

IN THE COURT OF APPEALS OF THE STATE OF WASHINGTON

2013 JUN -6 PM 1:17
STATE OF WASHINGTON
BY DEPUT

FILED
COURT OF APPEALS
DIVISION II

State V. Washington

DIVISION II

NO. 44984-6

PERSONAL RESTRAINT PETITION

Christopher Lee Olsen
Petitioner's Full Name

If there is not enough room on this form, use the back of these pages or use other paper. Fill out all of the form and other papers you are attaching before you sign this form in front of a notary.

A. STATUS OF PETITIONER

I, Christopher Lee Olsen #831898, Washington State Penitentiary
(Full name and current address)
1313 North 13th Ave Walla Walla, Wa 99362-8817

Apply for relief from confinement. I am am not now in custody serving a sentence upon conviction of a crime. (If not serving a sentence upon conviction of a crime) I am now in custody because of the following type of court order:
(Identify type of court order)

- The court in which I was sentenced is: Thurston County Superior Court
- I was convicted of the crime of: Felony Murder 1
- I was sentenced after (check one) Trial Plea of Guilty on 07-23-08
Date of Sentence
- The Judge who imposed sentence was The honorable Christina Pomeroy
- My lawyer at trial court was Richard Allen Woodrow 3732 Pacific Ave S.E.
Name and Address if known
Olympia, Wa 98501

6. I did did not appeal from the decision of the trial court. (If the answer is that I did), I appealed to: C.O.A. Division II
Name of court or courts to which appeal took place

7. My lawyer for my appeal was: Jodi Backlund & Manek Mistry P.O. Box 6490 Olympia, Wa 98507
Name and address if known or write "none"

The decision of the appellate court was was not published. (If the answer is that it was published, and I have this information) the decision is published in State v. Sublett

156 Wn. App. 160, P.3d 23 (2010)

8. Since my conviction I have have not asked a court for some relief from my sentence other than I have already written above. (If the answer is that I have asked, the court I asked was N/A. Relief was denied on
Name of court

Date of Decision or, if more than one, all dates)

(If you have answered in question 7 that you did ask for relief), the name of your lawyer in the proceedings mentioned in my answer was N/A
Name and address if known

9. If the answers to the above questions do not really tell about the proceedings and the courts, judges and attorneys involved in your case, tell about it here: Please See My Memorandum

in Support of this PRP (Attachment #1)

(It should be noted the Prosecuting attorney David Brunson has committed this misconduct before and most recently in the case of State v. Herbin (Deshone) May 2013 CoA Div II decision)

B. GROUNDS FOR RELIEF:

(If I claim more than one reason for relief from confinement, I will attach sheets for each reason separately, in the same way as the first one. The attached sheets should be numbered "First Ground", "Second Ground", "Third Ground", etc). I claim that I have 6 reason(s) for this court to grant me relief from the conviction and sentence described in Part A.

First Ground
(First, Second, etc)

1. I should be given a new trial or released from confinement because (State legal reasons why you think there was some error made in your case which gives you the right to a new trial or release from confinement): The prosecutor in my case (David Harold Bruneau

WSBA# 6830) Committed misconduct that deprived me of the
right to a fair trial. (It should be noted Mr. Bruneau has done this in other
cases as well see State v. Deshone Herbin 2011)

2. The following facts are important when considering my case. (After each fact statement put the name of the person or persona who know the fact and will support your statement of the fact. If the fact is already in the record of your case, indicate that also) The prosecutor in my

case did in fact use an unadmitted booking photo of me altered to

Show the word "guilty" across my face in front of the jury during his closing argument
See Attachments # (3,4,5)

3. The following reported court decisions (indicate citations if possible) in cases similar to mine show the error I believed happened in my case. (If none are known, state "None Known". _____)

In Re Glasmann, 175 Wn.2d 696 (2012) see attachment # (2)
STATE V. Herbin (Deshone) Court of appeals ^{DIV. II} May 2013.

4. The following statutes and constitutional provisions should be considered by the court. (If none are now, state, "None Known") The 6th and 14th amendments to the U.S.

Constitution and article I, section 22 of the Washington State Constitution

5. This petition is the best way I know to get the relief I want, and not other way will work as well because: My direct appeals are exhausted and this is new case law not available to me when I filed my direct appeal.

C. STATEMENT OF FINANCES:

If you cannot afford to pay the \$250 filing fee or cannot afford to pay an attorney to help you, fill out this form. If you have enough money for these, do not fill this part of the form. If currently in confinement you will need to attach a copy of your prison finance statement.

1. I do do not _____ ask the court to file this without making me pay the \$250 filing fee because I am so poor and cannot pay the fee.

2. I have \$ 0⁰⁰ in my prison or institution account.

3. I do do not ask the court to appoint a lawyer for me because I am so poor and cannot afford to pay a lawyer.

4. I am am not employed. My salary or wages amount to \$ _____ a month. My employer is _____
Name and address of employer

5. During the past 12 months I did did not get any money from a business, profession or other form of self-employment. (If I did, it was _____
Type of self-employment

And the total income I received was \$ 0.

6. During the past 12 months I:

Did Did Not Receive any rent payments. If so, the total I received was \$ _____

Did Did Not Receive any interest. If so, the total I received was \$ _____

Did Did Not Receive any dividends. If so, the total I received was \$ _____

Did Did Not Receive any other money. If so the total I received was \$ _____

Do Do Not Have any cash except as said in question 2 of Statement of Finances. If so the total amount of cash I have is \$ _____

Do Do Not Have any savings or checking accounts. If so, the total amount in all accounts is \$ _____

Do Do Not Own stocks, bonds or notes. If so, their total value is: \$ _____

7. List all real estate and other property or things of value which belong to you or in which you have an interest. Tell what item or property is worth and how much you owe on it. Do not list household furniture and furnishings and clothing which you or your family need.

Items	Value
<u>0</u>	

8. I am am not married. If I am married, my wife or husband's name and address is:

9. All of the persons who need me to support them are listed below:

Name & Address	Relationship	Age
<u>N/A</u>		

10. All the bills I owe are listed here:

Name & Address of Creditor	Amount
<u>Thurston County 2000 Lake ridge Dr SW Olympia wa 98502</u>	<u>\$13,000⁰⁰</u>

D. REQUEST FOR RELIEF:

I want this court to:

Vacate my conviction and give me a new trial

^{or} Vacate my conviction and dismiss the criminal charges against me without a new trial

Other: _____
(Please Specify)

E. OATH OF PETITIONER

STATE OF WASHINGTON

COUNTY OF Walla Walla

After being first duly sworn, on oath, I depose and say: That I am the petitioner, that I have read the petition, know its contents, and I believe the petition is true.

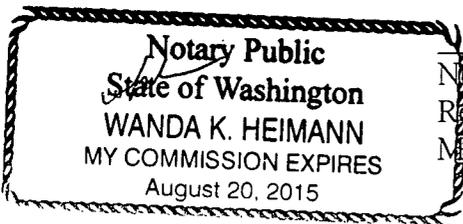
Christopher Lee Olsen

Signature

Christopher Lee Olsen #831898

Print Name & Number

SUBSCRIBED AND SWORN to before me this 11th day of April
2013



Wanda K. Heimann

Notary Public in and for the State of Washington

Residing at Walla Walla, WA

My commission expires 8/20/15

If a notary is not available, explain why none is available and indicate who can be contacted to help you find a Notary: _____

I declare that I have examined this petition and to the best of my knowledge and belief it is true and correct.

DATED This _____ day of _____, 2_____.

Print Name & Number

02/13/2013

Department of Corrections

PAGE : 01 OF 01

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WASHINGTON STATE PENITENTIARY

OIRPLRAR

10.2.1.18

PLRA IN FORMA PAUPERIS STATUS REPORT
FOR DEFINED PERIOD : 07/31/2012 TO 01/31/2013

DOC# :	0000831898	NAME :	OLSEN CHRISTOPHER	ADMIT DATE :	07/23/2008
DOB :	12/25/1980			ADMIT TIME :	14:00
	AVERAGE		20% OF		AVERAGE
	MONTHLY RECEIPTS		RECEIPTS		SPENDABLE BALANCE
	1.83		0.37		0.35
					20% OF
					SPENDABLE
					0.07



MLP
2-13-13

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WASHINGTON STATE PENITENTIARY

OTRATA

TRUST ACCOUNT STATEMENT

10.2.1.3

DOC#: 0000831898
LOCATION: E01-202-RB2121

Name: OLSEN, CHRISTOPHER L

DOB: 12/25/1980

ACCOUNT BALANCES Total: 13.73 CURRENT: 13.73 HOLD: 0.00
07/31/2012 02/13/2013

SUB ACCOUNT	START BALANCE	END BALANCE
SPENDABLE BAL	0.07	0.00
SAVINGS BALANCE	13.23	13.23
WORK RELEASE SAVINGS	0.00	0.00
EDUCATION ACCOUNT	0.00	0.00
MEDICAL ACCOUNT	0.00	0.00
POSTAGE ACCOUNT	2.60	0.50
COMM SERV REV FUND ACCOUNT	0.00	0.00



*MD
24313*

DEBTS AND OBLIGATIONS

TYPE	PAYABLE	INFO NUMBER	AMOUNT OWING	AMOUNT PAID	WRITE OFF	AMT.
HYGA	INMATE STORE DEBT	06032004	214.02	84.55		0.00
MEDD	MEDICAL COPAY DEBT	07302009	13.00	0.00		0.00
COSXD	COST OF SUPERVISION DEBT	10012011	590.47	0.00		0.00
TVD	TV CABLE FEE DEBT	06102006	2.50	0.00		0.00
HYGA	INMATE STORE DEBT	03212006	2.23	0.00		0.00
COI	COST OF INCARCERATION	05212004	UNLIMITED	0.68		0.00
CVC	CRIME VICTIM COMPENSATION	05212004	UNLIMITED	25.93		0.00
HYGA	INMATE STORE DEBT	04172006	84.86	18.49		0.00
TVD	TV CABLE FEE DEBT	08142004	0.00	1.48		0.00
MISCD	MISCELLANEOUS DEBT	09142004	26.82	0.00		0.00
LFO	LEGAL FINANCIAL OBLIGATIONS	20040728	UNLIMITED	133.97		0.00
WRBD	WR ROOM AND BOARD DEBT	08232006	93.10	321.11		0.00
644D	CSRF LOAN DEBT	HQ CK#2671	0.00	200.00		0.00
SPHD	STORES PERSONAL HYGIENE DEBT	10012008	72.44	0.00		0.00
COPD	COPY COSTS DEBT	01172013	11.00	0.00		0.00
MEDD	MEDICAL COPAY DEBT	07052006	2.90	0.00		0.00
COSFD	COS - FELONY DEBT (206)	06262010	0.00	0.00		0.00
HYGA	INMATE STORE DEBT	09292008	1197.15	18.56		0.00
POSD	POSTAGE DEBT	03012006	9.60	6.00		0.00
DCS	CHILD SUPPORT PAYMENTS	004742033	UNLIMITED	47.70		0.00
TVD	TV CABLE FEE DEBT	10112008	20.36	0.00		0.00
TVD	TV CABLE FEE DEBT	03112006	0.50	0.00		0.00
POSD	POSTAGE DEBT	10012008	203.22	0.00		0.00
EL	ESCORTED LEAVE	09-2011	UNLIMITED	0.00		0.00
MEDD	MEDICAL COPAY DEBT	07082004	3.00	9.00		0.00
COSFD	COS - FELONY DEBT (206)	05212004	0.00	1800.20		0.00
LMD	LEGAL MAIL DEBT	03022006	1.26	0.00		0.00
LMD	LEGAL MAIL DEBT	01302013	1.32	0.00		0.00
DEND	DENTAL COPAY DEBT	12222008	12.78	0.00		0.00

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OTRTASTA

T R U S T A C C O U N T S T A T E M E N T

10.2.1.3

DOC#: 0000831898
 LOCATION: E01-202-RB2121

Name: OLSEN, CHRISTOPHER L

DOB: 12/25/1980

DEBTS AND OBLIGATIONS

TYPE	PAYABLE	INFO NUMBER	AMOUNT OWING	AMOUNT PAID	WRITE OFF AMT.
TVRTD	TV RENTAL FEE DEBT	02172006	3.77	0.00	0.00
CVCS	CRIME VICTIM COMPENSATION/07112000	05212004	UNLIMITED	11.34	0.00
POSD	POSTAGE DEBT	03142006	4.53	0.00	0.00
UPSD	PERSONAL PROPERTY POSTAGE DEBT	06082004	7.84	0.00	0.00
COIS	COST OF INCARCERATION /07112000	05212004	UNLIMITED	35.59	0.00

TRANSACTION DESCRIPTIONS --

SPENDABLE BAL SUB-ACCOUNT

DATE	TYPE	TRANSACTION DESCRIPTION	TRANSACTION AMT	BALANCE
07/31/2012	HYGA	INMATE STORE DEBT (AUTO)	7.06	7.13
07/31/2012	CRS	CRS SAL ORD #6918256EAST	(7.06)	0.07
08/08/2012	DEND	DENTAL COPAY DEBT	4.00	4.07
08/08/2012	DEN	I05 - DENTAL COPAY	(4.00)	0.07
08/11/2012	TVD	TV CABLE FEE DEBT	0.50	0.57
08/11/2012	TV	I05 - TV CABLE FEE	(0.50)	0.07
08/14/2012	HYGA	INMATE STORE DEBT (AUTO)	58.03	58.10
08/14/2012	CRS	CRS SAL ORD #6933953EAST	(58.03)	0.07
08/14/2012	EVTRN	EVTRN- 4 TH OF JULY WINNER BAR UNITS	1.00	1.07
08/17/2012	POSD	POSTAGE DEBT	0.90	1.97
08/17/2012	POS	POSTAGE-INDIGENT 08/17/2012	(0.90)	1.07
08/27/2012	HYGA	INMATE STORE DEBT (AUTO)	10.20	11.27
08/27/2012	CRS	CRS SAL ORD #6951422EAST	(11.26)	0.01
08/27/2012	POSD	POSTAGE DEBT	1.10	1.11
08/27/2012	POS	POSTAGE-INDIGENT 08/27/2012	(1.10)	0.01
09/05/2012	POSD	POSTAGE DEBT	0.20	0.21
09/05/2012	POS	POSTAGE-INDIGENT 09/04/2012	(0.20)	0.01
09/08/2012	TVD	TV CABLE FEE DEBT	0.50	0.51
09/08/2012	TV	I05 - TV CABLE FEE	(0.50)	0.01
09/10/2012	HYGA	INMATE STORE DEBT (AUTO)	42.19	42.20
09/10/2012	CRS	CRS SAL ORD #6965737EAST	(42.19)	0.01
09/10/2012	MEDD	MEDICAL COPAY DEBT	4.00	4.01
09/10/2012	MED	I05 - MEDICAL COPAY	(4.00)	0.01
09/24/2012	HYGA	INMATE STORE DEBT (AUTO)	26.43	26.44
09/24/2012	CRS	CRS SAL ORD #6983506EAST	(26.43)	0.01
09/27/2012	POSD	POSTAGE DEBT	0.20	0.21
09/27/2012	POS	POSTAGE-Indigent 9/27/2012	(0.20)	0.01
10/05/2012	POSD	POSTAGE DEBT	0.20	0.21
10/05/2012	POS	POSTAGE-INDIGENT 10/05/2012	(0.20)	0.01
10/09/2012	HYGA	INMATE STORE DEBT (AUTO)	43.51	43.52
10/09/2012	CRS	CRS SAL ORD #7001470EAST	(43.51)	0.01
10/13/2012	TVD	TV CABLE FEE DEBT	0.50	0.51
10/13/2012	TV	I05 - TV CABLE FEE	(0.50)	0.01
10/17/2012	OTH	OTHER DEPOSITS-D. OLSEN	10.00	10.01
10/17/2012	DED	Deductions-LFO-20040728 D D	(0.01)	10.00

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WASHINGTON STATE PENITENTIARY

OTRTASTA

T R U S T A C C O U N T S T A T E M E N T

10.2.1.3

DOC#: 0000831898 Name: OLSEN, CHRISTOPHER L

DOB: 12/25/1980

LOCATION: E01-202-RB2121

DATE	TYPE	TRANSACTION DESCRIPTION	TRANSACTION AMT	BALANCE
10/22/2012	CRS	CRS SAL ORD #7018815EAST	(10.00)	0.00
10/29/2012	HYGA	INMATE STORE DEBT (AUTO)	33.17	33.17
10/29/2012	CRS	CRS SAL ORD #7027223EAST	(33.17)	0.00
11/10/2012	TVD	TV CABLE FEE DEBT	0.50	0.50
11/10/2012	TV	I05 - TV CABLE FEE	(0.50)	0.00
11/13/2012	HYGA	INMATE STORE DEBT (AUTO)	48.08	48.08
11/13/2012	CRS	CRS SAL ORD #7043864EAST	(48.08)	0.00
11/26/2012	HYGA	INMATE STORE DEBT (AUTO)	34.23	34.23
11/26/2012	CRS	CRS SAL ORD #7057698EAST	(34.23)	0.00
12/03/2012	POSD	POSTAGE DEBT	0.20	0.20
12/03/2012	POS	POSTAGE-INDIGENT 12/3/2012	(0.20)	0.00
12/08/2012	TVD	TV CABLE FEE DEBT	0.50	0.50
12/08/2012	TV	I05 - TV CABLE FEE	(0.50)	0.00
12/11/2012	HYGA	INMATE STORE DEBT (AUTO)	44.52	44.52
12/11/2012	CRS	CRS SAL ORD #7075342EAST	(44.52)	0.00
12/12/2012	POSD	POSTAGE DEBT	0.90	0.90
12/12/2012	POS	POSTAGE-INDIGENT 12/11/2012	(0.90)	0.00
12/13/2012	POSD	POSTAGE DEBT	0.90	0.90
12/13/2012	POS	POSTAGE-INDIGENT 12/13/12	(0.90)	0.00
12/20/2012	POSD	POSTAGE DEBT	0.65	0.65
12/20/2012	POS	POSTAGE-INDIGENT 12/19/2012	(0.65)	0.00
12/24/2012	POSD	POSTAGE DEBT	0.90	0.90
12/24/2012	POS	POSTAGE-INDIGENT 12/24/2012	(0.90)	0.00
12/26/2012	HYGA	INMATE STORE DEBT (AUTO)	28.44	28.44
12/26/2012	CRS	CRS SAL ORD #7092982EAST	(28.44)	0.00
12/31/2012	POSD	POSTAGE DEBT	0.45	0.45
12/31/2012	POS	POSTAGE-INDIGENT 12/31/2012	(0.45)	0.00
01/03/2013	POSD	POSTAGE DEBT	0.45	0.45
01/03/2013	POS	POSTAGE-INDIGENT 1/2/2013	(0.45)	0.00
01/04/2013	POSD	POSTAGE DEBT	0.90	0.90
01/04/2013	POS	POSTAGE-INDIGENT 1/3/2013	(0.90)	0.00
01/07/2013	HYGA	INMATE STORE DEBT (AUTO)	48.31	48.31
01/07/2013	CRS	CRS SAL ORD #7105414EAST	(48.31)	0.00
01/12/2013	TVD	TV CABLE FEE DEBT	0.50	0.50
01/12/2013	TV	I05 - TV CABLE FEE	(0.50)	0.00
01/17/2013	COPD	COPIES DEBT	4.00	4.00
01/17/2013	COP	COPIES-LEGAL	(4.00)	0.00
01/24/2013	POSD	POSTAGE DEBT	0.60	0.60
01/24/2013	POS	POSTAGE-INDIGENT 1/23/2013	(0.60)	0.00
01/24/2013	POSD	POSTAGE DEBT	0.45	0.45
01/24/2013	POS	POSTAGE-INDIGENT 01/24/2013	(0.45)	0.00
01/25/2013	COPD	COPIES DEBT	7.00	7.00
01/25/2013	COP	COPIES-legal	(7.00)	0.00
01/30/2013	LMD	LEGAL MAIL DEBT	1.32	1.32
01/30/2013	LM	LEGAL MAIL	(1.32)	0.00
02/04/2013	HYGA	INMATE STORE DEBT (AUTO)	57.06	57.06
02/04/2013	CRS	CRS SAL ORD #7138932EAST	(57.06)	0.00
02/05/2013	POSD	POSTAGE DEBT	0.46	0.46

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WASHINGTON STATE PENITENTIARY

OTRTASTA

T R U S T A C C O U N T S T A T E M E N T

10.2.1.3

DOC#: 0000831898 Name: OLSEN, CHRISTOPHER L

DOB: 12/25/1980

LOCATION: E01-202-RB2121

DATE	TYPE	TRANSACTION DESCRIPTION	TRANSACTION AMT	BALANCE
02/05/2013	POS	POSTAGE-INDIGENT 2/4/2013	(0.46)	0.00
02/06/2013	POSD	POSTAGE DEBT	0.46	0.46
02/06/2013	POS	POSTAGE-INDIGENT 2/6/2013	(0.46)	0.00
02/07/2013	POSD	POSTAGE DEBT	0.46	0.46
02/07/2013	POS	POSTAGE-INDIGENT 2/7/2013	(0.46)	0.00
02/09/2013	TVD	TV CABLE FEE DEBT	0.50	0.50
02/09/2013	TV	I05 - TV CABLE FEE	(0.50)	0.00
02/11/2013	POSD	POSTAGE DEBT	0.46	0.46
02/11/2013	POS	POSTAGE-INDIGENT 2/8/2013	(0.46)	0.00
02/12/2013	POSD	POSTAGE DEBT	0.92	0.92
02/12/2013	POS	POSTAGE-INDIGENT 2/11/2013	(0.92)	0.00
02/13/2013	POSD	POSTAGE DEBT	0.46	0.46
02/13/2013	POS	POSTAGE-INDIGENT 2/12/2013	(0.46)	0.00

