



Milano, 25 novembre 2024

CONCORSO PUBBLICO, PER TITOLI ED ESAMI, PER LA COPERTURA DI N. 3 POSTI DI DIRIGENTE BIOLOGO - DISCIPLINA MICROBIOLOGIA E VIROLOGIA - AREA DELLA MEDICINA DIAGNOSTICA E DEI SERVIZI, CON CONTRATTO DI LAVORO A TEMPO PIENO INDETERMINATO.

PROVE D'ESAME E CRITERI DI VALUTAZIONE

PROVA SCRITTA:

Prova scritta n. 1:

Oltre all'identificazione di genere e specie virale, indicare le potenzialità dell'utilizzo del sequenziamento genomico di nuova generazione nell'ambito della caratterizzazione dei patogeni virali; **(PROVA ESTRATTA)**

Prova scritta n. 2:

Indicare l'importanza dell'esecuzione del saggio di sieroneutralizzazione in vitro nella diagnostica delle arbovirosi;

Prova scritta n. 3:

Elencare le principali differenze tecnico/strutturali e standard procedurali dei laboratori con diversi livelli di sicurezza biologica

PARAMETRI DI VALUTAZIONE:

- chiarezza espositiva
- correttezza della risposta
- sufficiente compilazione elaborato

Punteggio: da 0 a 30 punti

PROVA PRATICA: vengono predisposte n. 3 prove contenenti ognuna 3 immagini da commentare, **Allegato 1**
PROVA ESTRATTA n. 3

PARAMETRI DI VALUTAZIONE:

- chiarezza espositiva
- accuratezza della valutazione

Punteggio: da 0 a 30 punti .

PROVA ORALE:

Prova orale n. 1:

Sorveglianza e diagnostica delle infezioni respiratorie virali;

Prova orale n. 2:

Diagnostica delle encefaliti virali da patogeni emergenti endemici nel bacino del mediterraneo;

Prova orale n. 3:

Tipizzazione dei virus influenzali **(PROVA ESTRATTA)**

Per la prova di inglese è stato dato da leggere e tradurre un paragrafo di un articolo scientifico - **Allegato 2** - e per la prova d'informatica e' stato posto il quesito uguale per tutti: **"Che cos'è il LIS?"**.

PARAMETRI DI VALUTAZIONE:

- conoscenza del quesito posto;
- chiarezza espositiva.

Punteggio: da 0 a 20 punti .

IL SEGRETARIO
DELLA COMMISSIONE ESAMINATRICE
Daniela Barbara Carnassale

1

Immagine1_1prova

Codice quesito: D00001

1



Descrivere l'immagine.

2

Immagine2_1prova

Codice quesito: D00002

2



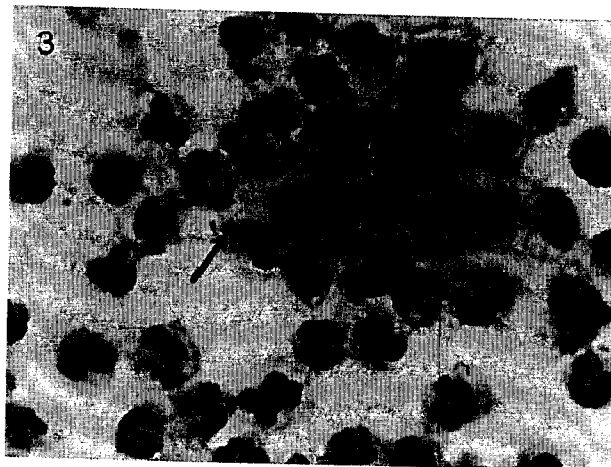
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3

Immagine3_1prova

Codice quesito: D00003

3



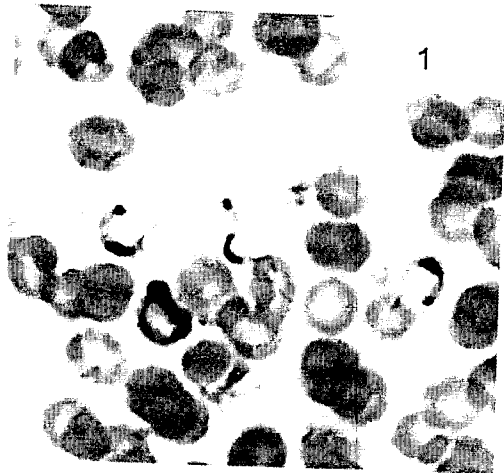
Descrivere l'immagine.

