Originality: and cc superfamilies’ and due to

Originality: This was a follow up study which was performed based on theknown relevance of leukocyte migration in Varicella zoster virus (VZV) spreadand subsequent pathogenesis, thus possible modulation of leukocyte migration byVZV was investigated.

The study found that cell-free VZV does indeed enhancechemokine activity, which increases pathogenesis as expected. This however is partially dependant on glycoprotein C(gC), other factors are currently unknown. This report also contains the firstdescription of vCKBP in VZV. VZV rSgC can be considered novel due to it bindinga large number of chemokines specifically from the CXC and CC superfamilies’and due to it enhancing their activity, phenomenon previously only observed nHSV glycoprotein C.

This report though not making a huge difference to theknowledge state of this field, it opens up questions and hopefully willencourage more research into the mechanisms of dissemination used by VZV. Reasons forUndertaking Study: The study focused on Varicella zoster virus (VZV), a virus whichestablishes latency in the neurons of the peripheral nervous system. Uponinfection VZV modifies the activity of important immune components such as Tcells and leukocytes, but despite its importance in pathogenies relativelylittle study has been done to investigate the mechanism of VZV disseminationthroughout the body.

The article aims to address the influence of VZV onleukocyte migration in particular due to glycoprotein C. Main Conclusions of Study: The report has found that cell free VZV does enhance chemokine activity andthis is partially dependent on gC. Glycoprotein C was identified as being involvedin the

recruitment of leukocytes by VZV. Data shows that VZV gC does enhanceleukocyte migration through the modulation of chemokine activity, which couldfacilitate VZV infection of host leukocytes and dissemination of the virus. Inaddition as chemokines are known to induce pain, enhancement of their activitymay play a role in VZV associated pathology. Results also show that VZV gC is a vCKBP that interacts with highaffinity. VZV rSgC binds to a broadrange of CXC and CC chemokines with nanomolar affinity and therefore acts as anovel vCKBP.

Flaws of Study: Though notmany flaws come to mind some minor issues were had with the layout of figuresand their clarity. E. g. Figure 1, graph C.

Also whilean interesting study it seems to only scrape the surface of this area ofresearch, the report itself admitted little is known about the mechanisms ofVZV dissemination. Future Directions of Study: What is the role of gC-protein mediated chemokine enhancement in vivio? What other factors play a role in VZV mediated enhancement of leukocytes and towhat extent? Does VZV gC enhance leukocyte migration to the initial site of infection? Overall Opinion of Paper: The articlewas an interesting look into what seem to be a neglected area of research. Thoughit may not have a large impact I believe it does represent a positivecontribution to the body of knowledge dealing with understanding the mechanismof VZV dissemination. Level of difficulty: 6