TRANSACTION DESCRIPTIONS --

SAVINGS BALANCE SUB-ACCOUNT

DATE	TYPE	TRANSACTION DESCRIPTION	TRANSACTION AMT	BALANCE
TRANSACTION DESCRIPTIONS --			WORK RELEASE	SUB-ACCOUNT
			SAVINGS	

DATE	TYPE	TRANSACTION DESCRIPTION	TRANSACTION AMT	BALANCE
TRANSACTION DESCRIPTIONS --			EDUCATION ACCOUNT	SUB-ACCOUNT

DATE	TYPE	TRANSACTION DESCRIPTION	TRANSACTION AMT	BALANCE
TRANSACTION DESCRIPTIONS --			MEDICAL ACCOUNT	SUB-ACCOUNT

DATE	TYPE	TRANSACTION DESCRIPTION	TRANSACTION AMT	BALANCE
TRANSACTION DESCRIPTIONS --			POSTAGE ACCOUNT	SUB-ACCOUNT

DATE	TYPE	TRANSACTION DESCRIPTION	TRANSACTION AMT	BALANCE
09/17/2012	LMPOST	LEGAL MAIL - POSTAGE SUBACCOUNT	(0.45)	2.15
10/29/2012	SPOST	POSTAGE SUBACCOUNT WITHDRAWAL	(0.20)	1.95
11/05/2012	SPOST	POSTAGE SUBACCOUNT WITHDRAWAL	(0.45)	1.50
11/13/2012	SPOST	POSTAGE SUBACCOUNT WITHDRAWAL	(0.20)	1.30
11/15/2012	SPOST	POSTAGE SUBACCOUNT WITHDRAWAL	(0.20)	1.10
12/03/2012	SPOST	POSTAGE SUBACCOUNT WITHDRAWAL	(0.20)	0.90
01/14/2013	LMPOST	LEGAL MAIL - POSTAGE SUBACCOUNT	(0.20)	0.70
01/31/2013	LMPOST	LEGAL MAIL - POSTAGE SUBACCOUNT	(0.20)	0.50

TRANSACTION DESCRIPTIONS --

COMM SERV REV SUB-ACCOUNT
FUND ACCOUNT

DATE	TYPE	TRANSACTION DESCRIPTION	TRANSACTION AMT	BALANCE
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Ground # II

I was denied my 6th amendment right to effective assistance of counsel when my attorney Richard A. Woodrow failed to request that a juror be removed for lying (committing misconduct) during my trial

Effective assistance of counsel is guaranteed by the 6th amendment to the U.S. Constitution, *Strickland v. Washington*, 466 U.S. 668 (1984). To establish that trial counsel's representation was constitutionally inadequate, Olsen must show that counsel's representation performance was deficient - i.e., that it fell below an objective standard of reasonableness - and that the deficient performance was prejudicial. *Strickland*, 466 U.S. at 687-88. The proper measure of attorney performance is reasonableness under prevailing professional norms. *Id.* at 688. In order to demonstrate prejudice arising from deficient counsel's deficient performance, Olsen must show that there is a reasonable probability that, but for counsel's errors, the result of the proceeding would have been different. *Strickland* 466 U.S. at 694. A reasonable probability is a probability sufficient to undermine confidence in the outcome. *Id.* the "reasonable probability" standard is not stringent, and requires a showing by less than a preponderance of the evidence that the outcome of the proceeding would have been different had the claimant's rights not been violated. See, e.g., *Pitler v. Morgan*, 313 F.3d 1160, 1172 (9th Cir. 2002), cert. denied, 539 U.S. 916 (2003), quoting *Strickland*, 466 U.S. at 694:

A "reasonable probability" is less than a preponderance:
"the result of a proceeding can be rendered unreliable, and hence the proceeding itself unfair, even if the errors of counsel cannot be shown by a preponderance of the evidence to have determined the outcome."

In recent years, trial counsel's duty to thoroughly investigate prior to making reasonable trial tactic calculations, has been clearly defined. See, e.g., *Rompilla v. Beard*, 545 U.S. 374 (2005); *Wiggins v. Smith*, 539 U.S. 510 (2003); *Williams v. Taylor*, 529 U.S. 362, 395-96 (2000). These three cases applied the Strickland rule - that counsel must conduct a competent investigation before making tactical choices.

In this case, defense Counsel was aware of Olsen's prior contact with the juror in question prior to trial. In fact Olsen notified his counsel during jury selection that He (Olsen) not only knew the juror ("Vince") from the jail church services, but in fact prayed with said juror during jail church services about the very case that the jury was being selected for. However, trial counsel failed to further investigate Olsen's claim and even declined to question the juror, about the lie he told on June 16, 2008, when the judge asked Him if he wanted to talk to the juror. ^{see attached #6} Had Counsel questioned the juror he could have proved that the juror lied not only about never seeing Mr. Olsen during church services but also when asked during Voir dire if he had any prior knowledge of the crime or the defendants involved in said crime.

Ground # III

Olsen was denied his 6th amendment right to effective assistance of counsel, when counsel failed to elicit testimony of Olsen's impairment at the time of the crime - Testimony that would have merited a Voluntary intoxication instruction.

Olsen now supplies sufficient evidence that his level of intoxication affected his ability or lack thereof to form the mental state required to establish the crimes charged. The evidence presented here was available to defense counsel had counsel either conducted a competent investigation or elicited testimony from petitioner. Given that Olsen's ingestion of mind altering substances was known to his jurors, there was no tactical reason to fail to present a case justifying a voluntary intoxication instruction. The fact that Counsel attempted to submit a Manslaughter 2^o instruction indicates his belief that Olsen would be found guilty of something. In any event, Olsen has certainly presented sufficient evidence for Court to remand this claim to the trial court for an evidentiary hearing. RAP 16.11.

At trial Olsen testified he was "high" on Methamphetamine and alcohol on the night of his release from jail. The same night he was alleged to have participated in this crime (see RP@83^{line 4} meth). He also testified that he had been using ^{meth} since he was 17 years old (RP 857 lines 3 and 4). However, trial counsel did not delve into this issue deeper - likely due to his failure to sufficiently investigate, including failing to sufficiently speak ^{with} to Mr Olsen

through 864 line 9
and 879 lines
14-25
and 954 lines
15-25

about his intoxication and mental state, at any point during his involvement in the alleged crime, prior to trial.

Mr. Olsen has a long, unfortunate history of drug and alcohol abuse. Due to his excessive and chronic use of substances, Olsen has suffered a few "blackouts". In addition, he suffers from extreme emotional swings and paranoia, common side effects of substance addiction see declaration of Olsen. During the months leading up to his initial arrest for the possession charge 12/4/06, Olsen was using between 10-13 grams of methamphetamine per day. As a result, Olsen went without sleep for many days, became "extremely paranoid and had episodes of broken reality and suicidal thoughts. Even while incarcerated in the Thurston County Jail between 12/04/06 and 1/29/07 Mr. Olsen was using meth every two or three days and on 1/29/07 (the date of his alleged participation in the crime for which he was convicted (on 7/23/08) He used about 6 1/2 grams of methamphetamine, 6-8 ounces of "patron" tequila, and a couple 1/2 pills of ecstasy at the little creek casino hotel.

This evidence if introduced at trial would have been sufficient to support a voluntary intoxication instruction.

However, there was more.

The effect of this massive ingestion of mind altering substance is evident from evidence both introduced at trial by states witness April Frazier (see RP 627 lines 12, 15, 628, 5-8) ^(blackmont #11) and available to defense counsel but not introduced for reasons unknown.

Ms. Frazier, the state's witness against Mr Olsen, also could ^{have} provided additional support as to Mr. Olsen's level of intoxication as evidenced by her observations of him at the Little Creek Casino the night he was bailed out by her and Mr. Sublett. It would have been easy to support this testimony with an experts opinion about the effects of these substances especially the methamphetamine, See attachments 7-9

Methamphetamine increases the release and blocks the reuptake of the brain chemical dopamine, leading to high levels of the chemical in the brain, a common mechanism of action for most drugs of abuse. Dopamine is involved in reward, motivation, the experience of pleasure, and motor function. Methamphetamine's ability to rapidly release dopamine in reward regions of the brain produces the intense euphoria or "rush", that many users feel after snorting, smoking, or injecting the drug. Attachment 7.

Methamphetamine enters the brain quickly and lingers longer than other similar drugs of abuse. Attachment 8.

Most importantly, methamphetamine is highly correlated with "Severe neurological and psychiatric adverse events, including the development of psychotic states." Attachment 9.

There was certainly evidence suggesting Olsen was psychotic at the time of the alleged participation in this crime. He was obviously still psychotic the day after his alleged participation in said crime as evidenced on page 35 of Ms Frazier's taped statement: lines 1-5 "Chris started crying and he was sitting on the

Floor underneath the dining room table and I just kept asking him, "are you okay?" are, you know, I mean, it really seemed to me like he had flipped out, Psychotically flipped out. He wasn't making, he was talking but he wasn't saying real words. He was sort of mumbling and crying" see attachment #13

However, without the necessary context and supporting evidence, these facts alone were insufficient for a voluntary intoxication instruction, had one been requested. However, adding the new evidence, available if only a minimally competent investigation had been undertaken, the evidence is clearly sufficient - not only for an instruction, but which raises a reasonable likelihood of a different outcome on at the very least the underlying felony that was the basis for the felony murder conviction

In short, evidence was available that would have supported a viable defense of voluntary intoxication.

"a criminal defendant is entitled to a voluntary intoxication instruction if: (1) one of the elements of the crime charged is a particular mental state; (2) there is substantial evidence of ingesting an intoxicant; and (3) the defendant presents evidence that this activity affected his ability to acquire the required mental state." State v. Harris, 122 Wn. app. 547, 552, 90 P.3d 1133 (2004) In other words, The evidence must reasonably and logically connect Olsen's intoxication with his asserted inability to form the requisite level of culpability to commit any of the underlying felonies that resulted in the death of the victim, ultimately resulting in the felony murder alternative. See State v. Griffin 100 Wn.2d 417,

418-19, 670 P.2d 265 (1983); State v. Kruger, 116 Wn. app. 685, 692, 67 P.3d 1147 (2003) (stating that mere intoxication isn't enough; rather, the evidence must show the effects of the intoxicant).

Although an expert opinion would have been most helpful, if counsel had conducted even an expert-less investigation and presented the evidence available, he could have obtained an instruction. But "if the issue involves a matter of common knowledge about which inexperienced persons are capable of forming a correct judgment, there is no need for expert opinion" State v. Smissaert, 41 Wash. app. 813, 815, 706 P.2d 647 (1985) and "certainly the effects of alcohol upon people are commonly known and all persons can be presumed to draw reasonable inferences therefrom." Id So where the court gives a voluntary intoxication instruction, the defendant can argue diminished capacity without an expert. Id

In this case defense counsel was aware of Olsen's intoxication prior to trial. However, trial counsel failed to further investigate, and as a result failed to present Olsen's available evidence. In fact trial counsel failed to request an instruction supporting a diminished capacity defense. As a result Olsen's jury was left with evidence of illegal drug use, but no vehicle to apply it to diminish Olsen's legal responsibility. In other words, counsel drew out all of the prejudicial nature of drug use, but sought none of the potential legal benefit. Washington courts have acknowledged that evidence of drug use - standing alone - is highly prejudicial. "in view of society's deep concern today with

drug usage and its consequent condemnation by many if not most, evidence of drug addiction is highly prejudicial in the minds of the average juror." State v. Renneberg 83 Wn. App. 753, 737, 522 P.2d 835 (1974). Thus, aside from laying the groundwork for a Voluntary intoxication defense, the voluntary introduction of drug use cannot be pinned to any legitimate Strategic Consideration. Without any testimony to explain the impact of ~~the~~ drug use on his state of mind, accompanied by an instruction allowing a jury to give effect to that condition, trial counsel left Olsen's jury with the Solid impression that he was simply a "law breaking drug user." See State v. Powell, 139 Wn. App. 808, 162 P3d 1180 (2007)

Aided by an evidentiary hearing (and the right to expert assistance that accompanies it pursuant to RAP 16.12), Olsen will be able to thoroughly establish that his level of intoxication and its effect on his state of mind would have supported a voluntary intoxication instruction - evidence that must under mine confidence in the verdict.

Thus, if the state contests Olsen's evidence in this claim, this court should remand for an evidentiary hearing. RAP 16.11 Otherwise, this court should grant Olsen's petition and remand for a new trial.

Ground IV

Mr Olsen was deprived of his 6th amendment right to effective assistance of counsel when counsel failed to investigate and call an expert witness who could have given testimony connecting Olsen's extreme intoxication with his corresponding ability, or lack of ability, to form the criminal intent required for conviction.

Although Mr. Olsen disputes his involvement with the murder itself, He did not dispute the fact that he was involved in criminal conduct after the murder occurred. So for Mr Olsen the issue was one of the degree of crimes committed. Counsel even sought (although was not successful in obtaining) lesser included offenses instructions. Therefore Olsen's state of mind would have been the best vehicle for defense had counsel conducted a competent investigation. Both the initial charge and the alternative carried great penalties and premeditated 1st murder is a specific intent crime as are the underlying felonies that form the basis for a felony murder conviction. Defense counsel asked for a 2nd manslaughter instruction (See RP @ 955 lines 11- 956 line 5 06-17-08)

Given this backdrop, counsel's failure is astonishing. The jury being presented with information about the affects of Olsen's drug ingestion on his state of

mind at the time of the crime would have been very powerful.

There was ample evidence available to establish Olsen's high level of intoxication, a point discussed in the previous claim of error. Counsel elicited testimony from Olsen that he was using drugs with the co-defendants.

Ms. Frasier (The state's primary witness against Mr. Olsen) testified that "[after Mr. Olsen was bailed out]" "We started drinking and getting high" "on methamphetamine" (See attachment #2 RP@ 520 lines 16-19) ⁰⁶⁻¹⁰⁻⁰⁸ and that "Mr. Olsen was sitting under the kitchen table with his knees drawn up and he was crying" (See RP@ 628 lines 12-15 06-10-08) and statement of A. Frasier page 35 lines 1-5) She told detectives and the state's prosecutor that Olsen had "flipped out, psychotically flipped out" (statement of A. Frasier page 35 lines 1-5)

Counsel even talked about Mr. Olsen being a drug addict and getting high on meth upon his release from jail, during his closing argument. However Counsel failed to conduct a competent investigation and failed to call an expert witness to testify about Olsen's impaired state of mind.

As indicated earlier there is no shortage of documentation available to establish the drastic effect of methamphetamine on the brain. The drug even in low to moderate amounts, causes anxiety, confusion, and mood disturbances and can lead to violent behavior. With chronic use and/or the consumption of larger doses, methamphetamine causes psychotic features, including paranoia, visual and auditory hallucinations. (See Attachments 7, 8, 9)

All of these disturbing consequences were on display

on the night of the crime. As a result it was deficient for trial counsel not to consult with an expert. Indeed, that expert would not necessarily need to assess Mr Olsen. It would have been sufficient, although not preferable, to discuss the impact of the amount of drugs ingested on the human central nervous system.

Counsel must, at a minimum, conduct a reasonable investigation enabling him to make an informed decision about how to best represent his client. In *Re Pers. restraint of Brett*, 142 Wn.2d 868, 873, 16 P.3d 601 (2001); *Serdel v. Merkel*, 146 F.3d 750, 755 (9th Cir. 1998).

If investigated, the evidence would have been admissible. 1 degree Murder is a specific intent crime. Thus, evidence of an inability to form the requisite intent is admissible. *State v. Martin*, 14 Wn. App. 74, 538 P.2d 873 (1975) Intoxication is an accepted basis for arguing lack of ability to form the requisite intent. RCW 9A.16.090. The right to present evidence includes the right to expert testimony.

A case particularly on point is *Miller v. Terhune*, 510 F. Supp.2d 486 (E.D. Cal. 2007) The central issue before the court in that case was whether petitioner's trial counsel rendered ineffective assistance when they failed to investigate and present evidence as to how petitioner's level of intoxication likely affected his perceptions, intentions, and actions on the night of the shooting.

After finding that counsel failed to conduct a minimally competent investigation (counsel did consult with mental health professionals, unlike this case), the court held:

"In sum, the record reflects that counsel failed to investigate the effects of intoxication on the petitioner. Accordingly, Counsel was in no position to make a reasoned or strategic decision regarding the use of intoxication evidence. It is well settled that under Strickland, "attorneys have considerable latitude to make strategic decisions about what investigations to conduct once they have gathered sufficient evidence upon which to base their tactical choices." *Jennings v. Woodford*, 290 F.3d 1006, 1014 (9th Cir. 2002). In the instant case, there is simply no indication that defense counsel gathered any evidence upon which to base their decision to not investigate or present evidence of intoxication. See *Williams*, 529 U.S. at 369, 120 S. Ct. 1495 (Counsel must conduct a "thorough investigation" before decision can be considered strategic under Strickland); *Sanders*, 21 F.3d at 1457 (citing *United States v. Gray*, 878 F.2d 702, 711 (3rd Cir. 1989)) (finding that "... Counsel can hardly be said to have made strategic choice when he has not obtained the facts on which a decision could be made.")

The ABA Standards for Criminal Justice provide guidance as to the obligations of criminal defense attorneys in conducting an investigation. *Rompilla v. Beard*, 545 U.S. 374, 125 S. Ct. 2456, 162 L. Ed. 2d 360 (2005); *Williams*, 529 U.S. at 396, 120 S. Ct. 1495. The standards in effect at the time of petitioner's trial clearly described the defense lawyers duty to investigate: (a) defense counsel should conduct a prompt investigation of the circumstances of the case and explore all avenues leading to facts relevant to the merits of the case and the penalty

in the event of conviction. The investigation should include efforts to secure information in the possession of the prosecution and law enforcement authorities. The duty to investigate exists regardless of the accused's admissions or statements to defense counsel of facts constituting guilt or the accused's stated desire to plead guilty.

ABA Standards for Criminal Justice, Defense Functions, Standard 4-4.1 3d Ed.).

When trial counsel is on notice that his client may have a particular mental impairment relevant to the case, the Ninth Circuit has repeatedly held that failure to investigate the mental state constitutes deficient performance under Strickland. See, e.g., *Douglas v. Woodford*, 316 F.3d 1079, 1085 (9th Cir. 2003) (citing *Bean v. Calderon*, 163 F.3d 1073, 1078 (9th Cir. 1998) (holding that "[t]rial counsel has a duty to investigate a defendant's mental state if there is evidence to suggest that the defendant is impaired.") see also *Caro v. Woodford*, 280 F.3d 1247, 1254 (9th Cir. 2002); *Hendricks v. Calderon*, 70 F.3d 1032, 1043 (9th Cir. 1995). In such circumstances, counsel must undertake at least "a minimal investigation in order to make an informed decision regarding the possibility of a defense based on mental health" *Seidel v. Merkle*, 146 F.3d 750, 756 (9th Cir. 1998). *Id.* at 499.

In *Jennings*, one of the cited cases, the 9th Circuit concluded that defense counsel was ineffective when counsel failed to investigate Jennings' use of methamphetamine and alcohol on the night of the crime, despite the fact that Jennings was insistent on an alibi defense. The court concluded that the decision to pursue the alibi defense was uninformed and

Therefore unreasonable. *Id.* at 1014.

Had defense counsel in this case simply looked to the relevant caselaw, he would have discovered *State v. Kruger*, *Supra*, a case with remarkably similar facts *Id.* at 692 ("The record reflects substantial evidence of Mr. Kruger's drinking and level of intoxication and there is ample evidence of his level of intoxication on both his mind and body. He was entitled to the instruction").

Had counsel conducted a simple search of the caselaw for "Methamphetamine" and "Mental State", he would have found a plethora of cases discussing, with approval, the use of an expert to opine about the interaction of drugs on the brain. See e.g., *State v. Ferrick*, 81 Wn.2d 942, 944, 506 P.2d 860 (1973); *State v. Coates*, 107 Wn.2d 882, 735 P.2d 64 (1987); *State v. Griffin*, 100 Wn.2d 417, 419, 670 P.2d 265 (1983); *State v. Hansen*, 46 Wn. App. 292, 730 P.2d 706 (1987); *State v. Thomas*, 109 Wn.2d 222, 743 P.2d 816 (1987); *State v. Cienfuegos*, 144 Wn.2d 222, 25 P.3d 1011 (2001).

Thus, Olsen has presented at least a *prima facie* claim of error. As before, he is entitled to an evidentiary hearing or reversal, if the state does not dispute these facts with their own competent evidence.

Ground V

Mr. Olsen was denied his 6th amendment right to effective assistance of counsel when counsel opened the door to a line of questioning that led to the prosecution being able to bring up a prior conviction for possession of a firearm by Mr. Olsen.

Prior to trial, the understanding was only Mr. Olsen's prior crimes of dishonesty (the 2 forgeries) would be brought before the jury for impeachment reasons.

