Journal of Invertebrate Pathology 114 (2013) 186–195

Contents lists available at ScienceDirect

Journal of Invertebrate Pathology

journal homepage: www.elsevier.com/locate/jip

Minireview

Arthropod viruses and small RNAs

Diveena Vijayendran, Paul M. Airs, Kelly Dolezal, Bryony C. Bonning *

Department of Entomology, Iowa State University, Ames, IA 50011, USA

ARTICLE INFO

Article history:
Received 1 July 2013
Accepted 28 July 2013
Available online 8 August 2013

Keywords:
Small interfering RNA
MicroRNA
PIWI-associated RNA
Antiviral immunity

ABSTRACT

The recently characterized small RNAs provide a new paradigm for physiological studies. These molecules have been shown to be integral players in processes as diverse as development and innate immunity against bacteria and viruses in eukaryotes. Several of the well-characterized small RNAs including small interfering RNAs, microRNAs and PIWI-interacting RNAs are emerging as important players in mediating arthropod host–virus interactions. Understanding the role of small RNAs in arthropod host–virus molecular interactions will facilitate manipulation of these pathways for both management of arthropod pests of agricultural and medical importance, and for protection of beneficial arthropods such as honey bees and shrimp. This review highlights recent research on the role of small RNAs in arthropod host–virus interactions with reference to other host-pathogen systems.

© 2013 Elsevier Inc. All rights reserved.

Contents

1. Introduction . . . 187
   1.1. Small RNA biogenesis and function . . . 187
      1.1.1. miRNA . . . 187
      1.1.2. siRNA . . . 187
      1.1.3. piRNA . . . 187
   1.2. Arthropod immunity against viruses . . . 187
2. MicroRNA in arthropod–virus interaction . . . 189
   2.1. Deregulation of host miRNA on pathogen infection . . . 189
   2.2. Host miRNA function in antiviral defense . . . 190
   2.3. Virus encoded miRNAs facilitate virus infection . . . 190
   2.4. Viral proteins interact with host miRNA . . . 190
3. Short-interfering RNA in arthropod–virus interactions . . . 191
   3.1. Profiles of RNA virus vsRNAs . . . 191
   3.2. vsRNAs are used by viruses to distract the host antiviral RNAi response . . . 191
   3.3. vsRNAs drive virus evolution . . . 191
   3.4. vsRNAs from DNA viruses . . . 191
   3.5. Endogenous siRNAs in antiviral immunity . . . 192
4. PIWI-interacting RNA in arthropod–virus interactions . . . 192
   4.1. Virus-derived piRNAs . . . 192
   4.2. Role of piRNAs in the absence of a functional siRNA pathway . . . 192
5. Summary . . . 193
   Acknowledgments . . . 194
   References . . . 194

Abbreviations: usRNA, unusually small RNA (13–19 nt); vsRNA, virus-derived small interfering RNA; VSR, viral suppressor of RNAi.

* Corresponding author. Fax: +1 515 294 2995.
E-mail address: bbonning@iastate.edu (B.C. Bonning).

0022-2011/$ - see front matter © 2013 Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.jip.2013.07.006

1. Introduction

1.1. Small RNA biogenesis and function

Small RNAs have emerged as key regulators of important biological processes in eukaryotes. The three major types of small RNAs identified in eukaryotes are microRNA (miRNA), small-interfering RNA (siRNA) and PIWI-interacting RNA (piRNA). The biogenesis of eukaryotic small RNAs shares a dependence on the Argonaute (Ago) protein family (Hock and Meister, 2008; Nayak et al., 2010). Each small RNA type has distinct functions and characteristics including their (1) biogenesis, (2) length and modifications, and (3) targets (Farazi et al., 2008). All three major classes of small RNA function in host-virus interactions in arthropods.

1.1.1. miRNA

MicroRNAs (miRNA) are a group of small RNA of 18–25 nt found in most eukaryotic cells. This class of small RNA regulates gene expression by modulating the availability of messenger RNA (mRNA) for translation into protein. A primary miRNA is transcribed in the nucleus by cellular RNA polymerase II. The primary miRNA is further processed into a precursor miRNA by the proteins Drosha and Pasha. The precursor miRNA is transported into the cytoplasm to be cleaved into double-stranded RNA (dsRNA) duplexes of 18–25 nt by the Dicer protein (Macrae et al., 2006). One strand of the duplex (guide strand) is loaded onto the Argonaute-protein complex (RNA-induced silencing complex, RISC) and targets mRNA in the cell. Near perfect complementarity of the miRNA to the target mRNA results in the cleavage and degradation of the target transcript, a process most commonly observed in plants (Brodersen et al., 2008). In animals, the miRNA seed region of 6–8 nt binds the target mRNA and prevents translation (Lim et al., 2005). Functional studies of miRNA in arthropods have shown regulation of important biological processes such as wing development, tissue differentiation and cell proliferation (Jones and Newbury, 2010; Schnall-Levin et al., 2010).

