
Malnutrition (Yes vs No)	157(11.6)	0.5(0.2-1.2)		0.124			
Antibiotics before admission (Yes vs No)	91(10.3)	0.8(0.6-1.1)		0.159			
Antibiotics during hospitalisation (Yes vs No)	91(11.2)	0.7(0.2-2.6)		0.655			
Chronic health condition (Yes vs No) ²	2(4.9)	0.4(0.1-1.7)		0.207			
ParaInfluenza virus 1 (Positive vs Negative)	3(10.7)	0.9(0.3-3.2)					
ParaInfluenza virus 2 (Positive vs Negative)	2(9.5)						
ParaInfluenza virus 3 (Positive vs Negative)	10(12.8)						
ParaInfluenza virus 4 (Positive vs Negative)	4(7.4)						
Parechovirus (Positive vs Negative)	2(16.6)	1.6(0.3-7.3)					
Influenza A (Positive vs Negative)	2(1.04)	0.07 (0.02–0.29)		<0.001 ***	0.08 (0.02–0.36)		0.001 ***
Influenza B (Positive vs Negative)	3(3.2)						
Influenza C (Positive vs Negative)	1(6.2)						
Human metapneumovirus (Positive vs Negative)	8(9.9)						
Enterovirus (Positive vs Negative)	37 (27.2)	3.53 (2.32–5.38)		<0.001 ***	3.20 (1.96–5.23)		<0.001 ***
Coronavirus OC43 (Positive vs Negative)	6(13.04)						
Coronavirus NL63 (Positive vs Negative)	5(15.6)						
Coronavirus HKU1 (Positive vs Negative)	1(3.1)	0.3(0.03-1.8)		0.175	---		---
Coronavirus 229E	1(9.1)	0.7(0.1-6.2)		0.823	---		---

Variable	Proportion HRV+ with severe outcome n (% HRV)	Crude (95% CI)	OR	p-value	Adjusted (95% CI)	OR	p-value
Bocavirus	15(13.4)	1.2(0.7-2.2)		0.449	---		---
Adenovirus Infection	39 (16.7)	1.95 (1.22–3.12)		0.005	1.95 (1.22–3.12)		0.005 **
<i>Staphylococcus aureus</i>	31(12.1)	1.1(0.7-1.7)		0.619	---		---
<i>Klebsiella pneumoniae</i>	70(13.9)	1.4(1.1-2.1)		0.017*	---		---
<i>Legionella pneumophila</i>	0(0)	---		---	---		---
<i>Legionella longbeach</i>							
<i>Streptococcus pneumoniae</i>	110(13.5)	1.7(1.2-2.4)		0.002* *	---		---
<i>Bordetellasp</i>	0(0)	---		---	---		---
<i>Chlamydia pneumoniae</i>	0(0)	---		---	---		---
<i>Haemophilus influenzae</i>	84 (14.1)	1.62 (1.17–2.24)		0.004* *	1.62 (1.17–2.24)		0.004 **
H. influenzae type b (Hib)	13 (48.1)	7.9 (3.6–17.1)		<0.001 ***	6.2 (2.4–15.8)		<0.001 ***
<i>Moraxella catarrhalis</i>	91(15.5)	2(1.4-2.8)		<0.001 ***	---		---
<i>Mycoplasma pneumoniae</i>	1(5.2)	0.4(0.1-3.3)		0.412	---		---
<i>Pneumocystis jirovecii</i>	7(17.5)	1.7(0.7-3.9)		0.207			
<i>Salmonella spp</i>	0(0)	---		---	---		---
Viral Coinfection (Yes vs No)	108 (10.8)	0.86 (0.61–1.22)		0.405	0.20 (0.12–0.32)		<0.001 ***
Bacterial Coinfection (Yes vs No)	146 (12)	1.7 (1.02–2.9)		0.041*	9.18 (4.92–17.11)		<0.001 ***
Virus and bacteria coinfection	146(14.8)	4.5(2.7-7.6)		<0.001 ***	---		---

1:dry season = November-April; rainy season May-October—2: sickle cell disease, asthma, diabetes, obesity, and other medical chronic conditions —3:severe outcomes included: death, hospitalisation in ICU, *p<0.05 ** p<0.01 ***p<0.001