When Olsen was on the stand, Counsel asked Mr. Olsen "There's been some conversation about a gun on the recorded phone calls. What kind of gun was that?" to which Olsen responded "That was a 25 millimeter flare gun from boaters world." and again on redirect "when you were talking about the gun that's on the recorded telephone call, did you make a comment that the gun would make things toasty?" to which Olsen responds "Yes, I did." Counsel: "What did you mean by that?" Olsen: "Well, it's a flare gun with a 25 millimeter flare in it. That's -- a really, really hot ball of fire basically is what comes out of the end of that gun." (See RP 915 at lines 14-21 06-16-08) which led to prosecution further questioning Olsen about the gun and opening the door to his 2006 conviction for unlawful possession of a firearm². (See RP 918 line 3 through 924 line 3) Thus making Olsen out to be both a career criminal and a liar.

This is a very straight forward case

(See RP
855 lines 23-25
06-16-08)

Of attorney incompetence. It was obviously not tactical, Counsel argued that he did not open the door, thus the deficient performance prong is easily satisfied. Prejudice, which is defined as undermining confidence in the verdict, is also established. Washington Caselaw is clear that "prior conviction evidence is inherently prejudicial when the defendant is the witness because it tends to shift the jury focus from the merits of the charge to the defendant's general propensity for criminality." State v. Jones, 101 Wn.2d 113, 120, 677 p.2d 131 (1984) overruled on other grounds State v. Brown, 111 Wn.2d 124, 761 p.2d 588 (1988). However this court must add to the inherent prejudice, the state's exploitation of this error all throughout their questioning and into their closing the state repeatedly and unfairly attacked Olsen's honesty. Thus, this serious misstep by counsel inflicted a great blow on the jury's assessment of Olsen. If he was willing to lie by minimizing his criminal history, he was also likely not honest in describing his own involvement in the events at issue. In the end, Counsel's blunder injured Olsen's credibility more than any cross examination, by itself, ever could.

Olsen has established his claim of ineffective assistance of counsel.

He is entitled to a new trial.

Ground VI

Mr. Olsen is entitled to a new trial based on the cumulative prejudice from multiple errors, including the multiple failures of defense counsel.

Where the cumulative effect of multiple errors so infected the proceedings with unfairness a resulting conviction or death sentence is invalid.

(See *Kyles v. Whitley*, 514 U.S. 419, 434-35, 115 S.Ct. 1555, 131 L. Ed. 2d 490 (1995). As the 9th Circuit pointed out in *Thomas v. Hubbard*, 273 F.3d 1164 (9th Cir. 2001), "In analyzing prejudice in a case in which it is questionable whether any single trial error examined in isolation is sufficiently prejudicial to warrant reversal, this Court has recognized the importance of considering the cumulative effect of multiple errors and not simply conducting a balkanized, issue by issue harmless error review" *Id.* at 1178 (internal quotations omitted) (citing *United States v. Frederick*, 78 F.3d 1370, 1381 (9th Cir. 1996); see also *Matlock v. Rose*, 731 F.2d 1236, 1244 (6th Cir. 1984) ("Errors that might not be so prejudicial as to amount to a deprivation of due process when considered alone, may cumulatively produce a trial setting that is fundamentally unfair").

Olsen asserts that each of the errors described previously merits relief. However, considered cumulatively they certainly resulted in prejudice sufficient to merit a new trial. Trial counsel's errors, measured cumulatively, were devastating to Olsen, Counsel failed to investigate

and question a juror he knew to be being untruthful about knowledge of Mr. Olsen and the trial he was a deciding juror in, (that counsel was made aware of during jury selection.) and failed to have said juror removed after he lied during trial, and failed to at the very least question the juror to see if he could make an unbiased decision as to Olsen's guilt or innocence, failed to investigate and present compelling evidence of the extent of Olsen's intoxication, failed to obtain an instruction, opened the door to devastating evidence (making his client into a perjurer in the meantime), and failed to correct and request a mistrial due to the prosecutor's misconduct. Misconduct he was obviously aware of as evidenced by his objection to it during the state's closing.

Thus Olsen is entitled to a new trial

Memorandum in Support

A. Status of Petitioner

Christopher L. Olsen (hereinafter "Olsen") challenges his Thurston County convictions for Felony Murder¹ and the subsequent 500 month sentence (Thurston County Case No. 07-1-01363-0)

Mr. Olsen is currently confined at the Washington State Penitentiary in Walla Walla, Washington. This petition, filed within one year of the February 12th, 2013 mandate from Olsen's direct appeal, is his first collateral attack on his Judgment of Conviction

B. Facts

procedural history

On August 7, 2007, Mr. Olsen was charged by information with Premeditated¹ murder or, in the alternative¹ Felony murder, from an incident occurring six months earlier - on Jan of 2007. After numerous continuances Olsen eventually proceeded to jury trial in June 2008.

Olsen's jury convicted him of the alternative charge of Felony Murder¹. He was subsequently sentenced on July 23, 2008 to a total of 500 months in prison.

Mr. Olsen appealed. His conviction was affirmed on appeal. This court's decision was issued on May 18, 2010. Olsen then sought review by the State Supreme Court. This court's decision was issued on November 21, 2012

Fair trial

The state deprived me of the rights afforded to me by the 6th and 14th amendments to the United States constitution and article I, section 22 of the Washington state constitution

On June 17th 2008 State's Prosecutor David Harold Bruneau (WSBA #6830) knowingly used an unadmitted exhibit to knowingly incite and inflame the Jurys passion to gain a conviction in an unlawful manner.

The exhibit was a Jail booking photo which showed me in Jail house clothing (orange jumpsuit) which was altered via computer with the word "Guilty" highlighted in bold crimson red on a large projection screen facing the Jury

Knowing that [a ^{People} jury] are sometimes unable to rationally consider how images affect our emotions or our decision making process. Mr. Bruneau stated "What is that saying? A picture says a thousand words" further emphasizing the weight and value of the unadmitted and unlawfully used exhibit thus using manipulation ~~of~~ ^{through} visual communication to gain a guilty verdict Washington state supreme courts have repeatedly and unequivocally denounced the type of conduct ~~in this~~ ^{that} occurred in this case they have also found it to be a violation of a defendants right to fair trial protected by the 6th and 14th amendments to the United States constitution.

The violation that occurred in my case is the same that happened in State v. Glasmann (see In re ^{Glasmann} ~~Glasmann~~ Wa St sup ct No 84475-59 filed 10-18-2012). In Glasmann the supreme court reversed the defendants convictions and remanded for a new trial.

C. Argument

1. Mr. Olsen was Deprived of due process and the right to a fair trial when the prosecutor used highly inflammatory illustrations and injected personal opinions during closing arguments.

During closing arguments, the prosecutor presented a slideshow to the jury. 4 of these slides are attached. See attachments 5-5c, (obtained through a public disclosure request). One of these slides which speaks for itself, shows a photo of Olsen at booking (in jail clothes) and the word "guilty" superimposed on it.

Not only is the booking photo unfair, the prosecutor repeatedly expressed his personal opinion about the credibility of Olsen - a decision that must be left in the exclusive hands and minds of jurors.

In order to establish prosecutorial misconduct, a defendant must prove that the prosecutor's conduct was improper and that it prejudiced his right to a fair trial. *State v. Carver*, 122 Wash. App. 300, 306, 93 P.3d 947 (2004) (citing *State v. Dhalwal*, 150 Wash.2d 559, 578, 79 P.3d 432 (2003)). A defendant can establish prejudice only if there is a substantial likelihood that the misconduct affected the jury's verdict. *Carver*, 122 Wash. App. at 306, 93 P.3d 947 (quoting *Dhalwal*, 150 Wash.2d at 578, 79 P.3d 432). If defense counsel fails to object to the prosecutor's statements, then reversal is required only if the misconduct was so flagrant and ill intentioned that no instruction could have cured the resulting prejudice. (State

v. Belgarde, 110 Wash.2d 504, 508, 755 P.2d 174 (1988).

It is improper for a prosecutor to personally vouch for a witness's credibility. See State v. Brett, 126 Wash.2d 136, 175, 892 P.2d 29 (1995) Cert. denied, 516 U.S. 121, 116 S.Ct. 931, 133 L.Ed.2d 858 (1996). Prosecutor's may, however, argue an inference from the evidence and this court will not find prejudicial error "unless it is ~~un~~clear and unmistakable that counsel is expressing a personal opinion" Brett, 126 Wash.2d at 175, 892 P.2d 29 (quoting State v. Sargent, 40 Wash. app. 340, 344, 698 P.2d 598 (1985)).

This Court views the "prejudicial or inflammatory effect" of the improper remarks "in context with the earlier evidence and the circumstances of the trial in which they were made." State v. Green, 71 Wn.2d 372, 381, 428 P.2d 540 (1967).

In this case, read in context, and especially considering the visual display accompanying the prosecutor's words, the argument was both inflammatory and unfair. Although the petitioner could make nuanced arguments about the impropriety of the argument, the picture of Olsen's booking photo accompanied by the superimposed word "Guilty" says it all. This Court should condemn such unfair arguments; In In re Glasmann, 175 Wn.2d 696, — P.3d — (2012) The Washington State Supreme Court said that "our courts have repeatedly and unequivocally denounced the type of conduct that occurred in this case." "First, we have held that it is error to submit evidence to the jury that has not been admitted at trial. State v. Pete, 152 Wn.2d 546, 553-55, 98 P.2d 803 (2004) The "long-standing rule" is that "consideration of any material by a jury not ~~admitted~~ properly admitted as evidence vitiates a

verdict when there is reasonable ground to believe that the defendant may have been prejudiced. *Id.* at 555 n.4 (quoting *State v. Rinkes*, 70 Wn.2d 854, 862, 425 P.2d 658 (1967)) (emphasis omitted). . . . "Although this is not a case where unadmitted evidence was sent to the jury room as in *Pete and Rinkes*, these cases nevertheless establish that a prosecutor must be held to know that it is improper to present evidence that has been deliberately altered, in order to influence the jury's deliberations." *In re Glasmann*, 175 Wn.2d 696, 706, P.3d (2012)

"During critical closing moments of trial, one of the last things the jury saw before it began ^{its} deliberations was the representative of the state of Washington, impermissibly flashing the word "guilty" over across an image of Glasmann's face, . . . predisposing the jury to return a harsh verdict." *Id.* at 709.

As in the Washington State Supreme Court's decision in *Glasmann* this court should reverse Olsen's convictions and remand for new trial.

175 WA.2d 646

P 705 Attachment # 2

P 099

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1/15/13 (initials)

IN THE SUPREME COURT OF THE STATE OF WASHINGTON

In the Matter of the Personal Restraint of)	No. 84475-5
)	
EDWARD MICHAEL GLASMANN,)	En Banc
)	
Petitioner.)	Filed October 18, 2012
_____)	

¶ 1 MADSEN, C.J.—Edward M. Glasmann was convicted of second degree assault, attempted second degree robbery, first degree kidnapping, and obstruction arising from incidents that occurred while he was intoxicated. During closing argument, the prosecuting attorney made an electronic presentation to the jury that graphically displayed his personal opinion that Glasmann was “guilty, guilty, guilty” of the crimes charged by the State. The prosecutor’s misconduct was flagrant, ill intentioned, and we cannot conclude with any confidence that it did not to have an effect on the outcome of the trial. We reverse the defendant’s convictions and remand for a new trial.

FACTS AND PROCEDURAL HISTORY

¶ 2 In celebration of his October 2004 birthday, Edward Glasmann and his fiancée, Angel Benson, rented a motel room in Lakewood, Washington. Over the course of the

evening, the two ingested methamphetamine, ecstasy, and alcohol. Glasmann and Benson had been arguing throughout that day and evening and around midnight, their argument escalated. Glasmann started punching and kicking Benson. He told Benson he wanted to go for a ride and then dragged her out of the motel room. Outside the motel room, another motel guest witnessed Glasmann punch and kick Benson before dragging her to the passenger side of his Corvette. This witness called 911 and provided an account of the events.

¶ 3 From the driver's seat, Glasmann reached over to open the passenger door and attempted to pull Benson into the car by her hair. Benson testified that she was partially in the car and stumbled when Glasmann ran the car up her leg, backed off of her leg, pulled her into the car, and drove out of the parking lot. Benson was then able to get the car into park. She next grabbed the car keys and ran into a minimart adjacent to the motel. F 2010

¶ 4 Inside the minimart, she hid on the floor behind the cashier's counter. Police soon arrived and attempted without success to apprehend Glasmann. Shouting at the officers to shoot him and claiming to possess a firearm, Glasmann ran into the convenience store. He ran behind the counter, held Benson in a choke hold, and threatened to kill her. As officers approached, Glasmann held Benson between himself and the officers. Benson was able to wiggle free enough to allow an officer to use a stun gun on Glasmann.

¶ 5 The officers subdued and arrested Glasmann. In the process, Glasmann was held

down by one officer while another officer stomped on his head approximately five times. Glasmann continued to struggle as he was dragged out of the minimart. His booking photograph shows extensive facial bruising. The incident inside the minimart was recorded on the store's security camera.

¶ 6 The State charged Glasmann with first degree assault, attempted first degree robbery, first degree kidnapping, and obstruction. Exhibits admitted into evidence included the minimart security video, photographs of Benson's injuries, the 911 recording, recordings of telephone calls between Glasmann and Benson, and Glasmann's booking photo. The defense offered Glasmann's booking photo to display Glasmann's facial injuries sustained during arrest.

¶ 7 At trial, Glasmann did not deny culpability. Rather, he disputed the degree of the crimes charged. He argued the jury should convict only on lesser included offenses. The prosecution sought to establish that Glasmann acted with intent, a necessary element of all the crimes charged.

¶ 8 In closing argument, the State used an extensive PowerPoint¹ presentation that included numerous slides incorporating the security camera video, audio recordings, photographs of Benson's injuries, and Glasmann's booking photograph. Each of the slides containing a video shot or photograph included a caption consisting of testimony, P 701

¹ "PowerPoint" is a registered trademark of a Microsoft graphics presentation software program.

recorded statements, or the prosecutor's commentary.²

99 One slide showed Glasmann crouched behind the minimart counter with a choke hold on Benson and a caption reading, "YOU JUST BROKE OUR LOVE." State's Resp. to Pers. Restraint Pet. (PRP), App. G at 1. Another slide featuring a photograph of Benson's back injuries appeared with the captions, "What was happening right before defendant drove over Angel . . . ," and ". . . you were beating the crap out of me!" *Id.* at 2. This slide also featured accompanying audio.

10 In addition, the prosecutor argued that jurors should not believe Glasmann's testimony. He told the jurors that the law required them to "[c]ompare Angel Benson's testimony and the testimony of the remainder of the State's witnesses to the defendant's." 8 Verbatim Report of Proceedings (VRP) at 458. The prosecutor then told jurors that in order to reach a verdict they must determine: "Did the defendant tell the truth when he testified?" *Id.*

11 At least five slides featured Glasmann's booking photograph and a caption. In one slide, the booking photo appeared above the caption, "DO YOU BELIEVE HIM?" State's Resp. to PRP, App. G at 5. In another booking photo slide the caption read,

² Having been obtained by public disclosure request, most of the prosecution's closing argument PowerPoint slides are attached to State's Response to Personal Restraint Petition, Appendix G (Wash. Ct. App. No. 39700-5-II). Although appendix G includes two versions of the presentation, we cite only to the shorter version, appearing second in the appendix. Three of the closing argument slides are attached to the Personal Restraint Petition, Appendix H at 8-10. None of the original slides are in the record.

“WHY SHOULD YOU BELIEVE ANYTHING HE SAYS ABOUT THE ASSAULT?”

Id. Near the end of the presentation, the booking photo appeared three more times: first with the word “GUILTY” superimposed diagonally in red letters across Glasmann’s battered face. PRP, App. H at 8. In the second slide the word “GUILTY” was superimposed in red letters again in the opposite direction, forming an “X” shape across Glasmann’s face. *Id.* at 9. In the third slide, the word “GUILTY,” again in red letters, was superimposed horizontally over the previously superimposed words. *Id.* at 10. As best as we can determine, the prosecutor stated the following while the “GUILTY” slides were being displayed:

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You’ve been provided with a number of lesser crimes if you believe the defendant is not guilty of the crimes for which the State has charged him, but the evidence in this case proves overwhelmingly that he is guilty as charged, and that’s what the State asks you to return in this case: Guilty of assault in the first degree; guilty of attempted robbery in the first degree; guilty of kidnapping in the first degree; and guilty of obstructing a police officer. Hold him accountable for what he did on October 23rd, 2004, by finding him guilty as charged. Thank you.

8 VRP at 465-66. Defense counsel did not object to these slides.

¶12 In closing argument, defense counsel emphasized the governing standard, proof beyond a reasonable doubt. He asked the jurors to focus on the actual charges, not Glasmann’s drug use, reckless driving, or “hitting Angel Benson in the motel room.” *Id.* at 470. Counsel reviewed the elements of each charge and argued that Glasmann’s conduct did not meet the definition of the charged crimes:

The issue for you to decide is[,] is there proof beyond a reasonable doubt that Mike Glasmann committed any crimes that night, and the answer to that is yes, but this case is overcharged.

What do I mean by that? I mean that the charges that the State has leveled against Mr. Glasmann are not reflective of what, in reality, happened that night or reflective of what has been proven beyond a reasonable doubt happened that night. He's charged with Assault 1 when only assault in the third degree or assault in the fourth degree reasonably fit these facts, arguably, beyond a reasonable doubt. He's charged with attempted robbery in the first degree when only attempted robbery in the second degree fits these facts beyond a reasonable doubt. He's charged with kidnapping in the first degree when only unlawful imprisonment fits these facts beyond a reasonable doubt. Obstructing a law enforcement officer is, I said, a proper charge.

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Id. at 494.

¶ 13 The jury convicted Glasmann of first degree kidnapping and obstruction, and the lesser included offenses of second degree assault and attempted second degree robbery. Glasmann appealed. He was sentenced to 210 months in prison. The Court of Appeals affirmed in an unpublished decision. *State v. Glasmann*, noted at 142 Wn. App. 1041, 2008 WL 186783. Thereafter, Glasmann filed a personal restraint petition and we granted review limited to whether the prosecutor's closing argument deprived Glasmann of a fair trial and whether assistance of Glasmann's trial counsel was ineffective.³ *In re Pers. Restraint of Glasmann*, 170 Wn.2d 1009, 245 P.3d 226 (2010).

ANALYSIS

³ We need not reach the ineffective assistance of trial counsel claim because we remand for a new trial based on the prosecutorial misconduct claim.

[1,2] 914 The right to a fair trial is a fundamental liberty secured by the Sixth and Fourteenth Amendments to the United States Constitution and article I, section 22 of the Washington State Constitution. *Estelle v. Williams*, 425 U.S. 501, 503, 96 S. Ct. 1691, 48 L. Ed. 2d 126 (1976); *State v. Finch*, 137 Wn.2d 792, 843, 975 P.2d 267 (1999). Prosecutorial misconduct may deprive a defendant of his constitutional right to a fair trial. *State v. Davenport*, 100 Wn.2d 757, 762, 675 P.2d 1213 (1984). “A “[f]air trial” certainly implies a trial in which the attorney representing the state does not throw the prestige of his public office . . . and the expression of his own belief of guilt into the scales against the accused.” *State v. Monday*, 171 Wn.2d 667, 677, 257 P.3d 551 (2011) (alteration in original) (quoting *State v. Case*, 49 Wn.2d 66, 71, 298 P.2d 500 (1956); see *State v. Reed*, 102 Wn.2d 140, 145-47, 684 P.2d 699 (1984)). P 704

[3-7] 915 Although a prosecutor has wide latitude to argue reasonable inferences from the evidence, *State v. Thorgerson*, 172 Wn.2d 438, 448, 258 P.3d (2011), a prosecutor must “seek convictions based only on probative evidence and sound reason,” *State v. Casteneda-Perez*, 61 Wn. App. 354, 363, 810 P.2d 74 (1991); *State v. Huson*, 73 Wn.2d 660, 663, 440 P.2d 192 (1968). “The prosecutor should not use arguments calculated to inflame the passions or prejudices of the jury.” American Bar Association, Standards for Criminal Justice std. 3-5.8(c) (2d ed. 1980); *State v. Brett*, 126 Wn.2d 136, 179, 892 P.2d 29 (1995); *State v. Belgarde*, 110 Wn.2d 504, 755 P.2d 174 (1988).

916 In order to prevail on a claim of prosecutorial misconduct, a defendant is required to show that in the context of the record and all of the circumstances of the trial, the

prosecutor's conduct was both improper and prejudicial. *Thorgerson*, 172 Wn.2d at 442. To show prejudice requires that the defendant show a substantial likelihood that the misconduct affected the jury verdict. *Id.*; *State v. Ish*, 170 Wn.2d 189, 195, 241 P.3d 389 (2010); *State v. Dhaliwal*, 150 Wn.2d 559, 578, 79 P.3d 432 (2003). Because Mr. Glasmann failed to object at trial, the errors he complains of are waived unless he establishes that the misconduct was so flagrant and ill intentioned that an instruction would not have cured the prejudice. *Thorgerson*, 172 Wn.2d at 443; *State v. Russell*, 125 Wn.2d 24, 86, 882 P.2d 747 (1994).

