Antiviral Research 123 (2015) 172–187

Contents lists available at ScienceDirect

Antiviral Research

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

Meeting Report

Meeting report: 28th International Conference on Antiviral Research in Rome, Italy

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ARTICLE INFO

Article history:
Received 26 September 2015
Accepted 28 September 2015
Available online 24 October 2015

Keywords:
Sofosbuvir
Hepatitis C virus
Ebola virus
RNA viruses
Emerging viruses
Antiviral chemistry

ABSTRACT

The 28th International Conference on Antiviral Research (ICAR) was held in Rome, Italy from May 11 to 15, 2015. This article summarizes the principal invited lectures. Phillip Furman, the Elion award recipient, described the research leading to sofosbuvir. Dennis Liotta, who received the Holý award, described how an investigation into HIV entry inhibitors led to a new therapy for cancer patients. Erica Ollmann Saphire, winner of the Prusoff Young Investigator award, explored the world of viral proteins and how they remodel to perform different essential roles in viral replication. The keynote addresses, by Raffaele De Francesco and Michael Manns, reported on the remarkable progress made in the therapy of chronic HCV infections. A third keynote address, by Armand Sprecher, related the difficulties and successes of Médicins Sans Frontières in West Africa ravaged by the Ebola outbreak. There were three mini-symposia on *RNA Viruses*, *Antiviral Chemistry* and *Emerging Viruses*. There was a good collection of talks on RNA viruses (norovirus, rabies, dengue, HEV, HCV, and RSV). A highlight of the chemistry was the preparation of prodrugs for nucleotide triphosphates as this opens a door to new options. The third mini-symposium emphasized how research work in the antiviral area is continuing to expand and needs to do so with a sense of urgency. Although this meeting report covers only a few of the presentations, it aims to illustrate the great diversity of topics discussed at ICAR, bringing together knowledge and expertise from the whole spectrum of antiviral research.

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http://dx.doi.org/10.1016/j.antiviral.2015.09.015
0166-3542/

1. Introduction

This article provides an overview of the invited lectures at the 28th International Conference on Antiviral Research (ICAR), sponsored by the International Society for Antiviral Research (ISAR), which was held in Rome, Italy, from May 11–15, 2015. It begins with reports of lectures by the recipients of ISAR's three major awards, held in memory of Gertrude (Trudy) Elion, Antonín (Tony) Holý and William (Bill) Prusoff. These are followed by summaries of the three keynote addresses and the main presentations within the three mini-symposia on "*RNA Viruses*", "*Antiviral Chemistry*" and "*Emerging Viruses*". Because this review article simply provides short accounts of oral presentations, it is not generally accompanied by references to the scientific literature. Any descriptions of favorable treatment outcomes should not be taken as a recommendation for clinical use. Generally, I have added my personal comments on the meeting within the conclusion. In a few instances, I have added my own comment within the main text, indicated either by the wording or by the use of square brackets. One of my aims has been to illustrate how the great diversity of topics can stimulate thinking in other areas of antiviral research, one of the strengths of ICAR.

2. Gertrude Elion Memorial award lecture: sofosbuvir: a search for a cure. Phillip (Phil) Furman., Furman Biotech Consulting, St Augustine, FL, USA.

Having joined Burroughs Wellcome in 1975, Phil (Fig. 1) worked with Trudy Elion for ten years. During this time, he was involved in the development of acyclovir (Zovirax®) and its prodrug, valacyclovir (Valtrex®). In 2004, Phil joined Pharmasset. The focus of this presentation was his research at Pharmasset, leading to the identification of the activity of sofosbuvir and to an understanding of its mechanism of action against hepatitis C virus (HCV).

Phil's account started with the cytidine analog, PSI-6130 (Fig. 2). This was one of the more active compounds in development at that time, better than the Roche and Idenix nucleosides but possibly a little less active than the Merck compound. An additional important

Fig. 1. Bob Buckheit congratulates Phil Furman on receiving the Elion award.

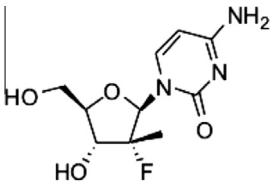

Fig. 2. Structure of PSI-6130, 2′-α-F, 2′-*C*-methylcytidine.

factor, PSI-6130 lacked detectable cytotoxicity (CC_{50} > 100 μM) in a panel of 5 cell lines (Clone A, Huh7, HepG2, CEM and PBM). Incidentally, the Merck compound showed no cytotoxicity in 3 cell lines but had a CC_{50} of 5 μM in CEM cells and therefore was rejected. In contrast to many reported cytotoxicity values, these are derived