1.1.2. siRNA

The RNA interference (RNAi) pathway has been established as a major antiviral defense pathway in arthropods. Long exogenous dsRNAs are cleaved by Dicer into siRNA duplexes of 21 to 22 nt (Bernstein et al., 2001). One strand of the duplex is loaded onto the RISC which contains the Argonaute protein (Martinez et al., 2002). The siRNA guides the complex to bind and cleave complementary viral RNA. Several recent review papers describe the major role of RNAi in the antiviral immune response of arthropods (Blair, 2011; van Mierlo et al., 2011; Vodovar and Saleh, 2012).

Small-interfering RNAs (siRNAs) can be divided into two categories, endogenous and exogenous siRNAs. Endogenous siRNAs (endo-siRNA) are primarily derived from retrotransposons and genomic RNA that can form double-stranded RNA by overlapping transcripts or local secondary structures (Ghildiyal et al., 2008; Okamura et al., 2008). The production of endogenous siRNA is dependent on the host RNAi pathway (Kawamura et al., 2008). Endogenous siRNAs function to silence mobile genetic elements and mRNA transcripts in the host (Kritikou, 2008). Exogenous siRNAs (exo-siRNA) on the other hand are primarily derived from invading nucleic acids such as those of viruses or experimentally introduced dsRNA. Exogenous siRNAs derived from replicating viruses are termed virus-derived small interfering RNAs (vsRNAs). Many parts of the virus genome can serve as a trigger for the host RNAi-based antiviral response. The triggers can include the complementary double-stranded RNA intermediate during replication, local secondary structures in the virus genome, virus encoded siRNA and overlapping virus transcripts.

1.1.3. piRNA

PIWI-associated RNAs (piRNA) are 25–30 nt small RNAs produced in a Dicer-independent mechanism (Houwing et al., 2007; Vagin et al., 2006). Argonaute-3 (Ago3) cleaves piRNA from a precursor sequence, after which the piRNA is incorporated into a RISC, along with PIWI and Aubergine (Aub), to guide degradation of complementary RNA sequences. A "ping–pong" mechanism for amplification of piRNAs is now also supported, in which a sense, primary piRNA as part of a RISC, binds to a complementary, antisense RNA sequence. The target sequence is cleaved with the help of Aub and PIWI such that the sense, primary piRNA and the cleaved, antisense target contain a 10 nt overlap. A conserved feature of this overlap is an A at the first 5' antisense position and a complementary U at the 10th 5' sense position. That antisense target is then further cleaved to become a 25–30 nt secondary piRNA. This secondary piRNA can then bind to the 10 overlapping nt in other sense sequences, from which the original piRNA was derived, and with the help of Ago3, cleaves another primary piRNA. The production of primary piRNAs thus drives the production of secondary piRNAs, and vice versa (Siomi et al., 2011). The presence and role of piRNAs was initially thought to be solely transposon repression in the germline (Juliano et al., 2011). However, recent studies have now shown piRNA expression in somatic tissues. First, the somatic tissue surrounding the *Drosophila* ovary was found to express PIWI proteins and piRNAs, albeit via an Aub- and Ago-3-independent version of the piRNA biogenesis, called the primary pathway (Malone et al., 2009). While it is still unclear if piRNAs are expressed more broadly in fly tissue, the whole repertoire of PIWI-family proteins and piRNAs are now known to be expressed in the somatic head and thorax tissue of *Aedes aegypti* and *Aedes albopictus* (Morazzani et al., 2012). These data set the stage to explore piRNA roles beyond transposon regulation, including antiviral activity.

1.2. Arthropod immunity against viruses

A robust immune response against viruses in arthropods includes JAK/STAT, Imd and Toll signaling pathways in addition to the antiviral RNAi pathway (Tsakas and Marmaras, 2010). This review will focus on antiviral responses involving the three major classes of small RNA in arthropods. For a comprehensive review of the antiviral signaling pathways and other inducible antiviral proteins, see Fragkoudis et al. (2009), Kingsolver and Hardy (2012), Merkling and van Rij (2013) (see Table 1).

The majority of RNAi based antiviral studies of arthropods have been carried out in the model organism *Drosophila melanogaster* and various mosquito species (Aliyari et al., 2008; Flynt et al., 2009; Sabin et al., 2013; Siu et al., 2011; Wu et al., 2010). The RNAi response produces vsRNAs to reduce the virus load of replicating viruses. The production of vsRNAs is an important part of the innate immune response against viruses in arthropods. The RNAi based-immunity pathway in arthropods is highly developed and recognition of non-native RNAs triggers the defense response (Keene et al., 2004).

The dynamics of RNA interference in establishment of persistent virus infection in insects were recently elucidated. An emerging concept was experimentally validated using Flock house virus (FHV: (+) ssRNA, Nodaviridae) *in vitro* in *Drosophila* S2 cells and *in vivo* in adult *Drosophila melonagaster*. Genome segments of FHV were reverse-transcribed to cDNA as early as 12 h after infection by host reverse transcriptases (originating from retrotransposons or endogenous retroviruses). Surprisingly, the cDNA form of FHV was a reorganized, recombinant form of the 2 RNA segments, RNA1 and RNA 2. The FHV cDNA was then transcribed and processed into vsRNAs by the host RNAi machinery. These vsRNAs are loaded onto the RISC and mediate RNAi of virus RNA. The cDNA

Download English Version:

https://daneshyari.com/en/article/4557778

Download Persian Version:

https://daneshyari.com/article/4557778

Daneshyari.com