[8,9] ¶17 Our courts have repeatedly and unequivocally denounced the type of conduct that occurred in this case. First, we have held that it is error to submit evidence to the jury that has not been admitted at trial. *State v. Pete*, 152 Wn.2d 546, 553-55, 98 P.2d 803 (2004). The “long-standing rule” is that “consideration of any material by a jury not properly admitted as evidence vitiates a verdict when there is a reasonable ground to believe that the defendant may have been prejudiced.” *Id.* at 555 n.4 (quoting *State v. Rinkes*, 70 Wn.2d 854, 862, 425 P.2d 658 (1967) (emphasis omitted)); *see also, e.g., State v. Boggs*, 33 Wn.2d 921, 207 P.2d 743 (1949), *overruled on other grounds by State v. Parr*, 93 Wn.2d 95, 606 P.2d 263 (1980). p753

¶18 In *Rinkes*, 70 Wn.2d at 855, for example, a newspaper editorial and cartoon highly critical of what it claimed was lenient court decisions and liberal probation policies was inadvertently sent to the jury room. The court stated that the material in the newspaper should not have gone to the jury and observed that the article was “clearly intended to

influence the readers of it [(the newspaper)] to be concerned about the purported leniency” of area judges and “may well have evoked a jury members feelings or convictions of the necessity for being stricter and less careful about observing legal principles and procedure in dealing with defendants accused of crime.” *Id.* at 862-63. The court said the material was “very likely indeed” to be prejudicial and assumed that “the requisite balance of impartiality was upset.” *Id.* at 863.

¶ 19 Here, the prosecutor intentionally presented the jury with copies of Glasmann’s booking photograph altered by the addition of phrases calculated to influence the jury’s assessment of Glasmann’s guilt and veracity. In the photograph, Glasmann is unkempt and bloody, a condition likely to have resulted in even greater impact because of captions that challenged the jury to question the truthfulness of his testimony. While the State argues that it merely combined the booking photograph, admitted as exhibit 89, with the court’s instructions and argument of the law and facts, the prosecutor’s conduct went well beyond this. Indeed, here the prosecutor’s modification of photographs by adding captions was the equivalent of unadmitted evidence. There certainly was no photograph in evidence that asked “DO YOU BELIEVE HIM?” *See* State’s Resp. to PRP, App. G at 5. There was nothing that said, “WHY SHOULD YOU BELIEVE ANYTHING HE SAYS ABOUT THE ASSAULT?” *See id.* And there were no sequence of photographs in evidence with “GUILTY” on the face or “GUILTY, GUILTY, GUILTY.” *See id.* Yet this “evidence” was made a part of the trial by the prosecutor during closing argument.

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¶ 20 Although this is not a case where unadmitted evidence was sent to the jury room,

as in *Pete* and *Rinkes*, these cases nevertheless establish that a prosecutor must be held to know that it is improper to present evidence that has been deliberately altered in order to influence the jury's deliberations. As in *Rinkes*, the multiple altered photographs here may well have affected the jurors' feelings about the need to strictly observe legal principles and the care it must take in determining Glasmann's guilt.

¶ 21 It is also well established that a prosecutor cannot use his or her position of power and prestige to sway the jury and may not express an individual opinion of the defendant's guilt, independent of the evidence actually in the case. The commentary on *American Bar Association Standards for Criminal Justice* std. 3-5.8 emphasizes:

The prosecutor's argument is likely to have significant persuasive force with the jury. Accordingly, the scope of argument must be consistent with the evidence and marked by the fairness that should characterize all of the prosecutor's conduct. Prosecutorial conduct in argument is a matter of special concern because of the possibility that the jury will give special weight to the prosecutor's arguments, not only because of the prestige associated with the prosecutor's office but also because of the fact-finding facilities presumably available to the office.

¶ 22 Likewise, many cases warn of the need for a prosecutor to avoid expressing a personal opinion of guilt. *E.g.*, *State v. McKenzie*, 157 Wn.2d 44, 53, 134 P.3d 221 (2006) (finding it improper for a prosecuting attorney to express his individual opinion that the accused is guilty, independent of the testimony in the case (citing *State v. Armstrong*, 37 Wash. 51, 79 P. 490 (1905))); *Dhaliwal*, 150 Wn.2d at 577 (permitting latitude to attorneys to argue the facts in evidence and reasonable inferences therefrom, but prohibiting statements of personal belief of a defendant's guilt or innocence); *State v.*

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Stith, 71 Wn. App. 14, 21-22, 856 P.2d 415 (1993) (deeming a prosecutor's comment in closing argument that the appellant "was just coming back and he was dealing [drugs] again" impermissible opinion "testimony"); *State v. Traweek*, 43 Wn. App. 99, 107, 715 P.2d 1148 (1986) (concluding it was error for a prosecutor to tell the jury he "knew" the defendant committed the crime). By expressing his personal opinion of Glasmann's guilt through both his slide show and his closing arguments, the prosecutor engaged in misconduct.

¶23 The case law and professional standards described above were available to the prosecutor and clearly warned against the conduct here. We hold that the prosecutor's misconduct, which permeated the state's closing argument, was flagrant and ill intentioned.

¶24 Moreover, the misconduct here was so pervasive that it could not have been cured by an instruction. "[T]he cumulative effect of repetitive prejudicial prosecutorial misconduct may be so flagrant that no instruction or series of instructions can erase their combined prejudicial effect." *State v. Walker*, 164 Wn. App. 724, 737, 265 P.3d 191 (2011) (citing *Case*, 49 Wn.2d at 73).

¶25 Highly prejudicial images may sway a jury in ways that words cannot. *See State v. Gregory*, 158 Wn.2d 759, 866-67, 147 P.3d 1201 (2006). Such imagery, then, may be very difficult to overcome with an instruction. *Id.* Prejudicial imagery may become all the more problematic when displayed in the closing arguments of a trial, when the jury members may be particularly aware of, and susceptible to, the arguments being presented.

Given the multiple ways in which the prosecutor attempted to improperly sway the jury and the powerful visual medium he employed, no instruction could erase the cumulative effect of the misconduct in this case. The prosecutor essentially produced a media event with the deliberate goal of influencing the jury to return guilty verdicts on the counts against Glasmann.

¶ 26 We also believe there is a substantial likelihood that the misconduct affected the jury verdict. As noted earlier, the State charged Glasmann with first degree assault, attempted first degree robbery, first degree kidnapping, and obstruction. The mental state required for the charged offenses, specifically intent, was critically important. Glasmann presented evidence that he lacked both the opportunity and capacity to form the intent necessary to commit the charged crimes. There was evidence that he consumed alcohol, methamphetamine, and ecstasy the night of the offenses and evidence that the events involving Glasmann, Benson, and law enforcement unfolded rapidly. Glasmann defended on the basis that the facts only supported a guilty verdict as to third or fourth degree assault, attempted robbery in the second degree, unlawful imprisonment, and obstruction. The jury convicted Glasmann of second degree assault, attempted second degree robbery, first degree kidnapping, and obstruction.

¶ 27 A prosecutor could never shout in closing argument that “Glasmann is guilty, guilty, guilty!” and it would be highly prejudicial to do so. Doing this visually through use of slides showing Glasmann’s battered face and superimposing red capital letters (red, the color of blood and the color used to denote losses) is even more prejudicial. *See*

Gregory, 158 Wn.2d at 866-67. “[V]isual arguments manipulate audiences by harnessing rapid unconscious or emotional reasoning processes and by exploiting the fact that we do not generally question the rapid conclusions we reach based on visually presented information.” Lucille A. Jewel, *Through a Glass Darkly: Using Brain and Visual*

Rhetoric to Gain a Professional Perspective on Visual Advocacy, 19 S. Cal. Interdisc. L.J. 237, 289 (2010). Further, P 709

[w]ith visual information, people believe what they see and will not step back and critically examine the conclusions they reach, unless they are explicitly motivated to do so. Thus, the alacrity by which we process and make decisions based on visual information conflicts with a bedrock principle of our legal system—that reasoned deliberation is necessary for a fair justice system.

Id. at 293 (footnote omitted) (citing William J. Bowers, Benjamin D. Steiner & Marla Sandys, *Death Sentencing in Black and White: An Empirical Analysis of the Role of Jurors’ Race and Jury Racial Composition*, 3 U. Pa. J. Const. L. 171, 261 (2001) (citing Jeffrey Abramson, *We, The Jury: The Jury System and the Ideal of Democracy* (1994) (generally discussing the basic democratic principle for jury trials is that deliberations should be a rational and reasoned process))).

¶ 28 During the critical closing moments of trial, one of the last things the jury saw before it began its deliberations was the representative of the State of Washington impermissibly flashing the word “GUILTY” across an image of Glasmann’s face three times, predisposing the jury to return a harsh verdict. Indeed, the entire 50-plus slide presentation used during closing argument was full of imagery that likely inflamed the

jury.⁴ The prosecutor's improper visual "shouts" of GUILTY urged the jury to find Glasmann guilty as charged, and without them, the jury might have returned verdicts on the offenses Glasmann agreed he had committed.⁵ Because Glasmann defended by asserting he was guilty only of lesser offenses, and nuanced distinctions often separate degrees of a crime, there is an especially serious danger that the nature and scope of the misconduct here may have affected the jury.

¶29 When viewed as a whole, the prosecutor's repeated assertions of the defendant's guilt, improperly modified exhibits, and statement that jurors could acquit Glasmann only if they believed him represent the type of pronounced and persistent misconduct that cumulatively causes prejudice demanding that a defendant be granted a new trial. *See Berger*, 295 U.S. at 89; *Thomas v. Hubbard*, 273 F.3d 1164, 1179-80 (9th Cir. 2001), *overruled on other grounds by Payton v. Woodford*, 299 F.3d 815 (2002); *United States v. Frederick*, 78 F.3d 1370, 1381 (9th Cir. 1996); *see also Matlock v. Rose*, 731 F.2d

⁴ "Sometimes, we are unable to rationally consider how images affect our emotions or our decision-making process. As we are processing an image in our pre-conscious sensory system, that image can activate an emotional reaction in our mind without us even knowing about it." Jewel, *supra*, 19 S. Cal. Interdisc. L.J. at 263 (citing Ann Marie Seward Barry, Visual Intelligence: Perception, Image, and Manipulation in Visual Communication 18 (1997); Joseph LeDoux, *The Emotional Brain* 165 (1996)). "[T]he danger in using emotionally vivid imagery is not that it is subliminally persuasive, but that it tends to generate emotionally driven reactions that can unconsciously affect a decision-maker's thought process." *Id.* at 254. "[T]here is evidence that gruesome photographs cause unconscious emotional reactions—reactions that may not be curable with a limiting instruction." *Id.* at 268-69 (citing Kevin S. Douglas, David R. Lyon & James R.P. Ogloff, *The Impact of Graphic Photographic Evidence on Mock Jurors' Decisions in a Murder Trial: Probative or Prejudicial?*, 21 Law & Hum. Behav. 485, 499 (1997) ("[I]f jurors cannot even recognize the extent to which [graphic] evidence affects them, it will be impossible for them to reduce or control the impact of the evidence when instructed to do so by a judge.")).

⁵ It is also possible that the jury might have acquitted Glasmann on a charge.

1236, 1244 (6th Cir. 1984).

¶ 30 The dissent, however, believes that reversal is not required with regard to three of the four crimes found by the jury and only the conviction for second degree assault should be reversed. The dissent says that Glasmann conceded the crimes of obstructing a law enforcement officer and second degree attempted robbery, and the jury accordingly convicted him of these crimes. With respect to the first degree kidnapping charge, the dissent maintains the evidence is overwhelming that this conviction must be upheld.

¶ 31 We have on a number of occasions established that reviewing claims of prosecutorial misconduct is not a matter of determining whether there is sufficient evidence to convict the defendant. In *State v. Charlton*, 90 Wn.2d 657, 665, 585 P.2d 142 (1978), we concluded the discussion of prosecutorial misconduct in that case, which required reversal, by noting that “[i]n spite of our frequent warnings that prejudicial prosecutorial tactics will not be permitted, we find that some prosecutors continue to use improper, sometimes prejudicial means in an effort to obtain convictions. *In most of these instances, competent evidence fully sustains a conviction.*” (Emphasis added.) The issue is whether the comments deliberately appealed to the jury’s passion and prejudice and encouraged the jury to base the verdict on the improper argument “rather than properly admitted evidence.” *State v. Ferman*, 122 Wn.2d 440, 468-69, 858 P.2d 1092 (1993) (quoting and discussing *Belgarde*, 110 Wn.2d at 507-08). The focus must be on the misconduct and its impact, not on the evidence that was properly admitted. p 711

¶ 32 Thus, deciding whether reversal is required is not a matter of whether there is

sufficient evidence to justify upholding the verdicts. Rather, the question is whether there is a substantial likelihood that the instances of misconduct affected the jury's verdict.

Dhaliwal, 150 Wn.2d at 578. We do not decide whether reversal is required by deciding whether, in our view, the evidence is sufficient. *See Monday*, 171 Wn.2d at 678-80 (racist arguments required reversal; no weighing of evidence by the court); *Belgarde*, 110 Wn.2d at 507-10 (inflammatory remarks associating defendant with an organization the prosecutor described as "deadly group of madmen; misconduct required reversal; no weighing of evidence by the court); *Charlton*, 90 Wn.2d at 664 (prosecutor commented on the defendant's spouse's failure to testify, despite the marital privilege, with the inference being that the defendant was concealing or withholding testimony; reversal required—jury might have been inclined to believe the defendant's version in the absence of the improper argument).

¶ 33 The dissent says it agrees that whether the error requires reversal is not a matter of whether there is sufficient evidence to uphold the verdicts. Dissent at 4-5 n.3. But weighing the evidence is in fact what the dissent does. We do not believe this analysis is appropriate and it is contrary to our precedent, as explained. If the misconduct cannot be linked to a specific count, and the misconduct is so egregious that we must conclude reversal is required on one charge, then how can we conclude the misconduct did not sway the jury on another charged crime without engaging in an inappropriate sufficiency of the evidence analysis, like the dissent has done?

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¶ 34 In this case, the use of highly inflammatory images unrelated to any specific count

was misconduct that contaminated the entire proceedings. The prosecutor's unacceptable argument announced to the jury that the defendant was intrinsically GUILTY GUILTY GUILTY. The misconduct distracted the jury from its duty to consider the evidence unaffected by the overlaid message that emphatically and repeatedly conveyed the prosecutor's belief to the jury that Glasmann is "absolutely guilty!", and which constituted an appeal to passion and prejudice on all counts.

¶ 35 There is a substantial likelihood here that the jury returned guilty verdicts for the offenses the jurors found because they were influenced by the prosecutor's improper closing argument and the altered "evidence" presented during argument. We cannot say that the jury would not have returned verdicts for lesser offenses, or even acquittal, i.e., we cannot even presume the jury would have accepted defense counsel's concessions even as to the obstruction charged. The impact of such powerful but unquantifiable material on the jury is exceedingly difficult to assess but substantially likely to have affected the *entirety* of the jury deliberations and its verdicts. Even the dissent agrees that the misconduct mandates reversal of the assault conviction. The requisite balance of impartiality was upset. Mr. Glasmann's right to a fair trial must be granted in full. In this way, we give substance to our message that "prejudicial prosecutorial tactics will not be permitted," and our warnings that prosecutors must avoid improper, prejudicial means of obtaining convictions will not be empty words. *Charlton*, 90 Wn.2d at 665. P 713

¶ 36 Next, we turn briefly to Mr. Glasmann's claim that the prosecutor improperly misstated the burden of proof. Because we reverse Glasmann's conviction based on the

misconduct addressed above, we need not reach this issue, but do so in the interest of fully discussing the prosecutor's conduct.

[15] ¶ 37 Shifting the burden of proof to the defendant is improper argument, and ignoring this prohibition amounts to flagrant and ill intentioned misconduct. *E.g., State v. Fleming*, 83 Wn. App. 209, 213-14, 921 P.2d 1076 (1996); *Casteneda-Perez*, 61 Wn. App. at 362-63. Due process requires the prosecution to prove, beyond a reasonable doubt, every element necessary to constitute the crime with which the defendant is charged. *In re Winship*, 397 U.S. 358, 361, 90 S. Ct. 1068, 25 L. Ed. 2d 368 (1970). Misstating the basis on which a jury can acquit insidiously shifts the requirement that the State prove the defendant's guilt beyond a reasonable doubt. *Fleming*, 83 Wn. App. at 213.⁶

¶ 38 Similarly, in this case the prosecutor informed the jury that in order to reach a verdict, it must decide whether the defendant told the truth when he testified. Thus, the prosecutor strongly insinuated that the jury could only acquit (or find him guilty of lesser charges) if it believed Glasmann, when the proper standard is whether the evidence established that he was guilty of the State's charges beyond a reasonable doubt. This misconduct was not as egregious as the conduct in *Fleming*, however, and in and of itself

⁶ During the State's closing argument in *Fleming*, the prosecutor stated, "for you to find the defendants . . . not guilty of the crime of rape in the second degree, . . . you would have to find either that [the victim] has lied about what occurred . . . or that she was confused." *Fleming*, 83 Wn. App. at 213 (emphasis omitted) (quoting court proceedings). This was error because it misstated the basis upon which the jury could acquit and shifted the burden to the defendant to disprove the State's case. *Id.* at 214. A prosecutor who argues that to acquit the defendant the jury must find that the State's witnesses are lying or mistaken commits misconduct. *Id.*

would probably not justify reversal. However, it was clearly misconduct for the prosecutor to inform the jury that acquittal was only appropriate if the jury believed Glasmann, and shows the prosecutor's failure to prosecute this case as an impartial officer of the court.

CONCLUSION

¶ 39 The prosecutor's presentation of a slide show including alterations of Glasmann's booking photograph by addition of highly inflammatory and prejudicial captions constituted flagrant and ill intentioned misconduct that requires reversal of his convictions and a new trial, notwithstanding his failure to object at trial. Considering the entire record and circumstances of this case, there is a substantial likelihood that this misconduct affected the jury verdict. The principal disputed matter at trial was whether Glasmann was guilty of lesser offenses rather than those charged, and this largely turned on whether the requisite mental element was established for each offense. More fundamentally, the jury was required to conclude that the evidence established Glasmann's guilt of each offense beyond a reasonable doubt.

¶ 40 It is substantially likely that the jury's verdict were affected by the prosecutor's improper declarations that the defendant was "GUILTY, GUILTY, GUILTY!", together with the prosecutor's challenges to Glasmann's veracity improperly expressed as superimposed messages over the defendant's bloodied face in a jail booking photograph.

¶ 41 We reverse the defendant's convictions and remand for a new trial.

AUTHOR:

Chief Justice Barbara A. Madsen

WE CONCUR:

Justice Charles W. Johnson

Justice Debra L. Stephens

Gerry L. Alexander, Justice Pro Tem.

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11 my colleague, Mr. Jackson, made his opening remarks to
12 you, he made reference to the travels of the defendant,
13 Mr. Sublett, and his one-time paramour, April Frazier,
14 in terms of following the money. Of course he was
15 referring to the tracking that was done by bank security
16 agents and the police who managed to track and then
17 capture the defendant, Mr. Sublett, in Las Vegas. I
18 mention this, ladies and gentlemen, because this
19 following the money is a two-way street because this
20 money provides evidence of motive, motive of what
21 occurred at the residence of Jerry Totten on 320 I
22 Street on January 29th, 2007.

23 It was at this location, ladies and gentlemen --
24 this location, the residence of Jerry Totten, was for
25 this defendant, Mr. Sublett, and this defendant, Mr.

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1 Olsen, the pot of gold if you will at the end of the
2 rainbow. This was the residence of Jerry Totten, a then
3 69-year-old disabled man who it became known to the
4 defendants was worth some substantial amount of money,
5 enough money for them to want to go in and help
6 themselves, and of course these defendants did. They
7 burst into his home, forced him into this recliner;
8 gagged him with paper shoved down his throat, bound his
9 wrists, throttled him with the straps, and he died by
10 manual strangulation, the method of killing, as you know

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12 now, that takes two to three minutes of consistent
13 pressure, in this instance manual strangulation, that
14 is, the use of the hands for two to three minutes to
15 affect death. And so based upon this evidence, ladies
16 and gentlemen, we have these two defendants before you
17 who --

18 ~~MR. WOODROW: Your Honor, I'm gonna object at~~
19 ~~this time. The State is using unadmitted exhibits in~~
20 ~~this case. I'd ask that that exhibit be taken down.~~

21 ~~Thank you.~~

22 ~~THE COURT: Thank you, counsel.~~

23 ~~I will ask you to ladies and gentlemen of the~~
24 ~~jury, we are going to take --~~

25 ~~MR. BRUNEAU: Well, how about if I just move~~
~~along, Your Honor?~~

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1 THE COURT: Thank you.

2 MR. BRUNEAU: Ladies and gentlemen, you know
3 that these defendants are both charged with murder in
4 the first degree, and there are two methods of
5 committing murder in the first degree. One is what we
6 call killing with premeditation, and I will refer to
7 that in argument as premeditated murder. The other
8 method of committing the crime of murder in the first
9 degree is killing in the course of a burglary in the
10 first degree or a robbery in the first or second degree,
11 and that form of murder, ladies and gentlemen, is what

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23 because, after all, I am referring to the totality of
24 the evidence in the case. ~~why do people lie? why? why?~~
25 ~~lie? lie? lie? Because you're covering up your~~

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1 ~~participation in a crime. The only thing that's gonna~~
2 ~~save his ass, as he said, is his mouth, he hopes.~~

3 And along with all of the evidence in the case,
4 ladies and gentlemen, we have the remarks that the
5 defendant, Mr. Sublett, had to say about Elsie, to Elsie
6 Pray, Elsie Pray, who acknowledged that -- I believe she
7 said that she still regards Mr. Sublett as a friend.
8 She spoke to him when he was on the run and urged him to
9 turn himself in, and he tells his friend "I'm really
10 thinking hard about coming back and turning myself in."
11 "You need to turn yourself in." "Yeah, I know I do.
12 I'm really messed up." Well, that's what he told Elsie
13 Pray, but here we have Mr. Sublett in Boise. Really
14 messed up? ~~what is that saying, a picture says a~~
15 ~~thousand words?~~ Here we have got a man, here we have
16 got a killer, who is literally and figuratively in the
17 driver's seat, ladies and gentlemen. He might say to
18 his friend "I'm messed up," but he's got Jerry Totten's
19 credit cards. He's tapped into his line of credit, and
20 as far as we know, he's been tapping into about \$50,000.