PRATICA_PROVA 02

1

Immagine1_2prova

Codice quesito: **E00001**



Descrivere l'immagine.

2

Immagine2_2prova

Codice quesito: **E00002**

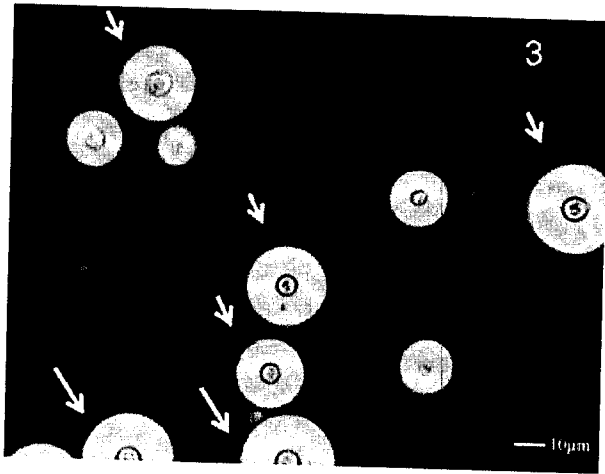


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3

Immagine3_2prova

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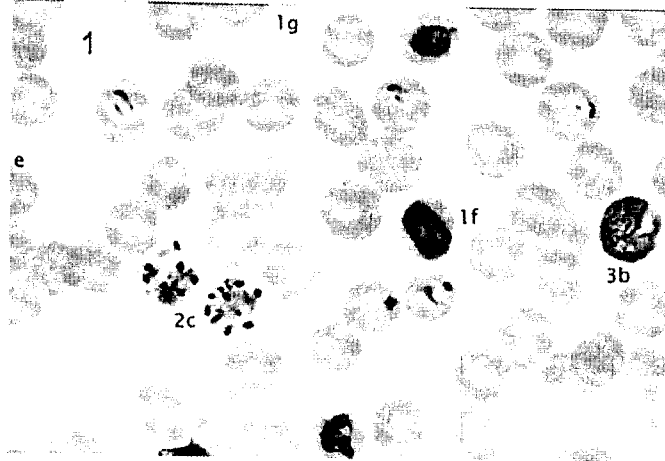


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1

Immagine1_3prova

Codice quesito: F00001

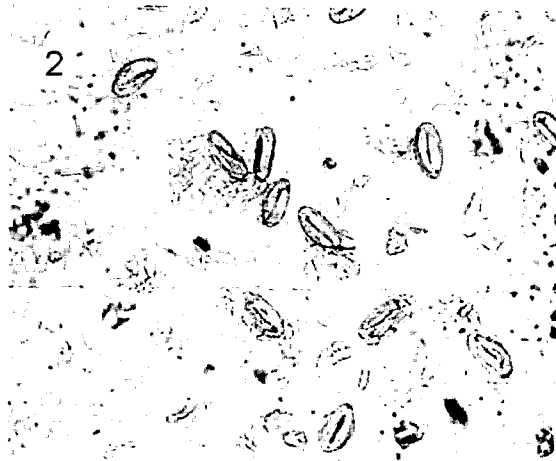


Descrivere l'immagine.

2

Immagine2_3prova

Codice quesito: F00002



Descrivere l'immagine.

3

Immagine3_3prova

Codice quesito: F00003



Descrivere l'immagine.

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Enterovirus D68 and acute flaccid myelitis—evaluating the evidence for causality

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Abstract

Increased circulation of enterovirus D68 in 2014 and 2016 temporally and geographically coincided with increases in cases of acute flaccid myelitis, an uncommon condition of paralysis due to lesions in the anterior horn of the spinal cord. The identification of enterovirus D68 in respiratory specimens from cases of acute flaccid myelitis worldwide further supports an association, yet the absence of direct virus isolation from affected tissues, infrequent detection in cerebrospinal fluid, and the absence, until recently, of an animal model has left the causal nature of the relationship unproven. In this Personal View we evaluate epidemiological and biological evidence linking enterovirus D68 and acute flaccid myelitis. We applied the Bradford Hill criteria to investigate the evidence for a causal relationship and highlight the importance of comprehensive surveillance and research to further characterise the role of enterovirus D68 in acute flaccid myelitis and pursue effective therapies and prevention strategies.