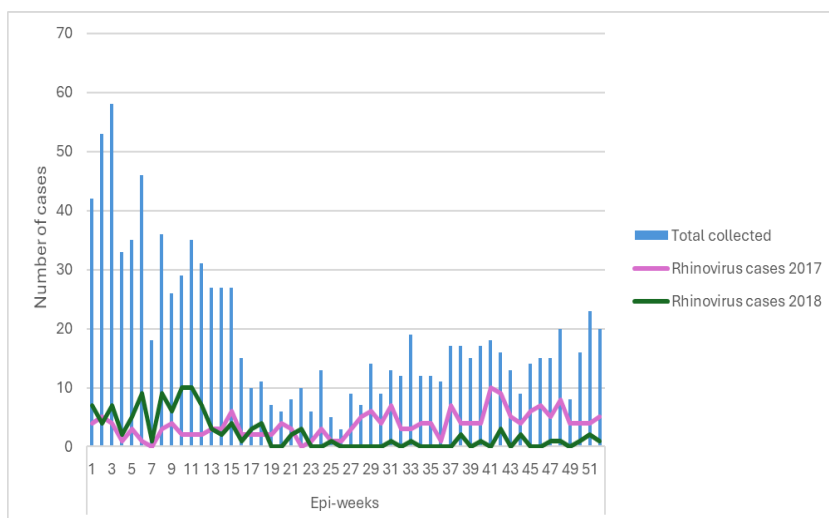


Figure 1: Weekly distribution of Rhinovirus cases in 2017 and 2018

III. Discussion

Our study aimed to shed light on HRV as an essential infectious agent responsible for ARI in hospitalised patients, focusing on its prevalence and the factors associated with infection and severe outcomes in a resource-constrained setting. Using data from routine country-wide surveillance, we were able to demonstrate a high overall prevalence of HRV among SARI cases and to identify the main determinants of both infection and severe outcomes, including death.

HRV has long been considered primarily associated with mild upper respiratory tract infections; however, growing evidence, including our findings, indicates that it is also a significant cause of lower respiratory tract infections and severe ARI requiring hospitalisation (2, 3). In our study population, HRV was detected in 32.6% of hospitalised SARI patients, underlining its etiologic significance, consistent with previous studies in Burkina Faso and the West African region (5, 24). For instance, past work has shown HRV prevalence ranging from 9.6% in hospitalised pediatric patient cohorts in Niger (29) to 33% among outpatient children presenting with influenza-like symptoms in Senegal (20), reflecting variable burden across populations and study designs. This high prevalence of HRV can be explained by the relatively high transmissibility of the virus via two main transmission routes (hands-to-hands and aerosol droplets) (30).

The weekly data also show rhinovirus seasonality over a 2-year period and demonstrate year-round circulation without a pronounced seasonal peak. Our findings align with evidence that rhinovirus exhibits limited seasonality and is detected year-round. Ouédraogo *et al.* reported year-round circulation and a high prevalence of rhinovirus among children with respiratory infections in Burkina Faso (31). Similarly, Kenmoe *et al.* in Cameroon found that rhinovirus was persistently detected with no evident seasonal pattern and multiple peaks across various months (32). This suggests that, unlike influenza, the temporal distribution of rhinovirus in West Africa is influenced by factors beyond climate alone.

Our results highlight that younger children are especially vulnerable: over one-third of those aged less than 5 years were infected with HRV, a finding statistically significant. Children under five represent the group at greatest risk for acute respiratory infections, including HRV, and are more likely than older age groups to experience complications or death (7). Notably, HRV prevalence was not negligible among adults in our study, supporting recent research that HRV can precipitate severe

ARI for adult patients (33, 34). Meta-analytic observations found that HRV is second only to respiratory syncytial virus (RSV) among pediatric causes of bronchiolitis and is also implicated in wheezing and asthma exacerbations in hospitalised patients.

It is also well-known that HRV can be detected in individuals without any symptoms. This asymptomatic HRV carriage is a well-documented phenomenon in the literature, especially when using RT-PCR as a detection tool. It complicates the analysis of causality in acute respiratory infections (ARIs), particularly in the context of infections involving multiple pathogens (35, 36). Our study, conducted in a hospital setting among patients presenting with mild to severe ARI symptoms, allows us to tentatively correlate clinical presentations with HRV biological findings. Analyses based on quantitative polymerase chain reaction (qPCR) could help distinguish between cases of asymptomatic HRV carriage and those of HRV-associated infection by assessing viral load (37, 38).

Urban residence was associated with a higher HRV infection risk in our study. One plausible explanation is the location of our urban surveillance site in a tertiary care hospital (CHU Bogodogo), where patients may have greater exposure to circulating viruses and an increased likelihood of severe presentations, although bias due to case mix must be considered. Additionally, urban settings are usually more crowded and present a more intense transmission of viral infection, such as HRV, especially during seasonal rhinovirus trends (39, 40).