21 Ladies and gentlemen, the Court gives you what we
22 call the reasonable doubt instruction. I'd like to in

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23 my completing remarks touch upon this. Another one of
24 those great things about our system is that a defendant
25 is presumed innocent, and when a person pleads not

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1 guilty, that presumption of innocence continues
2 throughout the entire case until you, ladies and
3 gentlemen, are satisfied beyond a reasonable doubt that
4 a defendant is guilty.

5 I put this up on the screen because this is not
6 something that I just talk about. The presumption of
7 innocence and the burden of proof, which we welcome, is
8 not just something we talk about, but it is a living,
9 breathing reality. It is a factor that we deal with
10 every day. I put this up on the board, ladies and
11 gentlemen, because a reasonable doubt is something for
12 which a reason exists and may arise from the evidence or
13 lack of evidence. It is such a doubt as would exist in
14 the mind of a reasonable person after fully and
15 carefully considering the evidence. Keep in mind,
16 ladies and gentlemen, that we're talking about
17 reasonable. We're talking about reasonable people, such
18 as yourselves, considering evidence and scrutinizing
19 that evidence with a view towards reasonableness. And
20 if, after such consideration, you have an abiding belief
21 in the truth of the charge, then you're satisfied beyond
22 a reasonable doubt.

23 A reasonable doubt, ladies and gentlemen, is not
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24 any doubt. It is not proof to a moral certainty. It is
25 not proof beyond any doubt whatsoever. It is proof that

CLOSING ARGUMENTS

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1 excludes reasonable doubts. The judge concludes by
2 telling you that the law says if you have an abiding
3 belief, then you are satisfied. If you have an abiding
4 belief in the truth of the charge, then you're satisfied
5 beyond a reasonable doubt. Now, I don't -- I don't
6 know. People react in various ways. An abiding belief
7 might be something you know in your head. It might be
8 something that you feel in your heart. It might be
9 something that you know in your gut; I know he's guilty.
10 If you have that abiding belief, then you're satisfied.

11 When you consider, ladies and gentlemen, the
12 totality of the evidence of motive, of the planning, of
13 the execution, of the burglary, the robbery, of the
14 death of Jerry Totten --

15 ~~MR. WOODROW: Your Honor, I'm going to object~~
16 ~~again to unadmitted evidence in the State's closing.~~

17 ~~MR. BRUNEAU: When you consider the --~~

18 ~~MR. WOODROW: Objection. I'd ask Your Honor~~
19 ~~to make a ruling on that.~~

20 ~~THE COURT: I'm going to ask that we move on,~~
21 ~~that you take that picture off. Thank you, counsel.~~

22 ~~MR. BRUNEAU: They are guilty as indicated.~~
23 ~~These defendants, ladies and gentlemen, are guilty as~~

24 17-JUNE SUBLETT - IX
~~charged and guilty as proven.~~

25 ~~Thank you for your attention.~~

CLOSING ARGUMENTS

1003

1 THE COURT: Thank you.

2 Ladies and gentlemen of the jury, we will now take
3 our morning recess. First a word of caution. You have
4 heard only one closing argument. Please don't talk
5 about the case.

6 If you will go with the bailiff, I ask the
7 attorneys to remain in session.

8 (Jury out.)

9 THE COURT: I ask for 15 minutes. Is there
10 anything else? Thank you.

11 THE CLERK: Please rise.

12 (Recess.)

13 THE COURT: Mr. Bruneau? I am on the record,
14 Cheri. I am going to ask you not to use the photos that
15 were not admitted. Thank you.

16 Are we ready to proceed?

17 MR. LANE: Ready, Your Honor.

18 THE COURT: Bring them in.

19 (Jury in.)

20 THE COURT: Thank you. Ladies and gentlemen
21 of the jury, please be seated, and be seated in the
22 courtroom.

23 Ladies and gentlemen of the jury, please give your
24 attention to Mr. Lane for his closing argument.

attachment #4

RICHARD WOODROW

ATTORNEY AT LAW

December 19, 2012

Christopher Olsen
DOC #831898 RB212.WSP
1313 North 13th Avenue
Walla Walla, WA 99362

Re: PRP

I know you probably know your appeal was denied. When you file your PRP make sure to include the following:

1. Violation of right to fair trial when Bruneau used an exhibit that had your mug shot displayed on a projector screen. Your picture was highlighted with a red banner that said guilty. I objected and the judge ordered the prosecutor to remove the exhibit from the jury's view. The prosecutor took his time about it. I believe under recent case law¹ this issue will get you a new trial.

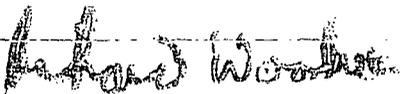
I will submit an affidavit on your behalf. I will also get Sublett's attorney to submit one. Make sure you ask for the exhibit. I don't think it was filed. Make a motion in your PRP for it. Make a public records request for it.

2. Make sure you get a copy of the voir dire transcript. According to the court record a transcript was produced. I don't think we had any closures of the court but you never know.

Good luck.

Sincerely,

Richard Woodrow
RAW:



¹In re Glasman No 84475-5. This case is very similar to your case. We had asked for a lesser included charges just like in Glasman. I think this use of an un-admitted exhibit made the jury come back on murder one.

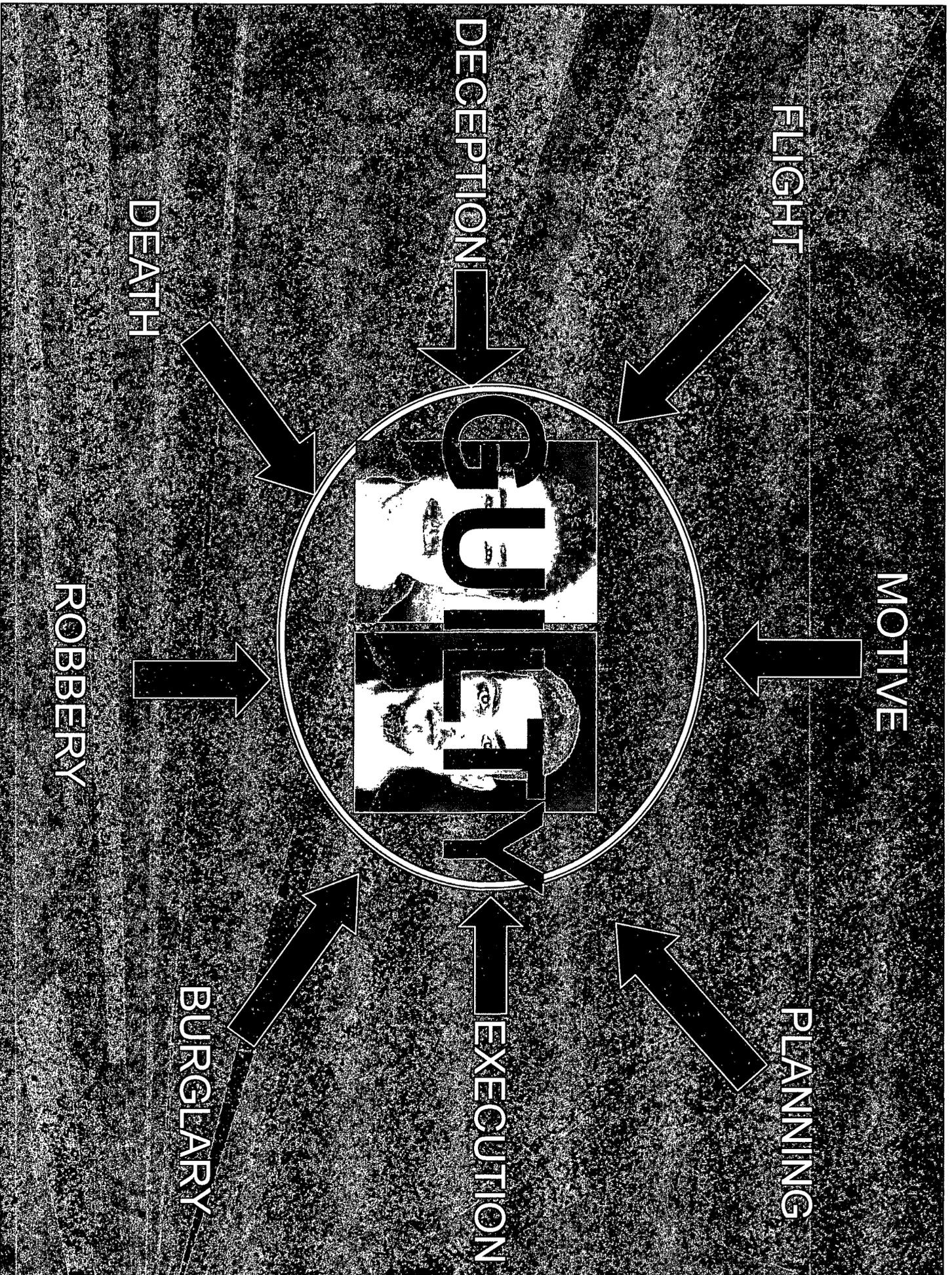
STATE OF WASHINGTON

VS.

**MICHAEL SUBLETT AND
CHRISTOPHER OLSEN**

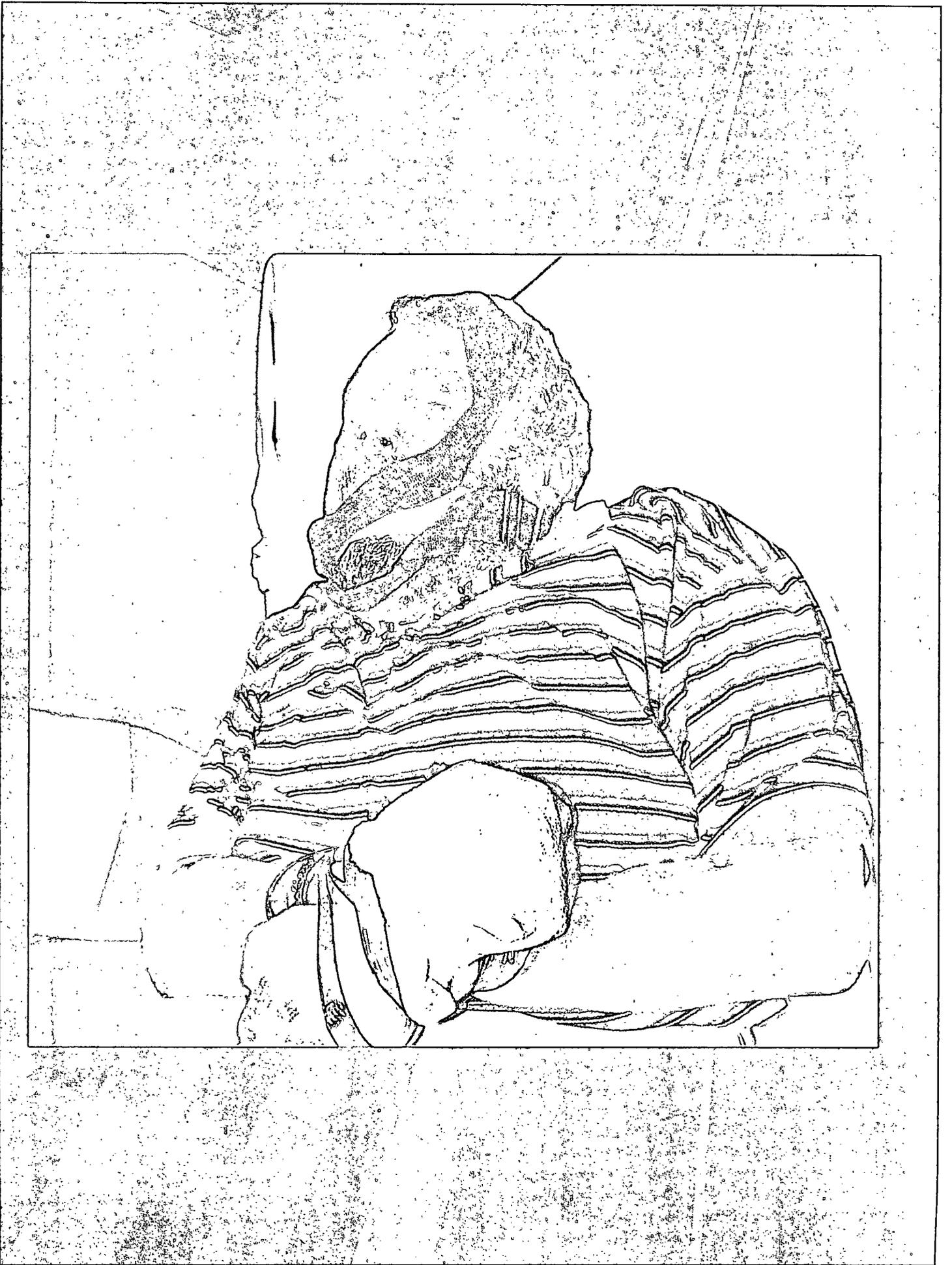


**MURDER IN THE FIRST DEGREE
PREMEDITATED MURDER
OR
FELONY MURDER**





ATT. # 5 B



AT #5C

16-JUNE SUBLETT - VIII

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JUNE 16, 2008

* * * * *

(Jury out.)

THE COURT: Please be seated. Good morning.

MR. LANE: Good morning.

MR. JACKSON: Good morning, Your Honor.

THE COURT: It's freezing. Let's try to regulate it a little bit.

Okay. This morning the bailiff came to me, and a juror came to her. I'm just going to have her explain it to -- where is Mr. Woodrow? Thank you. Good morning.

MR. WOODROW: Good morning, Your Honor.

THE COURT: It's not 9 o'clock yet. It's a couple minutes to. She will explain it to the two of you. Go ahead.

THE BAILIFF: One of my jurors mentioned to me that he has gone to church services down in the jail before, and last week during some of the recordings he heard Mr. Olsen mention the fact that he had been to one of those church services, so I asked my juror if he had seen Mr. Olsen there when he went to the church service, and he said no, and he hasn't said anything to any of the other jurors, but he wanted me to know that he understood Mr. Olsen to say that he had been in one of

1 the church services down in the jail.

2 That's all there is to it.

3 THE COURT: Do you want to bring the juror
4 out? I don't understand the significance of this or why
5 the juror said it, but I'll let any of the four of you,
6 if you want to talk to the juror, bring him out and
7 explore it.

8 MR. WOODROW: I don't, Your Honor.

9 THE COURT: Pardon me, Mr. Woodrow?

10 MR. WOODROW: I don't.

11 THE COURT: You don't want him?

12 MR. WOODROW: (Shakes head negatively.)

13 MR. BRUNEAU: I'm good.

14 MR. LANE: That's fine, Your Honor.

15 THE COURT: Bring the jury in.

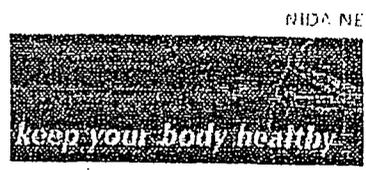
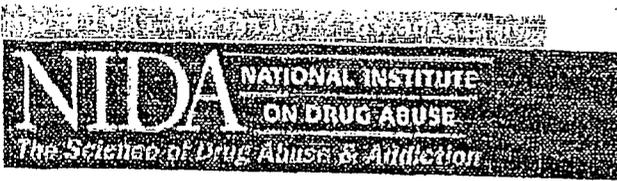
16 (Jury in.)

17 THE COURT: Good morning. Please be seated,
18 and be seated in the courtroom. Thank you.

19 Ladies and gentlemen of the jury, we are trying to
20 regulate the temperature in this room. I will tell you
21 that you weren't with us Friday, but it was about
22 40 degrees. Now we're trying to bring it up and down,
23 so I'm going to try to regulate it the best I can. I
24 ask your cooperation with that.

25 With that, Mr. Woodrow, I'll turn to you for your

Attachment #7



DRUGS OF ABUSE & RELATED TOPICS

NIDA Home > Drugs of Abuse/Related Topics > Methamphetamine > InfoFacts > Methamphetamine

NIDA InfoFacts: Methamphetamine

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En Español



Methamphetamine is a central nervous system stimulant drug that is similar in structure to amphetamine. Due to its high potential for abuse, methamphetamine is classified as a Schedule II drug and is available only through a prescription that cannot be refilled. Although methamphetamine can be prescribed by a doctor, its medical uses are limited, and the doses that are prescribed are much lower than those typically abused. Most of the methamphetamine abused in this country comes from foreign or domestic superlabs, although it can also be made in small, illegal laboratories, where its production endangers the people in the labs, neighbors, and the environment.

Recommended Reading

- [NIDA Research Report: Methamphetamine Abuse and Addiction](#)
- [Community Drug Abuse Bulletin: Methamphetamine](#)

How Is Methamphetamine Abused?

Methamphetamine is a white, odorless, bitter-tasting crystalline powder that easily dissolves in water or alcohol and is taken orally, intranasally (snorting the powder), by needle injection, or by smoking.

Other NIDA Web Sites

- [NIDA for Teens: Mind Over Matter - Methamphetamine](#)
- [NIDA for Teens: Sara's Quest - Methamphetamine](#)

How Does Methamphetamine Affect the Brain?

Methamphetamine increases the release and blocks the reuptake of the brain chemical (or neurotransmitter) dopamine, leading to high levels of the chemical in the brain, a common mechanism of action for most drugs of abuse. Dopamine is involved in reward, motivation, the experience of pleasure, and motor function. Methamphetamine's ability to rapidly release dopamine in reward regions of the brain produces the intense euphoria, or "rush," that many users feel after snorting, smoking, or injecting the drug.

Chronic methamphetamine abuse significantly changes how the brain functions. Noninvasive human brain imaging studies have shown alterations in the activity of the dopamine system that are associated with reduced motor skills and impaired verbal learning.¹ Recent studies in chronic methamphetamine abusers have also revealed severe structural and functional changes in areas of the brain associated with emotion and memory,^{2,3} which may account for many of the emotional and cognitive problems observed in chronic methamphetamine abusers.

Repeated methamphetamine abuse can also lead to addiction—a chronic, relapsing disease, characterized by compulsive drug seeking and use, which is accompanied by chemical and molecular changes in the brain. Some of these changes persist long after

methamphetamine abuse is stopped. Reversal of some of the changes, however, may be observed after sustained periods of abstinence (e.g., more than 1 year).⁴

What Other Adverse Effects Does Methamphetamine Have on Health?

Taking even small amounts of methamphetamine can result in many of the same physical effects of other stimulants, such as cocaine or amphetamines, including increased wakefulness, increased physical activity, decreased appetite, increased respiration, rapid heart rate, irregular heartbeat, increased blood pressure, and hyperthermia.

Long-term methamphetamine abuse has many negative health consequences, including extreme weight loss, severe dental problems ("meth mouth"), anxiety, confusion, insomnia, mood disturbances, and violent behavior. Chronic methamphetamine abusers can also display a number of psychotic features, including paranoia, visual and auditory hallucinations, and delusions (for example, the sensation of insects crawling under the skin).

Transmission of HIV and hepatitis B and C can be consequences of methamphetamine abuse. The intoxicating effects of methamphetamine, regardless of how it is taken, can also alter judgment and inhibition and lead people to engage in unsafe behaviors, including risky sexual behavior. Among abusers who inject the drug, HIV and other infectious diseases can be spread through contaminated needles, syringes, and other injection equipment that is used by more than one person. Methamphetamine abuse may also worsen the progression of HIV and its consequences. Studies of methamphetamine abusers who are HIV-positive indicate that HIV causes greater neuronal injury and cognitive impairment for individuals in this group compared with HIV-positive people who do not use the drug.^{5,6}

What Treatment Options Exist?

Currently, the most effective treatments for methamphetamine addiction are comprehensive cognitive-behavioral interventions. For example, the Matrix Model—a behavioral treatment approach that combines behavioral therapy, family education, individual counseling, 12-step support, drug testing, and encouragement for non-drug-related activities—has been shown to be effective in reducing methamphetamine abuse.⁷ Contingency management interventions, which provide tangible incentives in exchange for engaging in treatment and maintaining abstinence, have also been shown to be effective.⁸ There are no medications at this time approved to treat methamphetamine addiction; however, this is an active area of research for NIDA.

How Widespread Is Methamphetamine Abuse?

Monitoring the Future Survey*

According to the 2008 Monitoring the Future survey—a national survey of 8th-, 10th-, and 12th- graders, methamphetamine abuse among students has shown a general decline in recent years; however, it remains a concern. Survey results show that 2.3 percent of 8th-graders, 2.4 percent of 10th-graders, and 2.8 percent of 12th-graders have used methamphetamine in their lifetime. In addition, 0.7 percent of 8th-graders, 0.7 percent of 10th-graders, and 0.6 percent of 12th-graders were current (past-month) methamphetamine abusers. Past-year use of methamphetamine remained steady across all grades surveyed from 2007 to 2008.

Methamphetamine Prevalence of Abuse Monitoring the Future Survey, 2008

	8th Grade	10th Grade	12th Grade
Lifetime**	2.3%	2.4%	2.8%
Past Year	1.2	1.5	1.2
Past Month	0.7	0.7	0.6

National Survey on Drug Use and Health***

The number of individuals aged 12 years or older reporting past-year methamphetamine use declined from 1.9 million in 2006 to 1.3 million in 2007. An estimated 529,000 Americans were current (past-month) users of methamphetamine (0.2 percent of the population). Of the 157,000 people who used methamphetamine for the first time in 2007, the mean age at first use was 19.1 years, which is down from the mean age of 22.2 in 2006.