Introduction

In 2012, cases of a polio-like neurological disease, now designated acute flaccid myelitis (AFM), occurred in California, with enterovirus D68 identified in respiratory tract specimens.¹ Subsequently, temporal and geographic correlations of increased enterovirus D68 circulation with clusters of AFM cases worldwide have further suggested a possible

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Contributors
KM wrote the first draft of the manuscript. KM and SRD did the literature review. AMH created figures for the manuscript. KM, EJA, AMH, CVL-B, HGMN, KLT, MJA, and SRD critically reviewed the manuscript and contributed to the content. All authors approved the submitted version of the manuscript.

Declaration of interests
We declare no competing interests.

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causal relationship.² However, inability to identify enterovirus D68 in the CNS in most cases of AFM leaves the association without direct proof of causality. Thus, the relationship between the emergence of enterovirus D68 and the rise in AFM cases is controversial.

Enterovirus D68 and acute flaccid myelitis

Enterovirus D68 was discovered in 1962 after being isolated from respiratory specimens of children with pneumonia in California.³ Enterovirus D68 is a non-polio enterovirus with biological and clinical properties so similar to those of human rhinoviruses that it was initially classified as rhinovirus 87.^{4,5} Enterovirus D68 grows optimally at 33°C and primarily binds sialic acid receptors in the upper and, less commonly, lower respiratory tract.^{4,6,7} Enterovirus D68 is transmitted mainly via the respiratory route and detected in respiratory specimens early in the course of disease. Unlike acid-stable and heat-stable enteroviruses, enterovirus D68 is uncommonly detectable in stool.⁴

Only 26 cases of enterovirus D68 infection were reported through the passive US National Enterovirus Surveillance System from 1970 to 2005.⁸ Numbers of small clusters of enterovirus D68 respiratory illness increased in Europe, Asia, and the USA from 2008 to 2010.⁹ In 2014, the USA experienced a large enterovirus D68 outbreak, with 1153 confirmed infections coinciding with a surge in respiratory illnesses detected by syndromic surveillance that suggested millions of cases.¹⁰ Retrospective analysis showed enterovirus D68 circulation in Europe during the same period.¹¹ In 2015, no enterovirus D68 isolates were reported in the USA, suggesting little to no circulation¹²⁻¹⁴ but in the late summer to autumn of 2016, enterovirus D68 was again detected at sites doing active surveillance in the USA and Europe.¹³⁻¹⁷

AFM has recently been described as acute onset of flaccid limb weakness, with imaging showing spinal cord grey matter lesions suggestive of anterior myelitis.¹⁸ The inclusion of imaging criteria in the case definition for AFM was intended to provide more specificity to the broader epidemiological case definition of acute flaccid paralysis (AFP), which is primarily used for global poliovirus surveillance. Although use of the term AFM is new, the clinical condition it describes is not, encompassing cases previously described as poliomyelitis, polio-like illness, and acute flaccid paralysis with anterior myelitis.¹⁹ Throughout this paper, AFP refers to cases of acute flaccid limb weakness without further characterisation, whereas AFM is used to refer to the subset of cases of AFP with additional imaging findings suggestive of myelitis. The asymmetric lower motor-neuron-specific deficits and characteristic longitudinal anterior horn predominant spinal cord grey matter lesions can help distinguish AFM from other causes of AFP, including Guillain-Barré syndrome, acute disseminated encephalomyelitis, and transverse myelitis, although some overlap in epidemiological case definitions and clinical findings exists.² Most commonly associated with poliovirus and other non-polio enteroviruses, AFM has also been described in association with endemic and epidemic neurotropic flaviviruses, most notably West Nile virus and Japanese encephalitis virus.²⁰⁻²² Although AFM is now rare in countries with adequate poliovirus vaccination, several AFM clusters occurred in the USA, Canada, and Europe in 2014 and 2016,²³⁻³⁰ coincident with emergence of enterovirus D68.

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