We also observed a significant association between the rainy season and HRV infection in univariate analyses, while the dry season exhibited more cases. Conversely, other studies found an association between the dry season and HRV infection, confirming that HRV, human bocavirus, and human metapneumovirus are "all-year-viruses" that can be detected throughout the seasons, with geographical variations in the trends (40). These findings suggest that although univariate analyses point toward possible seasonality, confounding factors may underlie the observed trend, highlighting the complexity and ongoing controversy regarding climate-related patterns of HRV infection (32, 41, 42).

Coinfections were a major risk factor for severe HRV disease in our cohort. Bacterial coinfections in particular were found in patients with HRV, and the presence of bacterial or mixed viral-bacterial infections markedly increased the risk of severe complications (9-fold risk for severe outcome with bacterial coinfection, supported by prior data on

coinfection consequences in viral ARI (43, 44). *Haemophilus influenzae* and its type b variant were associated with severe HRV outcomes, as were *Streptococcus pneumoniae* and *Moraxella catarrhalis* in our study. Bacterial coinfection in viral ARI is common and has been largely reported in previous studies and reviews. Coinfections were also found to be associated with severe outcomes of ARI. Many studies emphasize the need for robust bacterial surveillance and preventative strategies in respiratory virus management (34, 45, 46).

Enterovirus coinfection with HRV was found to be significantly associated with severe outcomes. These results can be linked to the fact that both enteroviruses and rhinoviruses are members of the *Picornaviridae* family with similar genetic and structural characteristics and trends. Additionally, many RT-PCR tests for respiratory infections target both viruses and report both results as “HRV/ENT” detection, which was not the case in our study, as we used FTD-33® kits. But potential assay limitations should be considered in our results interpretation. The overlapping tropism and seasonality of HRV and Enterovirus, along with the amplified immune response induced by cytokine release, are additional possible explanations reported in previous studies (47, 48).

Overall, 39.3% of HRV-infected patients in our study experienced complicated outcomes, including severe clinical forms, prolonged hospitalisation, or death. Patients with chronic medical conditions such as sickle cell disease, asthma, diabetes, and obesity were disproportionately affected by HRV and more prone to severe outcomes in our series. This supports previous literature indicating that underlying chronic illness increases susceptibility to respiratory complications in HRV. It is now well established that HRV infections are associated with complications of upper and lower respiratory tract infections, including wheezing and asthma (49, 50). Several explanations are highlighted in the literature, including the virus, the host, and the environment. The HRV species A and C are known to cause more severe cases compared to HRV-B(51). Protective effects of viral coinfection alone (compared to bacterial or mixed coinfection) were observed, including influenza A, raising questions about viral-viral interactions in modulating host response that could be an area warranting further studies.

Several limitations of our study should be acknowledged. First, our research relied on routine surveillance data, which can introduce potential selection bias, as patients presenting to the hospital may differ systematically from those with SARI in the community. Second, due to variations in diagnostic capabilities between reference laboratories and over time, misclassification of infection status is possible, especially where HRV detection can be confounded with enteroviruses because of assay limitations. Third, the retrospective nature of surveillance and the variability in clinical and laboratory documentation may have impacted the accuracy and completeness of data on comorbidities, coinfections, and outcomes.

Despite these constraints, our study offers insights into the burden, risk factors, and outcomes of HRV infection in a large, country-wide multisite population, contributing meaningfully to the evolving understanding of this common but often underestimated pathogen in resource-limited settings.

Conclusion

In conclusion, this study highlights the significant role of human rhinovirus (HRV) as a causative agent of severe acute respiratory infections (ARIs) in Burkina Faso. Young children, urban residents, and those with chronic health conditions or coinfections were found to be at higher risk of HRV infection and adverse outcomes. The findings reveal the need for improved surveillance and diagnostic capabilities, particularly in resource-limited settings such as Burkina Faso and many other sub-Saharan African countries, to identify and manage severe cases among vulnerable populations. The association between viral and bacterial coinfections and severe outcomes underscores the importance of integrated infection control measures in hospitals. HRV should benefit from greater recognition by African policymakers as a significant burden on public health systems. Policy efforts should prioritise strengthening laboratory testing, ensuring timely medical consultations, and targeting interventions towards high-risk groups. By understanding demographic and clinical predictors of HRV severity, public health strategies can be better tailored to reduce the overall burden of ARIs in Burkina Faso and other resource-limited settings.

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Conflicts of interest

The authors declare no conflicts of interest.

Contributions of authors

Study conceptualisation: AKI, ZT and SPD; Data collection: AKI, BWOK, AC, HSO; Laboratory analysis: AC, ZT; Data analysis: AKI; First Draft: AKI, AC, MS; manuscript review: ZT, SPD, GT, CS, JBO, NG, BWOK, BT, HSO, MS, AC, EWO; All authors read and approved the final manuscript submitted for publication.

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