Other Information Resources

For more information on the effects of methamphetamine abuse and addiction, visit www.drugabuse.gov/drugpages/methamphetamine.html.

To find publicly funded treatment facilities by State, visit www.findtreatment.samhsa.gov.

For street terms searchable by drug name, street term, cost and quantities, drug trade, and drug use, visit www.whitehousedrugpolicy.gov/streetterms/default.asp.

* These data are from the 2008 Monitoring the Future survey, funded by the National Institute on Drug Abuse, National Institutes of Health, Department of Health and Human Services, and conducted by the University of Michigan's Institute for Social Research. The study has tracked 12th-graders' illicit drug abuse and related attitudes since 1975; in 1991, 8th- and 10th-graders were added to the study. The latest data are online at www.drugabuse.gov.

** "Lifetime" refers to use at least once during a respondent's lifetime. "Past year" refers to use at least once during the year preceding an individual's response to the survey. "Past month" refers to use at least once during the 30 days preceding an individual's response to the survey.

*** NSDUH (formerly known as the National Household Survey on Drug Abuse) is an annual survey of Americans age 12 and older conducted by the Substance Abuse and Mental Health Services Administration. Copies of the latest survey are available at www.samhsa.gov and from NIDA at 877-643-2644.

1 Volkow ND, Chang L, Wang GJ, et al. Association of dopamine transporter reduction with psychomotor impairment in methamphetamine abusers. *Am J Psychiatry* 158:377-382, 2001.

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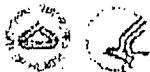
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8 Roll JM, Petry NM, Stitzer ML, et al. Contingency management for the treatment of methamphetamine use disorders. *Am J Psychiatry* 163:1993-1999, 2006.

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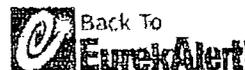


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 DOE/Brookhaven National Laboratory

Methamphetamine enters brain quickly and lingers

First study of methamphetamine uptake, distribution, and clearance in humans helps explain why the drug is so addictive and damaging to brain

UPTON, NY — Using positron emission tomography (PET) to track tracer doses of methamphetamine in humans' brains, scientists at the U.S. Department of Energy's (DOE) Brookhaven National Laboratory find that the addictive and long-lasting effects of this increasingly prevalent drug can be explained in part by its pharmacokinetics — the rate at which it enters and clears the brain, and its distribution. This study in 19 healthy, non-drug-abusing volunteers includes a comparison with cocaine and also looked for differences by race. It will appear in the November 1, 2008, issue of *NeuroImage*.

"Methamphetamine is one of the most addictive and neurotoxic drugs of abuse," said Brookhaven chemist Joanna Fowler, lead author on the study. "It produces large increases in dopamine, a brain chemical associated with feelings of pleasure and reward — both by increasing dopamine's release from nerve cells and by blocking its reuptake."

Studies by Fowler and others have shown that drugs that produce greater elevations in brain dopamine tend to be more addictive. But other factors, including the speed with which a drug enters and clears the brain and its distribution within the brain, can also be important in determining its addictive and toxic potential.

In undertaking this first study of methamphetamine pharmacokinetics, the researchers also wanted to know if there were differences between Caucasians and African Americans. "Reports that the rate of methamphetamine abuse among African Americans is lower than for Caucasians led us to question whether biological or pharmacokinetic differences might explain this difference," Fowler said.

The scientists measured brain uptake, distribution, and clearance of methamphetamine by injecting 19 normal healthy men (9 Caucasian, 10 African American) with a radioactively tagged form of the drug in "trace" doses too small to have any psychoactive effects. They used PET scanning cameras to monitor the concentration and distribution of the tagged methamphetamine in the subjects' brains. On the same day, the same subjects were injected with trace doses of cocaine and scanned for comparison. The scientists also used PET to measure the number of dopamine reuptake proteins, known as dopamine transporters, available in each research subject's brain.

Like cocaine, methamphetamine entered the brain quickly, a finding consistent with both drugs' highly reinforcing effects. Methamphetamine, however, lingered in the brain significantly longer than cocaine, which cleared quickly. In fact, some brain regions, particularly white matter, still showed signs of tracer methamphetamine at the end of the 90-minute scanning session, by which time all cocaine had been cleared. The distribution of methamphetamine in the brain was remarkably different from that of cocaine. Whereas cocaine was concentrated only in the 'reward' center and cleared rapidly, methamphetamine was concentrated all over the brain, where it remained throughout the study.

"This slow clearance of methamphetamine from such widespread brain regions may help explain why the drug has such long-lasting behavioral and neurotoxic effects," Fowler said. Methamphetamine is known to produce lasting damage not only to dopamine cells but also to other brain regions, including white matter, that are not part of the dopamine network.

Surprisingly, the researchers found significant differences in cocaine pharmacokinetics between African Americans and Caucasians, with the African Americans exhibiting higher uptake of cocaine, a later rise to peak levels, and slower clearance. In contrast, the scientists found no differences in methamphetamine pharmacokinetics between these groups.

"This suggests that variables other than pharmacokinetics and bioavailability account for the lower prevalence of methamphetamine abuse in African Americans," Fowler said. "The differences observed for cocaine pharmacokinetics are surprising considering there are no differences in cocaine abuse prevalence between these two ethnic groups." These differences may merit further study, and also suggest the need to match subjects by ethnic group in future studies to avoid interference from this potentially confounding variable.

Another interesting finding was that across all research subjects, the level of dopamine transporters was directly related to the level of methamphetamine taken up by the brain. This finding suggests that transporter proteins somehow play a role in regulating the brain's uptake of this drug.

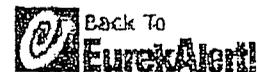
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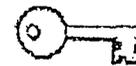
This research was funded by the National Institute on Drug Abuse, the National Institute on Alcohol Abuse and Alcoholism Intramural Program, and by the Office of Biological and Environmental Research within DOE's Office of Science. Brain-imaging studies such as PET are a direct outgrowth of DOE's long-standing investment in basic research in chemistry, physics, and nuclear medicine. The ongoing neuroimaging research at Brookhaven is a prime example of how DOE's national laboratories bring together the expertise of chemists, physicists, and medical scientists to address questions of profound significance for society.

One of ten national laboratories overseen and primarily funded by the Office of Science of the U.S. Department of Energy (DOE), Brookhaven National Laboratory conducts research in the physical, biomedical, and environmental sciences, as well as in energy technologies and national security. Brookhaven Lab also builds and operates major scientific facilities available to university, industry and government researchers. Brookhaven is operated and managed for DOE's Office of Science by Brookhaven Science Associates, a limited-liability company founded by the Research Foundation of State University of New York on behalf of Stony Brook University, the largest academic user of Laboratory facilities, and Battelle, a nonprofit, applied science and technology organization.

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Neurotoxicity of Substituted Amphetamines: Molecular and Cellular Mechanisms

JEAN LUD CADET*, IRINA N. KRASNOVA, SUBRAMANIAM JAYANTHI and JOHNALYN LYLES

Molecular Neuropsychiatry Branch, DHHS/NIH/NIDA, Intramural Research Program, 5500 Nathan Shock Drive, Baltimore, Maryland 21224, USA: jcadet@intra.nida.nih.gov

(Submitted 06 November 2006, In final form 11 January 2007)

The amphetamines, including amphetamine (AMPH), methamphetamine (METH) and 3,4-methylenedioxymethamphetamine (MDMA), are among abused drugs in the US and throughout the world. Their abuse is associated with severe neurologic and psychiatric adverse events including the development of psychotic states. These neuropsychiatric complications might, in part, be related to drug-induced neurotoxic effects, which include damage to dopaminergic and serotonergic terminals, neuronal apoptosis, as well as activated astroglial and microglial cells in the brain. The purpose of the present review is to summarize the toxic effects of AMPH, METH and MDMA. The paper also presents some of the factors that are thought to underlie this toxicity. These include oxidative stress, hyperthermia, excitotoxicity and various apoptotic pathways. Better understanding of the cellular and molecular mechanisms involved in their toxicity should help to generate modern therapeutic approaches to prevent or attenuate the long-term consequences of amphetamine use disorders in humans.

Keywords: Substituted amphetamines; Methamphetamine; Methylenedioxyamphetamine; MDMA; Serotonergic neurons; Dopaminergic neurons; Hyperthermia; Neurotoxicity

AMPHETAMINE

Amphetamine (AMPH) is a psychostimulant that belongs to widely used illegal drugs in the world. AMPH is a popular drug of abuse in Australia (Baru *et al.*, 2004), Belgium (Raes and Verstraete, 2005), Brazil (Silva and Yonamine, 2004), Switzerland (Augsburger *et al.*, 2005) and UK (Wylie *et al.*, 2005). AMPH is a common drug of abuse in Sweden and other northern European countries (Jones, 2005; Gustavsen *et al.*, 2006). In the USA, non-medical use of medications prescribed for ADHD treatment, including those that contain AMPH, is high among high school and college students (McCabe *et al.*, 2004; 2005). It has been reported that the abuse of these drugs is second only to marijuana (Brown *et al.*, 2001).

AMPH abuse is associated with very serious harms. These include increased psychological morbidity, dependence and health problems. For example, acute AMPH side-effects include tachycardia, hypertension, hyperthermia, increased muscle tension, liver and renal failure, nausea, blurred vision, ataxia, anxiety, psychosis and seizures (Kalant and Kalant, 1975; Janowsky and Risch, 1979; Alldredge *et al.*, 1989; Murray, 1998). Other severe and fatal AMPH intoxications have also been reported (Ginsberg *et al.*, 1970; Kalant and Kalant, 1975; Salanova and Taubner, 1984; De

Letter *et al.*, 2006; Steentoft *et al.*, 2006). Chronic AMPH abuse is associated with impairments in attention and memory, problems with learning, as well as compromised decision making (McKetin and Mattick, 1997; Rogers *et al.*, 1999; Ornstein *et al.*, 2000). Some of these neuropsychiatric complications are thought to be related to AMPH-induced neurotoxic effects which consist of decreases in tyrosine hydroxylase (TH) activity (Ellison *et al.*, 1978), long-term dopamine (DA) depletion (Wagner *et al.*, 1980a), loss of dopamine transporters (DAT) (Scheffel *et al.*, 1996; Krasnova *et al.*, 2001), as well as decreases in vesicular monoamine transporter proteins (Krasnova *et al.*, 2001). In addition to its effects on monoaminergic terminals, AMPH can cause cell death of primary cortical cells, TH-positive mesencephalic neurons, and of PC12 cells *in vitro* (Stumm *et al.*, 1999; Lotharius and O'Malley, 2001; Oliveira *et al.*, 2002) as well as degeneration of cell bodies in the cortex of AMPH-treated rodents (Jakab and Bowyer, 2002). The drug can also cause the activation of caspase-3 and appearance of TUNEL-positive cells in the striatum (Krasnova *et al.*, 2005). Calbindin- and DA- and cAMP-regulated phosphoprotein, Mr 32 kD (DARPP-32)-positive medium spiny projection neurons, but not choline acetyltransferase (ChAT)-, parvalbumin- or somatostatin-positive interneurons undergo AMPH-induced apoptosis (Krasnova *et al.*, 2005). Although the mechanisms for AMPH-mediated toxicity are not completely clear, they appear to include uptake into DA terminals, DA release, oxidative stress and the activation of p53-dependent and mitochondria-mediated cell death pathways. Herein, the data supporting these mechanisms in AMPH toxicity are reviewed.

AMPH Toxicity Involves ROS Formation and ROS-mediated Transcriptional Changes.

AMPH-induced redistribution of DA from synaptic vesicles to the cytosol followed by its release to the extracellular space by reverse transport through DAT causes increased DA levels in the synaptic cleft (Sulzer *et al.*, 1995). DA metabolism is accompanied by the production of hydroxyl (Huang *et al.*, 1997) and superoxide (Krasnova *et al.*, 2001) radicals that participate in the toxic effects of the drug via free radical-mediated destruction of monoaminergic terminals (Huang *et al.*, 1997; Cadet

and Brannock, 1998; Wan *et al.*, 2000; Krasnova *et al.*, 2001). This occurs because reactive oxygen species (ROS) induced by AMPH administration can exceed the compensating abilities of antioxidant enzymes such as superoxide dismutases (SODs), catalase and glutathione peroxidase (Cadet and Brannock, 1998). The possible involvement of superoxide radicals in AMPH toxicity is also supported by the findings that transgenic mice that overexpress CuZnSOD are partially protected against the toxic effects of the drug on dopaminergic systems (Krasnova *et al.*, 2001).

Because ROS play a role in cellular signaling processes, including the regulation of transcriptional factors (Poli *et al.*, 2004), induction or suppression of transcription factors with subsequent activation or repression of genes that encode proteins involved in various neuronal functions might be critical steps in AMPH-induced cascades of toxic events. These ideas are supported by the demonstration that administration of AMPH causes activation of AP-1 transcription factors (Persico *et al.*, 1995; Ferguson *et al.*, 2003; Milanovic *et al.*, 2006). The possibility that superoxide radicals might be involved in AMPH-induced transcriptional responses has been tested using microarray analyses (Krasnova *et al.*, 2002). This allowed the identification of 37 genes that show superoxide-mediated responses. Among these are genes that belong to classes of transcription factors, growth factors, heat shock proteins (HSPs), and xenobiotic metabolism. In response to neuronal damage, organisms initiate and elaborate events that trigger neuroprotective pathways that serve to minimize or prevent damage; they also function to increase the chance of functional recovery (Wieloch and Nikolich, 2006). These pathways include the increased synthesis and release of growth factors and cytokines such as the neuronal protein, activin A (Werner and Alzheimer, 2006), which is activated by AMPH in a superoxide-responsive manner (Krasnova *et al.*, 2002). The participation of activin A in protective mechanisms is illustrated by the reports that it reduces MPP1-induced cellular damage to DA neurons *in vitro* (Krieglstein *et al.*, 1995) and rescues striatal neurons from excitotoxic lesioning with quinolic acid (Hughes *et al.*, 1999). Another AMPH-responsive superoxide-mediated gene is macrophage colony-stimulating factor which is involved in the pro-

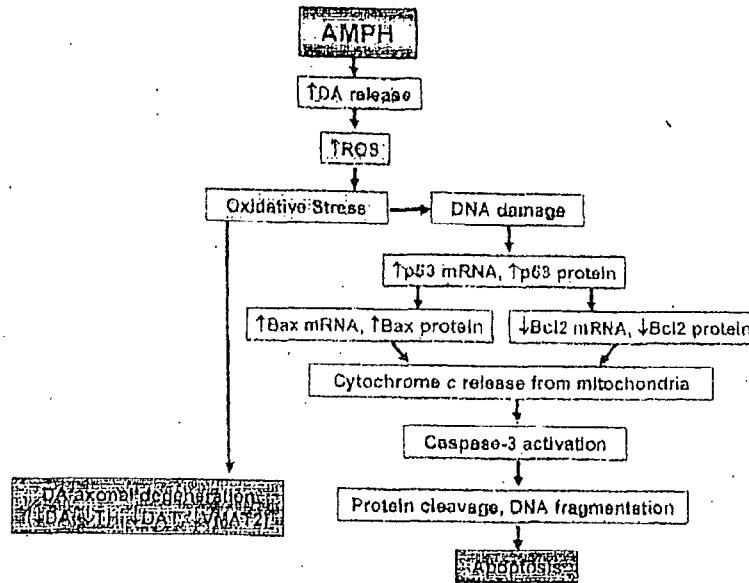


FIGURE 1 Overview of the molecular mechanisms involved in AMPH neurotoxicity. Oxidative stress, p53 and mitochondrial pathway play an essential role in the AMPH-induced neuronal apoptosis and DA terminal degeneration.

proliferation and migration of activated microglia into injured sites of the brain (Inai and Kohsaka, 2002). Additional genes whose transcript levels are induced by AMPH code for HSPs such as HSP110 and HSC70. HSPs have been shown to protect cells against oxidative damage (Papp *et al.*, 2003; Macario and Conway de Macario, 2005).

AMPH Toxicity Involves Activation of the p53-mediated Cell Death Pathway.

ROS-induced stresses are known to be associated with DNA damage and p53 accumulation *in vitro* (Lombard *et al.*, 2005). p53 activation has been shown to participate in events that cause neuronal apoptosis (Culmsee and Mattson, 2005). This is thought to be related to the influence exerted by p53 on the expression of the Bcl-2 family of proteins which include the pro-apoptotic protein, Bax and the anti-apoptotic protein, Bcl-2 (Moll *et al.*, 2005; Chowdhury *et al.*, 2006). Specifically, p53 causes upregulation of Bax and downregulation of Bcl-2 (Moll *et al.*, 2005; Chowdhury *et al.*, 2006). As reported above, AMPH has been shown to cause neuronal cell death in various brain regions (Jakab and Bowyer, 2002; Krasnova *et al.*, 2005). The AMPH-induced neuronal apoptosis has been

recently shown to involve the activation of the p53 pathway with secondary increases in Bax levels and decreases in Bcl-2 levels in the mouse striatum (Krasnova *et al.*, 2005). The role of Bax activation in AMPH-related apoptosis was further supported by experiments showing that Bax-deficient mice were partially resistant to drug-induced cell death (Krasnova *et al.*, 2005). Figure 1 shows a schematic representation of the mechanisms that may underlie AMPH-related apoptosis and DA terminal degeneration.

AMPH Treatment and Temperature Regulation

Temperature regulation appears to be also an important factor in the toxic responses to AMPH. In rodents, the psychostimulant was shown to produce biphasic effects with low doses (≤ 2.5 mg/kg) inducing hypothermia and higher doses (≥ 5 mg/kg) causing hyperthermia at ambient temperature above 20°C (Seale *et al.*, 1985; Krasnova *et al.*, 2001; Baker and Meert, 2003). This effect was found to be dose-dependent, with the degree of hyperthermia correlating to AMPH and DA levels in rat striatal microdialysate (Clausing and Bowyer, 1999). Several studies have also hinted to connections between hyperthermic and neurotoxic

actions of AMPH (Clausing *et al.*, 1995; Miller and O'Callaghan, 1996). Conditions that reduce or prevent AMPH-induced increases in core body temperature are, at least, partially neuroprotective (Clausing *et al.*, 1995; Miller and O'Callaghan, 1996). In contrast, hyperthermia could exacerbate AMPH toxicity because the formation of free radicals in the brain is elevated by temperature increase (Kil *et al.*, 1996) and because hyperthermia also potentiates the cytotoxic effects of ROS (Lin *et al.*, 1991). These ideas are also supported by the report that hyperthermia significantly increases DA quinone formation (LaVoie and Hastings, 1999) since quinones derived from DA have the ability to inhibit proteasome (Zafar *et al.*, 2006) which is involved in detoxification mechanisms.

Although AMPH-induced increases in temperature are thought to be involved in the toxicity of the drug, the manner by which the temperature is induced remains to be determined. DA release (Clausing and Bowyer, 1999) and D₁ receptor stimulation (Sanchez, 1989; Zarrindast and Tabatabai, 1992; Verma and Kulkarni, 1993) have been implicated. The observations that animals with severe hyperthermia released more DA in the striatal extracellular space provide partial support for this contention (Clausing and Bowyer, 1999). Moreover, the idea is also supported by the reports that hyperthermia is induced by administration of the D₁ agonist SKF 38393 in mice (Sanchez, 1989; Zarrindast and Tabatabai, 1992; Verma and Kulkarni, 1993), the effect that could be blocked by D₁ antagonist SCH 23390 (Sanchez, 1989; Zarrindast and Tabatabai, 1992). It has to be pointed out that since AMPH can cause release of other monoamines (Seiden *et al.*, 1993), their possible involvement also needs to be considered. For example, lesions of ventral norepinephrine bundle innervating the hypothalamus and limbic system cause attenuation of AMPH-induced hyperthermia in rats (Kostowski *et al.*, 1982).

In addition to DA release, AMPH-induced production of free radicals might also contribute to thermal instability (Krasnova *et al.*, 2001). Mice that overexpress the antioxidant enzyme, CuZnSOD, in the brain show no hyperthermic responses after AMPH treatment and are protected against long-term neurotoxic drug effects (Krasnova *et al.*, 2001). Thus, the possibility of complex interactions between thermoregulation and free radical load in

the long-term neurotoxicity induced by this illicit drug needs to be considered.

It is also of interest to note that various strains of mice show different hyperthermic responses to AMPH. Specifically, psychostimulants caused substantial hyperthermia in CD-1 (Krasnova *et al.*, 2001), Swiss-Webster (Craig and Kupferberg, 1972), DBA/2 (Seale *et al.*, 1985), and BALB/c mice (Jori and Rutzynski, 1978), while C57BL/6 (Seale *et al.*, 1985; Krasnova *et al.*, 2001) and C₃H animals (Jori and Rutzynski, 1978) had low to moderate transient temperature increase. These differences in temperature responses may help to further dissect the role of hyperthermia in AMPH toxicity. For example, it seems there is no simple algorithm to predict toxicity based on temperature responses because CD-1 mice are more resistant to AMPH neurotoxicity than C57BL/6 mice in spite of showing greater and longer-lasting hyperthermia than C57BL/6 mice (Krasnova *et al.*, 2001).

Finally, the issues of temperature regulation have major clinical implications because AMPH can cause fatal hyperpyrexia in humans (Ginsberg *et al.*, 1970; Kalant and Kalant, 1975; Callaway and Clark, 1994; De Letter *et al.*, 2006). Thus, understanding of the root causes of AMPH-induced hyperthermia might help to develop therapeutic approaches that can prevent or attenuate the disastrous effects of this drug when taken in high doses.

METHAMPHETAMINE

Metamphetamine (METH, Speed, crank) is abused worldwide due to its powerful stimulant properties that cause the user to feel "high" and to have increased energy (McCann *et al.*, 1998b; Sekine *et al.*, 2001; Farrell *et al.*, 2002). METH is easily available because it can be synthesized cheaply and distributed to various communities throughout the world. Presently, there is widespread abuse in the United States where it has migrated from the West Coast to other states (Puder *et al.*, 1988; Derlet *et al.*, 1989; Cho and Melega, 2002). METH can be abused via multiple routes which include oral, intravenous and smoking administration. In addition to its euphorogenic effects, METH can also cause anxiety, increased agitation, delirium, psychotic states, cognitive and psychomotor impairments, seizures, and death (Wilson *et al.*, 1996;

Lan *et al.*, 1998; Buffenstein *et al.*, 1999; Yui *et al.*, 1999; Simon *et al.*, 2000; Volkow *et al.*, 2001a; London *et al.*, 2004; Dore and Sweeting, 2006). Cerebral vasculitis, cerebrovascular accidents due to hemorrhage or vasospasm, and cerebral edema have also been reported in METH abusers (Chynn, 1968; Salanova and Taubner, 1984). The drug can also cause neurodegenerative changes in the brains of human addicts. These pathological changes include loss of striatal DAT observed in positron emission tomographic (PET) studies (Volkow *et al.*, 2001b; Sekine *et al.*, 2003) and in post-mortem investigations, loss of serotonin transporters (5-HTT) (Sekine *et al.*, 2006), decrease in the levels of DA, serotonin (5-HT) and their metabolites (Wilson *et al.*, 1996). A number of studies have documented that METH can cause long-term damage to presynaptic dopaminergic and serotonergic terminals in rodents (Ricaurte *et al.*, 1980; Wagner *et al.*, 1980b). More recently, it has also been shown that the drug can cause death of cell bodies both *in vitro* (Cadet *et al.*, 1997; Stumm *et al.*, 1999) and *in vivo* (Eisch and Marshall, 1998; Deng *et al.*, 1999; Deng and Cadet, 2000; O'Dell and Marshall, 2000). In what follows, we discuss some of the mechanisms that have been implicated in METH-induced neurodegenerative effects.

Role of Oxidative Stress in METH-induced Toxicity

The biochemical actions of the drug depend on its entry into monoaminergic terminals (Berger *et al.*, 1992; Iversen, 2006), followed by entry into monoaminergic vesicle consequent to its interaction with vesicular monoamine transporters (Sulzer *et al.*, 1995). This is followed by displacement of monoamines into the cytoplasm of the terminals and METH-induced monoamine release into respective synaptic clefts (Baldwin *et al.*, 1993; Marshall *et al.*, 1993; Cubells *et al.*, 1994; Sulzer *et al.*, 1995; Schwartz *et al.*, 2006). METH neurotoxicity appears to depend on both, DA released within terminals and on DA released in synaptic clefts (Cadet and Brannock, 1998). These suggestions are supported by reports that DAT knockout mice are resistant to METH-induced degeneration of DA axons (Fumagalli *et al.*, 1998) and by observations that psychostimulant toxicity depends on quinone forma-

tion consequent to increased DA levels within nerve terminals (LaVoie and Hastings, 1999). METH-related quinone formation is thought to be associated with the generation of superoxide radicals and hydrogen peroxide during quinone redox cycling (Stokes *et al.*, 1999; Miyazaki *et al.*, 2006). A role for oxidative mechanisms in the neurotoxic effects of the drug is supported by observations that administration of *N*-acetyl-L-cysteine, ascorbic acid or vitamin E was able to protect against METH-induced destruction of monoaminergic terminals (Wagner *et al.*, 1985; De Vito and Wagner, 1989; Fukami *et al.*, 2004). In addition, selenium and melatonin can also provide protection against METH toxicity (Ali *et al.*, 1999; Imam and Ali, 2000). The participation of superoxide radicals in the neurotoxic effects of METH on DA nerve terminals was tested by injecting METH to transgenic mice that overexpress the human CuZnSOD gene (Cadet *et al.*, 1994a; Hirata *et al.*, 1996; Jayanthi *et al.*, 1998). These mice have much higher CuZnSOD activity than wild-type animals from similar backgrounds (Jayanthi *et al.*, 1998; Jayanthi *et al.*, 1999) and were indeed protected against the toxic effects of the drug. In contrast, inhibition of SOD by diethyldithiocarbamate potentiates the nefarious effects of METH (De Vito and Wagner, 1989). Furthermore, bromocriptine, which scavenges hydroxyl radicals, was also able to attenuate METH-induced DA depletion in mice (Kondo *et al.*, 1994). When taken together, these observations support the notion that DA release caused by METH is accompanied by redox cycling of dopaquinone and consequent formation of oxygen-based radicals such as superoxide radicals. Reports that METH can induce changes in the levels of glutathione (Harold *et al.*, 2000) and of antioxidant enzymes (Jayanthi *et al.*, 1998), increase lipid peroxidation (Jayanthi *et al.*, 1998; Gluck *et al.*, 2001), and induce the formation of protein carbonyls (Gluck *et al.*, 2001) provide further support for the thesis that oxygen-based radicals are involved in METH-induced toxicity (Cadet and Brannock, 1998).

METH Toxicity and Excitotoxicity

METH-induced neurotoxicity also appears to occur via excitotoxic damage secondary to glutamate

release and activation of glutamate receptors. Glutamate toxicity is dependent, in part, on the production of nitric oxide (NO) (Dawson and Dawson, 1998; Chung *et al.*, 2005). The idea of the involvement of glutamate in METH toxicity is supported by observations that METH can cause glutamate release in the brain (Nash *et al.*, 1988; Baldwin *et al.*, 1993; Marshall *et al.*, 1993; Abekawa *et al.*, 1994; Mark *et al.*, 2004). In addition, some glutamate antagonists have been shown to attenuate METH-induced dopaminergic toxicity (Sonsalla *et al.*, 1989; Battaglia *et al.*, 2002) (see later discussion on temperature). Glutamate-mediated NO formation appears to also be involved in METH toxicity because knockout mice that are deficient in either neuronal (nNOS) or inducible (iNOS) nitric oxide synthase (NOS) are resistant to drug-induced toxic damage to monoaminergic terminals (Itzhak *et al.*, 1998). These data have solidified the argument for a role of the glutamate/NO pathway in METH neurotoxicity (Itzhak *et al.*, 1998; Imam *et al.*, 2001; Itzhak and Ali, 2006). Finally, various nNOS inhibitors, which do not affect hyperthermia, can also protect against destruction of monoaminergic axons caused by METH administration (Itzhak *et al.*, 2000; Sanchez *et al.*, 2003). In addition to their roles in the damage of monoaminergic terminals, oxygen-based radicals and NO appear to be involved in METH-related cell death because CuZnSOD transgenic mice show partial protection against drug-induced apoptosis (Deng and Cadet, 2000). Moreover, death of rat fetal mesencephalic cells caused by METH treatment was abrogated by the use of NOS inhibitors (Sheng *et al.*, 1996).

Role of Thermal Instability in METH Toxicity

There is substantial evidence that hyperthermia participates in METH-induced toxicity on monoaminergic systems. Manipulations that result in higher temperatures cause increases in METH toxicity, whereas those that decrease temperatures have been shown to provide some degree of protection (Böwyer *et al.*, 1994; Miller and O'Callaghan, 1994; Albers and Sonsalla, 1995; Farfel and Seiden, 1995). The potentiative effects of hyperthermia might occur through increased formation of DA-dependent reactive oxygen species. In contrast, there are pharmacological agents that block METH toxicity without influencing the thermal responses

in animals. For example, inhibition of nNOS blocks METH toxicity without altering the hyperthermic response (Itzhak *et al.*, 2000; Sanchez *et al.*, 2003). DA uptake blockers also protect in a fashion that appear to be independent of any effects on temperature (Callahan *et al.*, 2001).

In addition to its effects on monoaminergic terminals, METH can also cause cell death. Potential protective effects of various genetic and pharmacological manipulations have been tested in that model. For example, knockout mice that are partially deficient of c-Jun show protection against METH-induced neuronal apoptosis, an effect that is independent of hyperthermia (Deng *et al.*, 2002b). Intracerebral injection of neuropeptide Y (NPY) has recently been shown to cause attenuation of the apoptotic effects of the drug in mice (Thiriet *et al.*, 2005). Because NPY is involved in thermoregulation (Richard, 1995; Levine *et al.*, 2004) and because METH-related increases in body temperature are thought to participate in METH toxicity (Cadet *et al.*, 2003, for review), the possibility that NPY might have prevented drug-induced hyperthermia was tested (Thiriet *et al.*, 2005). NPY was found to attenuate body temperature increases after the second of the four METH injections but not during the later phases of hyperthermia (Thiriet *et al.*, 2005). These observations suggest that NPY-induced protection is, in part, dependent on its effects on body temperature. It appears that METH-related changes in body temperature participate, but are not essential in the manifestations of drug toxicity.

Microglial Reactions and METH Toxicity

Microglial cells are the major immunocompetent cells in the brain. They express chemokines, cytokines and their receptors. Under normal conditions, these cells provide extensive and continuous surveillance of their cellular environment (Raivich, 2005). Microglial cells are activated by various types of pathological states including infectious processes (Rock *et al.*, 2004) and neural injuries (Ladeby *et al.*, 2005). This activation includes dramatic changes in appearance, migration to the site of the damage, and phagocytosis of dying and dead cells. Microglia can also produce small signaling molecules, called cytokines, to trigger astrocytes to respond to the injury site. Recently,

reactive microgliosis has been implicated in a number of neurological disorders including Alzheimer's (Xiang *et al.*, 2006) and Huntington's (Sapp *et al.*, 2001) diseases.

Evidence accumulating from several laboratories has recently implicated reactive microglial cells as culprits in the manifestation of METH toxicity. Asanuma *et al.* (2003) reported that the non-steroidal anti-inflammatory drug, ketoprofen, caused protection against METH-induced dopaminergic toxicity and suppressed drug-mediated microgliosis. Thomas and colleagues (2004) subsequently reported that METH caused dose-dependent microglial activation which coincided with DA terminal degeneration. LaVoie *et al.* (2004) have also provided evidence that microgliosis precedes METH-induced pathological states in striatal dopaminergic terminals. More importantly, manipulations such as the use of MK-801 and dextromethorphan which protect against METH toxicity also inhibit microglial activation (Thomas and Kuhn, 2005). In contrast, minocycline has been reported to block microglial activation without providing protection against METH-induced damage (Sriram *et al.*, 2006). Microglial cells might potentiate drug-related damage by releasing toxic substances such superoxide radicals and NO which have already been implicated in METH neurotoxicity (see discussion above). When taken together, these observations suggest that identifying the specific role that microglial cells play in DA terminal degeneration might help to develop specific therapeutic approaches for patients who have been exposed to METH.

Involvement of AP-1 Related Transcription Factors in METH-induced Neurotoxicity

The accumulated evidence had suggested that some effects of oxygen-based radicals might be mediated by activation of AP-1 transcription factors (Dalton *et al.*, 1999). Tests for the possibility that METH toxicity might also be associated with variations in the expression of these proteins have revealed changes in the expression of a number of AP-1 related genes within 2 hours after drug administration (Cadet *et al.*, 2001). These include up-regulation of c-jun, c-fos, jun B, as well as jun D (Cadet *et al.*, 2001). These changes are probably related to METH-induced generation of free radi-

icals. ROS such as hydroxyl and superoxide radicals can induce the expression of many genes via their regulation of AP-1 transcription factors (Dalton *et al.*, 1999). The role for c-fos in METH-induced neuropathological changes has been confirmed by using c-fos +/- mice which show increased degeneration of DA terminals and increased cell death after psychostimulant treatment (Deng *et al.*, 1999). These observations suggest a protective role for c-fos against METH damage. Some of the factors that might be involved in causing this partial protection include integrins that belong to cell adhesion receptors and are also involved in the regulation of signal transduction (Gilcrease, 2006). This idea is supported by the evidence of decreased basal levels of integrin expression in c-fos +/- mice and the further reduction of these receptors in response to toxic doses of METH (Betts *et al.*, 2002). This conclusion is further supported by the observations that integrins can promote cell survival after injury and apoptotic insults via signaling through the PI3K-Akt pathway which leads to BAD phosphorylation, therefore reducing BAD ability to block the anti-apoptotic effects of Bcl-2 (Martin and Vuori, 2004; Gilcrease, 2006). In contrast, inhibition of integrins increases apoptotic cell death (Martin and Vuori, 2004; Gilcrease, 2006).

Because c-jun knockout mice show partial protection against the adverse effects of METH (Deng *et al.*, 2002b), it is likely that c-jun is involved in the pro-death effects of the drug. Moreover, because the c-jun knockout mice and their wild-type counterparts show similar degree of METH-induced dopaminergic toxicity, c-jun appears to only be involved in the mediation of neuronal apoptosis in cells postsynaptic to DA terminals.

Role of DNA Damage in METH-induced Toxicity

As mentioned above, METH has been shown to cause neuronal apoptosis in several brain regions (Deng *et al.*, 2001). Because apoptosis is associated with DNA damage, it was thought possible that administration of the drug might trigger responses meant to repair the METH-induced DNA damage. Microarray analyses have indeed revealed that METH administration caused changes in the expression of several genes that participate in DNA repair processes (Cadet *et al.*, 2002). These changes

are probably related to METH-induced prooxidant states because oxidative stress can cause single and double DNA strand breaks (Li and Trush, 1993). These breaks can be repaired via base excision repair (BER), nucleotide excision repairs (NER), mismatch repair (MMR), and DNA damage reversal (Petit and Sancar, 1999; Hsieh, 2001; Nilsen and Krokan, 2001). Thus, the observations that METH treatment can cause upregulation of APEX, PolB, and LIG1 suggest that these changes might be compensatory increases aimed at counteracting METH-mediated ROS-induced DNA damage through the BER pathway. If the psychostimulant can cause similar DNA damage in humans, these observations might offer a partial explanation for the developmental abnormalities observed in babies born of METH abusing mothers (Smith *et al.*, 2006).

Involvement of Mitochondrial Death Pathway in METH-induced Apoptosis

Another interesting group of proteins that are differentially regulated by METH includes Bcl-2 family (Stumm *et al.*, 1999; Cadet *et al.*, 2001; Jayanthi *et al.*, 2001). Specifically, METH caused upregulation of pro-apoptotic proteins, BAX and BID, and downregulation of the anti-death proteins, Bcl-2 and Bcl-X_L. The changes in pro-death proteins are consistent with observations that METH administration is associated with release of mitochondrial contents into the cytosol (Deng *et al.*, 2002a; Jayanthi *et al.*, 2004). These include cytochrome *c* and apoptosis inducing factor (AIF). When taken together with the recent *in vitro* demonstration that METH can cause release of cytochrome *c* from mitochondria, activation of caspases 9 and 3, as well as activation of DFF40 and its transit to the nucleus (Deng *et al.*, 2002a), the *in vivo* data implicate a formal role of mitochondria in METH-induced neuronal degeneration. Other factors released from mitochondria such as Smac/DIABLO, endonuclease G, and AIF also participate in dismantling cells during apoptosis (Ravagnan *et al.*, 2002). These proteins, including AIF and Smac/DIABLO, have now been shown to be involved in METH-induced apoptosis (Jayanthi *et al.*, 2004). Their release is followed by activation of caspase 3 and the breakdown of several structural cellular proteins (Jayanthi *et al.*, 2004). Thus, these observations implicate the mitochondrial death pathway as a major player in METH-

related cell death in the rodent brain (Cadet *et al.*, 2005). This suggestion is supported by the fact that overexpression of Bcl-2 can protect against drug-induced apoptosis (Cadet *et al.*, 1997).

Involvement of the Endoplasmic Reticulum (ER)-dependent Death Pathway in METH-induced Apoptosis

In addition to its effects on mitochondria, METH-induced oxidative stress appears to also cause dysfunctions of other organelles such as the endoplasmic reticulum (ER) (McCullough *et al.*, 2001). The ER helps to maintain cellular homeostasis by regulating calcium signaling (Ferri and Kroemer, 2001). Dysregulation of intracellular calcium homeostasis can cause ER stress and ER-mediated apoptosis (Paschen, 2001). ER stress and calcium dysregulation appear to participate in METH-induced cell death because apoptotic doses of the drug can cause activation of calpain, a calcium-responsive cytosolic cysteine protease (Murachi *et al.*, 1980), which is involved in ER-dependent cell death (Nakagawa and Yuan, 2000). A role for the ER in METH toxicity is supported by the fact that apoptotic doses of METH (Jayanthi *et al.*, 2004) also influence the expression of proteins, such as caspase-12, GRP78/BiP (glucose-regulated protein/immunoglobulin heavy chain binding protein) and CHOP/GADD153 (C/EBP homologous protein/growth arrest and DNA damage 153) that participate in ER-induced apoptosis (Zinszner *et al.*, 1998). The observed ER stress in METH-induced neurotoxicity might be secondary, in part, to direct effects of the psychostimulant (Asanuma *et al.*, 2000), to METH-mediated oxidative stress (Cadet *et al.*, 1994a; Cadet and Brammick, 1998; Jayanthi *et al.*, 1998), and to shifts in BAX/Bcl-2 ratios induced by the drug (Jayanthi *et al.*, 2001).

Involvement of the Fas/Fas Ligand Death Pathway in METH-induced Apoptosis

In addition to the mitochondrial death pathway, cell death can occur consequent to activation of Fas receptors by Fas ligand (FasL) (Barnhart *et al.*, 2003; Choi and Benveniste, 2004). FasL (TNFSF6) (Li-Weber *et al.*, 1999; Li-Weber and Krammer, 2002; Droin *et al.*, 2003) is a member of the TNF superfamily of cytokines (Locksley *et al.*, 2001) and is involved in causing apoptosis in various models

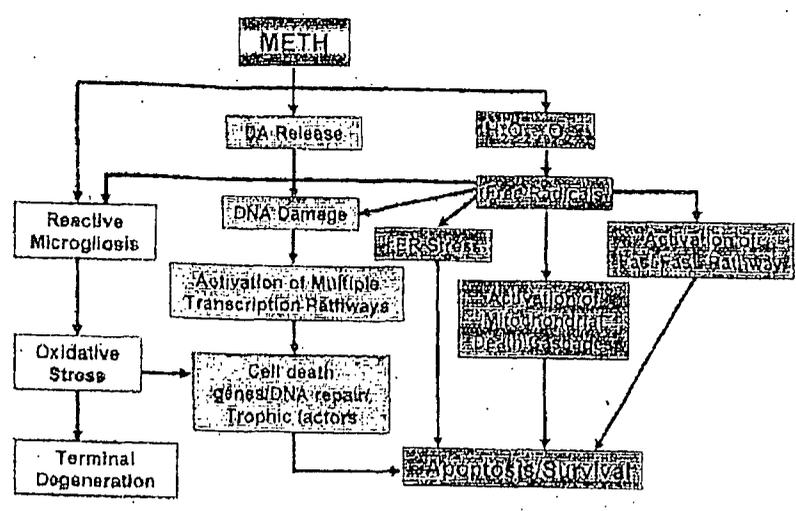


FIGURE 2 METH-regulated molecular events that lead to neuronal apoptosis and terminal degeneration in the striatum. This figure summarizes findings of the various papers that have addressed the issue of METH-induced neurotoxicity in the mammalian brain. The data indicate that oxidative mechanisms and cell death pathways are involved in the manifestation of METH toxicity.

of neuronal injury (Qiu *et al.*, 2002). METH was shown to increase the expression of FasL (Jayanthi *et al.*, 2005; reviewed in Cadet *et al.*, 2005). It was also shown that METH can induce cleavage of caspase 8, which is a known participant in the Fas death pathway (Nagata, 1999).

We have summarized these molecular mechanisms in a theoretical scheme that represents the sequence of events leading to METH-induced neuronal apoptosis and terminal degeneration (Fig. 2).

METHYLENEDIOXYMETHAMPHETAMINE (MDMA, Ecstasy)

3,4-Methylenedioxyamphetamine (MDMA, Ecstasy) is an abused ring-substituted phenyl-isopropylamine that is related to both amphetamines and hallucinogens (McKenna and Peroutka, 1990). MDMA effects which include increased locomotor activity (Matthews *et al.*, 1989) are thought to be mediated, in part, by the release of 5-HT (Liechti *et al.*, 2000) and subsequent stimulation of its receptors (Bankson and Cunningham, 2001). In addition to MDMA behavioral effects, the drug is known to cause marked decreases in markers of 5-HT terminals (White *et al.*, 1996). Specifically, levels of 5-HT and its metabolite, 5-hydroxyindoleacetic acid

(5-HIAA) (Colado and Green, 1994), tryptophan hydroxylase (TPH) activity (Stone *et al.*, 1987) and the number of 5-HT uptake sites (see Lyles and Cadet, 2003) are all decreased after MDMA administration. MDMA can also cause cell death in some *in vitro* models (Simantov and Tauber, 1997; Stumm *et al.*, 1999).

MDMA Neurotoxicity in Animals and Humans

Neurochemical and anatomical studies have shown that MDMA can cause long-term abnormalities in 5-HT systems of rodents (Schmidt *et al.*, 1986; Stone *et al.*, 1986; Commins *et al.*, 1987; Schmidt, 1987; O'Hearn *et al.*, 1988; Molliver *et al.*, 1990). These include decreased levels of 5-HT and its major metabolite, 5-HIAA (Commins *et al.*, 1987; Schmidt *et al.*, 1987; Schmidt, 1989; Molliver *et al.*, 1990), decreased number of 5-HTT (Battaglia *et al.*, 1987; Commins *et al.*, 1987; De Souza *et al.*, 1990), and decreased activity of the rate-limiting enzyme of 5-HT synthesis, TPH (De Souza *et al.*, 1990; Molliver *et al.*, 1990). These changes occur in the rodent neocortex, striatum, and hippocampus (Battaglia *et al.*, 1987; Slikker *et al.*, 1988; De Souza *et al.*, 1990; Molliver *et al.*, 1990). These abnormalities are reported to last for months or even years after drug administration (Battaglia

et al., 1988; Scanzello *et al.*, 1993; Fischer *et al.*, 1995; Lew *et al.*, 1996; Sabol *et al.*, 1996; Hatzidimitriou *et al.*, 1999).

Similar adverse effects have been reported in non-human primates (Ricaurte *et al.*, 1988a,b; Slikker *et al.*, 1988; Insel *et al.*, 1989; Scheffel *et al.*, 1998; McCann *et al.*, 2000). There are dose-dependent reductions in 5-HT concentrations in the cortex, caudate nucleus, putamen, hippocampus, hypothalamus and the thalamus (Ricaurte *et al.*, 1988b). Reduced 5-HT levels were evident for up to seven years following exposure to the drug (Scheffel *et al.*, 1998; Hatzidimitriou *et al.*, 1999). The MDMA-induced deficits in nonhuman primates are also reflected in the levels of 5-HIAA in the cerebrospinal fluid (CSF) (Ricaurte *et al.*, 1988a; Insel *et al.*, 1989). Living baboons treated with MDMA (5 mg/kg s.c., 2 X daily, 4 days) also show marked and prolonged decreases in 5-HTT density measured by PET imaging of (+)[¹¹C]McN-5652, a radioligand that selectively binds to the 5-HTT (Scheffel *et al.*, 1998). Brain tissues from these animals (sacrificed 3 weeks after the last PET and 13 months after MDMA administration) showed marked loss of 5-HT terminals (Scheffel *et al.*, 1998).

A number of investigators have also tested the possibility that MDMA can cause degenerative effects in the human brain (Ricaurte *et al.*, 1988a; 1990; Price *et al.*, 1989; McCann *et al.*, 1994; 1998a; 1999; Bolla *et al.*, 1998; Semple *et al.*, 1999; Gerra *et al.*, 2000; Kish *et al.*, 2000; Buchert *et al.*, 2001). Some of these studies have concluded that MDMA is also toxic to humans because CSF 5-HIAA levels are reduced in MDMA abusers (Ricaurte *et al.*, 1988a; 1990; McCann *et al.*, 1994; 1999; Bolla *et al.*, 1998). PET imaging studies, using [¹¹C]McN-5652 to selectively label 5-HTT, have reported significant differences in 5-HTT binding in MDMA abusers compared to non-MDMA users (McCann *et al.*, 1998a). 5-HTT sites were decreased in a manner that correlated with the extent of abuse (McCann *et al.*, 1998a; Ricaurte *et al.*, 2000). In a similar study, using [¹²⁵I]β-CIT, Reneman *et al.* (2001) investigated the effects of ecstasy abuse on the density of cortical 5-HTT. They also found decreases in cortical 5-HTT in recent MDMA abusers. However, there were no significant reductions in ecstasy abusers who had not used the drug in the past year or longer (Reneman *et al.*, 2001).

The biochemical and molecular bases of MDMA-induced neurotoxicity are being actively investigated. These pathways are thought to involve the formation of toxic MDMA metabolites, temperature dysregulation, dopamine-based quinone formation, and excitotoxic events.

Formation of Toxic Metabolites

MDMA metabolites, which generate free radicals, associated oxidative stress, and membrane damage, are thought to be involved in drug-induced neurodegeneration (Paris and Cunningham, 1992; Colado and Green, 1995). This idea is supported by observations that subcutaneous administration of MDMA metabolites, MeDA and HMA can cause decreases in 5-HT concentrations in the frontal cortex (Yeh and Hsu, 1991), although this line of research has remained controversial. The formation of hydroquinones, quinones and the subsequent generation of superoxides and hydrogen peroxide might be important to the manifestation of MDMA toxicity. These ideas are supported by the observations that the spin trap reagent and free radical scavenger, α-phenyl-*N-tert*-butyl nitron (PBN), prevented MDMA-induced toxicity (Colado and Green, 1995). In addition to MDMA metabolites, the participation of a toxic metabolite of 5-HT has also been invoked because the drug causes marked increases in 5-HT release (Gudelsky and Nash, 1996; O'Shea *et al.*, 2005; Amato *et al.*, 2006).

DA-induced quinone formation is also one possible cause of MDMA toxicity. This suggestion is supported by the fact that MDMA elicits DA release (Shankaran and Gudelsky, 1998; Amato *et al.*, 2006). In addition, destruction of DA terminals by injections of 6-hydroxydopamine protects against MDMA toxicity (Schmidt *et al.*, 1990). In contrast, pretreatment with L-DOPA, which increases DA levels, exacerbates MDMA toxicity (Schmidt *et al.*, 1990). Thus, DA, which is released by MDMA into synaptic clefts, might be taken up by 5-HT terminals where it is converted into quinone by-products that damage 5-HT terminals (Schmidt and Kéhne, 1990; Sprague and Nichols, 1995). It is important to point out that the DA hypothesis does not account for the fact that MDMA can damage 5-HT terminals in areas of the brain such as the hippocampus (Shankaran and Gudelsky, 1998) that are almost devoid of DA terminals and for the fact

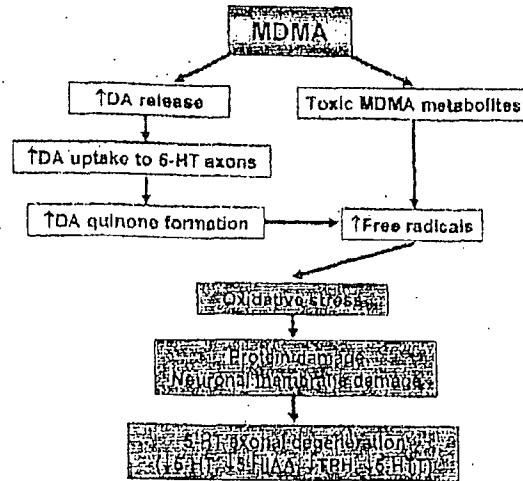


FIGURE 3 Mechanisms implicated in MDMA-induced 5-HT terminal degeneration. The schematic diagram shows that formation of toxic MDMA metabolites, DA quinones and oxidative stress may underlie MDMA toxicity towards 5-HT terminals in the brain.

that, in almost all animal species, except for mice (Cadet *et al.*, 1995), MDMA toxic effects appear to involve 5-HT systems. There is also molecular evidence for the involvement of a number of metabolic pathways in MDMA-induced neurotoxic damage to the brain. Using techniques of microarray analyses, it has been shown that MDMA administration influences the expression of several genes that code for proteins that are involved in metabolism and stress responses (Thiriet *et al.*, 2002). These changes in expression include increases in mRNA levels for Gpx-1 and heme oxygenase (Thiriet *et al.*, 2002). Because MDMA is metabolized via pathways that can induce the formation of superoxides and peroxides via redox-cycling (Cadet *et al.*, 1994b; 1995, Buchert *et al.*, 2001), the changes in these enzymes might constitute compensatory responses to incipient oxidative damage. A schematic diagram of MDMA-induced events that might cause degeneration of 5-HT terminals is presented in Fig. 3.

Possible Role of Glutamate and Nitric Oxide in MDMA-induced Toxicity

Glutamate is a neurotransmitter that can cause cell death both *in vitro* and *in vivo* (Dawson and Dawson, 1998). It has been suggested that glutamate might also be involved in MDMA toxicity (Atlante *et al.*, 2001; Battaglia *et al.*, 2002; Stewart *et al.*, 2002). For example, blockade of NMDA

receptors with the antagonist, MK-801, was able to provide some protection against MDMA-induced 5-HT depletion (Farfel *et al.*, 1992; Colado *et al.*, 1993; Atlante *et al.*, 2001; Battaglia *et al.*, 2002; Stewart *et al.*, 2002), although MK-801 had no effect on drug-related decreases in TPH activity (Johnson *et al.*, 1989). The role of NO in MDMA toxicity also has been investigated in rats. It has been reported that NG-nitro-L-arginine methyl ester (L-NAME), an inhibitor of NO synthase, protects against the neurotoxic effects of MDMA via a mechanism that involves temperature-regulation *in vivo* (Taraska and Finnegan, 1997).

Role of Hyperthermia in MDMA Neurotoxicity

The amphetamines, including MDMA, are known to cause hyperthermic responses (Nash *et al.*, 1988; Gordon *et al.*, 1991; Dafters, 1995; Dafters and Lynch, 1998). A number of drugs that attenuate MDMA toxicity also prevent the marked drug-induced hyperthermia. Specifically, 5-HT₂ receptor antagonists that block the hyperthermic response also protect from MDMA toxicity (Nash *et al.*, 1988). Moreover, preventing the hyperthermic responses produced by ketanserin also abolished its protective effects (Malberg *et al.*, 1996). In contrast, some agents, such as fluoxetine, that provide protection against MDMA neurotoxicity do not block the MDMA-induced temperature increase (Nash

et al., 1988; Mechan *et al.*, 2002). The evidence suggests that hyperthermia might be a member of a complex set of events that participate in the toxic cascades caused by the drug.

It is interesting to note that interactions between the hypothalamic-pituitary-thyroid axis and sympathetic nervous system might be involved in MDMA-related hyperthermic responses (Sprague *et al.*, 2003). For example, removal of either the pituitary or thyroid glands was shown to prevent hyperthermia produced by drug treatment (Sprague *et al.*, 2003). In addition, the use of antagonists of $\alpha 1$ and $\beta 3$ adrenergic receptors was able to attenuate MDMA-induced temperature increase when used alone and could abolish the thermic response when the drugs were co-administered (Sprague *et al.*, 2003; 2005). Of further interest is the report that the skeletal muscle uncoupling mitochondrial protein 3 (UCP-3) is also involved in mediating MDMA-mediated hyperthermia because UCP-3-deficient mice treated with the drug showed blunted hyperthermic responses (Mills *et al.*, 2003).

CONCLUDING REMARKS

The amphetamines have a long history of illicit use among the various classes in societies around the world. The abuse of these drugs has continued unabated inspite of the documentation of the clinical and basic toxicology. In this review, we have presented evidence that oxidative and excitotoxic mechanisms, hyperthermic responses, and other metabolic processes are involved in causing the neurodegenerative effects of AMPH, METH and MDMA. In addition, both AMPH and METH have now been shown to cause cell death in various regions of the rodent brain via mechanisms that involve mitochondrial pathways. Moreover, METH-induced neuronal apoptosis appears to also be dependent on the activation of caspase-12 through the endoplasmic reticulum (ER) death pathway. More recently the Fas/FaL receptor-mediated cell death mechanisms were also shown to be involved in METH toxicity. Microarray analyses have also documented the involvement of molecular pathways that were not initially thought to participate in mediating the effects of these drugs. Thus, modern neurobiological techniques are offering more information on the nefarious effects of

these drugs. It is hoped that this review will provide a substratum for other investigators to build upon.

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Declaration of Christopher Lee Olsen

I, Christopher Lee Olsen, declare the following:

1. Around about the age of 13 I started experimenting with drugs and alcohol.
2. By the age of 14½ I was regularly smoking marijuana, taking mushrooms and drinking alcohol, and had taken methon several occasions.
3. At 16 I was expelled from school for being drunk and ordered to have a drug and alcohol evaluation.
4. By 17 I was addicted to methamphetamine and using intravenously every day.
5. From July of 1998 to August of 2001 I used methamphetamine 2 and 3 times a day almost every day.
6. In September of 2001 I was arrested for possession of Vicodin at the time of my arrest I was extremely high on methamphetamine. After my release I immediately continued my drug use.
7. On 7/26/02, 8/13/03, ~~9/1/03~~, ~~3/10/04~~
I was arrested for forgeries ^{committed} ~~committed~~ to get high.

8. on 1/24/06 I was sentenced to 33 months on Dosa from two incidents arising from a July 18 and a September 29 arrest from 2005 for possession of meth and a firearm

9 in October of 2006 I was released from the Olympia WA DOC work release.

10 by October 15th I started using methamphetamine again

11 from October ²⁰⁰⁶ to December of 2006 I lost roughly 43 pounds.

12. During From the end of October ²⁰⁰⁶ to the early week of December 2006 I was injecting between 10-13 grams of methamphetamine everyday and suffered many episodes of broken Reality and suicidal thoughts and Blackouts (considerable amounts of time I cannot recall)

13 I November of 2006 I wrecked my car during a meth related Blackout.

14 On the night of January 29, 2007 I used about 65 grams of Methamphetamine and drank about 6-8 ounces of Patron Tequila and took 3 pills of ecstasy at the Little Creek casino hotel.

15. By January 29, 2007 I had not had a full nights sleep in almost 2 ~~months~~ weeks and I was frequently told by people around me that I was talking to myself from time to time mumbling and behaving "like a ding" (ding is a slang term for crazy person).

16. I told my attorney Mr. Woodrow on several occasions that, the night of my release from jail as soon as we (myself, Sublett and Frazier) reached the hotel we immediately started using meth and that I used a diabetic syringe to inject about 2 1/2 grams of meth into my veins, then I ate another 1 1/2 grams, and we smoked 6-7 bowls of meth while taking shots of "petron" and that I had taken 3 half pills of ecstasy that a friend at the casino hotel had given me.

17. I also hereby swear that I told my attorney Mr. Woodrow that I knew the juror referenced in ground II and that I had in fact prayed about the case in question with him and therefore he knew about the case prior to trial, knew that I was currently in jail while going to trial, and most likely had a preformed opinion of me when he came into this case.

18. That on June 16, 2008 I informed my attorney Mr. Woodrow that the juror in question was being untruthful when he said he had not seen me in the jail during church.

Services, And Mr. Woodrow did nothing about it.

I declare under penalty of perjury pursuant to the
Laws of the state of Washington that the foregoing is true and
correct to the best of my knowledge.

Christopher Olsen

May-2-2013

Christopher L. Olsen

Dated May 2nd 2013

Attachment # 11

10-JUNE SUBLETT - VI

12 Q. Now, you also testified on direct examination that Mr.
13 Olsen was sitting under the kitchen table with his knees
14 drawn up and he was crying, right?

15 A. Yes.

16 Q. But then a little bit later on he says he liked killing
17 people, right?

18 A. No, first he made the statement, and then a little while
19 later he was sitting underneath the table crying after
20 Michael had left and went to the store to try out credit
21 cards and buy us juices and pop.

22 Q. So Mr. Olsen liked crying, sitting under the table,
23 hugging his own knees. Is that what you're saying?

24 A. I was just telling you what happened.

25 Q. I mean, that was Mr. Olsen's reaction to the murder,

APRIL FRAZIER/Cross (Mr. Woodrow)

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1 right?

2 A. I believe that when he said the statement that he liked
3 it and would do it again that he was trying to impress
4 Michael.

5 Q. In fact, Mr. Olsen was acting so scared, so panicky, so
6 upset that Mr. Sublett took him for a ride in his car,
7 right?

8 A. Yes. Everybody's emotions were up and down, up and
9 down.

10 Q. But Mr. Olsen liked killing Mr. Totten, right? He liked
11 it?

12 MR. BRUNEAU: I'm going to object to the
Page 30

10-JUNE SUBLETT - VI

- 12 A. Yes.
- 13 Q. Now, I met with you in the jail, right, with my private
14 investigator and your attorney, Mr. Meyer?
- 15 A. Yes.
- 16 Q. And I was there. I said, you know, my name is Richard
17 Woodrow, I'm the attorney for Mr. Olsen, right?
- 18 A. Yes.
- 19 Q. And the interview started about 2:45 and lasted till
20 about 4:30 or so, right?
- 21 A. Yes.
- 22 Q. So we were there a good two hours?
- 23 A. Yes.
- 24 Q. And we talked about everything about that case, right?
- 25 A. No, I don't believe we talked about everything.

APRIL FRAZIER/Cross (Mr. Woodrow)

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- 1 Q. But when we were there talking, you never said anything
2 about Mr. Olsen saying I like killing people and I'm
3 gonna do it again, right?
- 4 A. No.
- 5 Q. Were you saving it up?
- 6 A. I was answering the questions that you were asking.
- 7 Q. And I never asked you a question like what did Mr. Olsen
8 say?
- 9 A. I don't believe so.
- 10 Q. Nobody did? The PI didn't either?
- 11 A. Not that I recall, no.

a Attachment #12

9-JUNE SUBLETT - V

- 14 that right?
- 15 A. Yes.
- 16 Q. And when you got to the room, what did you three do?
- 17 A. Well, we started drinking and getting high.
- 18 Q. Getting high on what?
- 19 A. Methamphetamine.
- 20 Q. And during this period of time when you were drinking
21 and using meth, did the subject of robbing Jerry Totten
22 come up?
- 23 A. It was a couple hours after we'd gotten there. First we
24 were going to just sit back and get high for a little
25 bit and then, umm, the subject came up.

APRIL FRAZIER/Direct

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- 1 Q. How did the subject come up?
- 2 A. Umm, that was Chris's favor I guess you could say in
3 return for being bailed out. He was to help rob Jerry.
- 4 Q. And he was to help rob Jerry, and what was he supposed
5 to get out of it? What was Mr. Olsen supposed to get
6 out of the robbery, do you remember?
- 7 A. The money that it cost to bail him out.
- 8 Q. And this was payback for bailing him out?
- 9 A. Yes.
- 10 Q. And was the -- were any plans discussed between Mr.
11 Sublett and Mr. Olsen about how to do this?
- 12 A. The exact plans were not discussed in front of me.
- 13 Q. Do you know whether or not Mr. Sublett and Mr. Olsen had

- 14 any conversation about the way things would happen?
- 15 A. Yes, they did.
- 16 Q. How do you know that?
- 17 A. They spoke about it in the other room. We had a
- 18 two-room suite at the hotel, so I was not in the room.
- 19 Q. Now, you were aware that a robbery was supposed to take
- 20 place?
- 21 A. Yes.
- 22 Q. And what was your part in this crime?
- 23 A. I'm supposed to go over to Jerry's and finish the
- 24 laundry that I had started, and that was the way into
- 25 the house.

APRIL FRAZIER/Direct

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- 1 Q. All right. And who did you discuss -- well, if anyone,
- 2 who did you discuss your part in this plan with?
- 3 A. Michael.
- 4 Q. And do you recall when was it that you had left your
- 5 laundry at Jerry Totten's house? Was it after you came
- 6 back from Reno?
- 7 A. Yes.
- 8 Q. And would that have been about the time that you were
- 9 supposed to cook dinner for Jerry but didn't?
- 10 A. Yes, it was also during that day.
- 11 Q. All right. Now, did you -- did you call and let Jerry
- 12 know that you were coming over?
- 13 A. Yes.
- 14 Q. And would that have been at the casino or do you recall?

1 Chris started crying and he was sitting on the floor underneath the dining room
2 table and I just kept asking him, "Are you okay?" are, you know, I mean, it really
3 seemed to me like he had flipped out, psychotically flipped out. He wasn't
4 making, he was talking but he wasn't saying real words. He was sort of
5 mumbling and crying and I said, you know, "Michael's probably going to kill us
6 both if we don't just finish this," you know, and he knew that, and... um... so
7 while Chris and I were talking Michael was outside trying to start Jerry's light
8 brown truck. Jerry owns two trucks and the light brown truck was the one that
9 Michael wanted to take, but it wouldn't start. So after awhile of him being out
10 there trying to start it, he decided to get the other truck. So he started the other
11 truck, Michael did, and backed it up to the carport of Jerry's house, which was
12 the backdoor, and he told, while he was outside doing, messing with the truck,
13 the moving the truck, he told me and Chris to get Jerry's body out to the
14 doorway, and we couldn't. Chris and I tried.

15 Q: How did you try to get his body out?

16 A: Um... Chris... Michael suggested that we take this table that was like a plastic
17 buffet table, big white long table, and put Jerry on it. That would be easier to
18 scoot him than it was because we tried. There was no way, Jerry's a large man.
19 There's no way Chris and I could have carried him. No way. We tried to, Chris
20 just tried to lift him up at first and couldn't even budge him really, and um, so we
21 put the, Chris and I put the table up on the floor um, in front of the recliner and
22 Chris pulled Jerry off the recliner and onto the table.

23 Q: So uh, at this time were you able to see what Jerry looked like? I mean, the
24 blanket was over...

25 A: The blanket slipped off partially and I could see his face from about his nose up,
26 but I wasn't really looking hard. I did glance at his face and his face was purple.

27 Q: So Chris got him on the table, and uh, how'd you get him out of the house?

28 A: Well then it wasn't as easy as we thought to slide the table either. It was um, it
29 was really hard. So it wouldn't really budge um, we had a... nylon, yellow nylon
30 strap that has metal hooks on it and tried to hook that to the folded legs of the

